DISSERTATION

CHOLESTEROL REDUCTION IN AN "AT-RISK" POPULATION

Submitted by

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WE HEREBY RECOMMEND THAT THE DISSERTATION PREPARED UNDER OUR SUPERVISION BY JEFFREY M. JOHNSTON ENTITLED CHOLESTEROL REDUCTION IN AN "AT-RISK" POPULATION BE ACCEPTED AS FULFILLING IN PART REQUIREMENTS FOR THE DEGREE OF DOCTOR OF PHILOSOPHY.

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ABSTRACT OF DISSERTATION

CHOLESTEROL REDUCTION IN AN "AT-RISK" POPULATION

Two hundred and sixty-nine men and women aged 21-79, who had been identified as "at-risk" for developing coronary artery disease due to total serum cholesterol levels (TC) in excess of 200 mg/dl and/or a TC to HDL-cholesterol ratio (TC/HDL-C) greater than 4.5 took part in this 12 week study of cholesterol reduction in an "at-risk" population. This study was carried out in a hospital setting, where it examined the effectiveness of individual dietary counseling and a self-directed diet education program, Self-CARE for a Healthy Heart (Self-CARE), which was developed specifically for this study; and in a medical clinic setting where the effectiveness of group diet counseling and Self-CARE were evaluated. Two primary end points, the change in TC and the change in the TC/HDL-C ratio, were used to assess the effectiveness of each approach and in comparing individual and group diet counseling to Self-CARE. The 60 individuals that received individual diet counseling experienced a significant decrease in TC (4.7%) and in the TC/HDL-C ratio (6.3%), while the 62 individuals at that same location that received Self-CARE experienced a significant decrease in only TC (5.1%). None of the changes were significantly different from one another. In the clinic setting, group

instruction given to 73 individuals resulted in a significant decrease in TC (11.2%), while the 74 individuals assigned to Self-CARE demonstrated significant decreases in TC (17.6%) and TC/HDL-C ratio (12.6%), changes which were significantly different from the group instruction changes. Overall, it appears that the Self-CARE approach, could be considered a viable alternative to individual and group diet counseling.

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CHAPTER I

INTRODUCTION

There are over 5.4 million Americans with a diagnosis of coronary heart disease (CHD), plus millions who are at risk, but are still asymptomatic.[1] Although there are some widely publicized surgical remedies for these individuals, prevention remains the preferred approach for the management of CHD.[2] Prevention has come to focus on lifestyle, since there is a growing body of evidence accumulating from studies such as the Framingham Study, the Multiple Risk Factor Intervention Trial (MRFIT), the Lipid Research Clinic's Coronary Primary Prevention Trial (LRC-CPPT), and the Helsinki Heart Study which demonstrates that lifestyle affects both longevity and the cause of death.[3,4,5,6,7] Specifically, the LRC-CPPT found that for every 1 percent drop in serum cholesterol there is a 2 percent decrease in risk of heart disease[6] and the Helsinki Heart Study recently published conclusive evidence that each 1% drop in cholesterol produced a 4% decline in heart attacks.[7] These decreases in risk were the result of a combination of diet and drug therapy. However, it has been estimated that perhaps only about 5 percent of people may actually require drugs to keep their cholesterol levels down, while the

remainder of the population should be able to use diet alone to decrease their cholesterol to acceptable levels.[8]

Unfortunately, as revealed by two National Heart, Lung, and Blood Institute (NHLBI) surveys, the public at large, including many health care professionals, are still confused about the cholesterol issue.[9] These same surveys also established that there was a definite need for a national program to educate the public and the health profession community on the cholesterol issue. The NHLBI made plans to meet this need by developing the National Cholesterol Education Program (NCEP).[10]

The intent of the NCEP is to increase awareness of the importance of lowering elevated blood cholesterol levels and also to provide the information and skills necessary to effect the lowering through dietary modifications and medication(s) when appropriate. The NCEP concentrates on identification and treatment of high risk individuals.[10]

Today, the NCEP is providing a framework for the nutrition education efforts of most health professionals[11], pointing out that even though individuals require careful evaluation and individually tailored therapies, the first step in any cholesterol lowering program once it is determined that there are no underlying organic problems should be an attempt at dietary modification.[10,11] Since a significant proportion of patients can obtain a satisfactory response by dietary modification alone, attention turns to the most effective means of delivering this information.

However, the lack of summative evaluation in nutrition education makes it difficult to determine how successful our nutrition education efforts actually are.[12] For example, the study of adult learning has revealed that the adult learner has an approach to learning unique from that of the child, consequently, nutrition education programs that do not consider these peculiarities may not be successful. As delineated in the Theory of Adult Learning[13], the adult learner is problem oriented, that is the adult learner is motivated to learn through the recognition of needs that learning will satisfy. Additionally, adults tend to be self-directing, learning through analysis of past experiences. So while a particular nutrition education program may be consistent with the current knowledge in the field and be presented in a professional, organized manner, the effort may not be successful since the program may not be appropriate to the population being served and as is seen in many nutrition education programs may have focused on knowledge changes, failing to address attitude and behavior changes.[14] Also, the adult learner because of time constraints, job conflicts, or unwillingness to discuss personal problems in a group setting, will often not choose to participate in traditional nutrition education programs.[15]

Therefore, this research project, which I have titled Self-CARE for a Healthy Heart, was developed to evaluate three types of nutrition education programs designed to

reduce serum cholesterol levels in an "at-risk" population. Two approaches currently in use, individual and group programs, are evaluated and then compared to a self-instruction approach to nutrition education (Self-CARE) that was designed specifically for the adult learner and for this study. The final comparison being made to determine if a self-instruction approach to nutrition education could be considered a viable alternative to the traditional programs now being used.

CHAPTER II

REVIEW OF LITERATURE

Incidence, Palliation and Prevention of Coronary Artery Disease

Incidence

Heart and blood vessel disease is the major cause of death in the United States. The most recent statistical data released by the American Heart Association[16,17] show that heart and blood vessel diseases claim a life every 32 seconds. This means that almost one million Americans died from heart and blood vessel diseases in 1986, the most recent year for which data are available. Encouragingly, this number represents a decline in the proportional death rate of 24% for the preceding decade, but unfortunately, nearly one in four Americans still have some form of heart disease. In fact, nearly one-third of the deaths from all causes in persons aged 35-64 years are due to coronary heart disease (CHD).[2] It is estimated that over one million Americans suffer a heart attack each year, and of these victims, almost one-half will die from their affliction.[16] In total, there are over 5.4 million people with a diagnosis of CHD, plus millions who are at risk, but still asymptomatic.[1] This number is still alarming and, in spite

of the progress being made, demonstrates that the efforts to reduce the incidence of heart disease must be continued.

It is interesting to note that the decline in death rate reported for 1986 represents the continuation of a trend that began in the mid-1960's.[18] This trend represents a reversal of a gradual rise in CHD that was first noted in 1918, and that apparently continued to accelerate throughout the first half of this century. This rise has been referred to as "an epidemic of CHD".[19] By 1940, mortality due to heart disease had risen to 27.2% of all deaths, up from 8.0% in 1900. During this same period coronary artery disease (CAD) had become the major cause of heart disease deaths among persons aged 35-65.[20] Epstein[21] reported in 1965, that CAD was still the major cause of death for this age group and that CAD deaths were still rising, while overall cardiovascular disease was beginning to decline. During the mid 1960s, this "epidemic" came to an end[2] and the United States experienced a dramatic decline in mortality from CHD and stroke. While some scientists questioned whether an "epidemic" had really occurred,[19] Slater[22] has published a convincing article that reveals that 1) there was in fact a true epidemic that affected males exclusively or to a greater degree than females and that it is now on the decline, and 2) that a fairly stable and more nearly comparable mortality for both males and females for chronic ischemic heart disease exists.

The reason(s) for the decline in CHD has been the subject of many articles.[2,19,23-25] Some of the major causes mentioned have been changes in smoking habits, lifestyle, the environment, and dietary habits. Other authors have challenged each of these explanations.[22] Stallones[25] in a 1980 review of the evidence concluded that no single factor can be found that will account for the increase and subsequent decline of CHD mortality over the last 70 years. Support for Stallones' findings came from Ragland et al.[26] who analyzed the onset of decline in CHD mortality in the United States. These researchers found that the temporal and spatial patterns of the decline suggested that "the declines in mortality ... are due to a series of changes that took place at various times in different regions." Similar conclusions were reached by Levy[2] and Slater et al.[22] Although not yet established, the decrease in incidence of CHD is most likely due to the spread of health-related changes such as smoking cessation, adoption of low-fat diets, control of high blood pressure, and improvement in medical care.

Palliation

Improved medical care has also been shown to be responsible for the decreased case-fatality rate[1,27] which makes its own contribution to the overall decrease in CHD mortality. While agreeing that improved medical care has had an effect, Stallones[25] claims that it is not likely that

it would have a major effect on population mortality data. Others disagree, and in reviews by Levy,[23] Feinleib[27] and Peters[1] the impact of treatment methods on mortality rates are detailed. For example, since 1963, coronary care units have been standard features in all hospitals. Cardiopulmonary resuscitation is a skill learned in childhood, and physicians now have a variety of drugs (beta-blocker, calcium channel blockers and prostaglandin inhibitors) at their disposal for preventing life-threatening rhythm problems. Coronary artery bypass surgery has become common, with over 140,000 such surgeries being performed each year. Other less prominent procedures and techniques such as angioplasty, the use of thrombolytic agents such as streptokinase and left ventricular assist devices are now in use but have not been used long enough to gage their effectiveness. Unfortunately, even for the established procedures, Levy[23] makes a cogent case for the lack of conclusive evidence that they have had a major impact on CHD. However, there is evidence linking better medical care with beneficial effects on CHD mortality.[28] Even if improved treatment is successful, as Levy[2] points out, "palliation and repair are costly...It is evident that we must focus on prevention of CHD as our long-term goal."

Prevention

As the discussion above has pointed out, there has been an extension in the life-span for Americans, an extension

that has been the result of a tremendous decrease in the acute illnesses that once routinely struck our population. However, as we have seen most of the acute diseases conquered or controlled, there has been an increase in the average age of the population, and consequently an increase in chronic diseases of the elderly. This shift has led to a tremendous increase in the cost of medical care as society has been called upon to pay for the increasing morbidity within our population. This introductory discussion was inspired by a series of articles by James Fries.[28-31] In the last of his articles[31] he asks the central question, "is it easier to prevent mortality or to prevent morbidity?" Present information does show that improved treatment has played a significant role in the decrease of CAD mortality;[23] however, it appears that the role played by lifestyle changes was important.[16,22-24,26,27] Additionally, as Harper[19] points out, the human life span is finite and even if we were to completely eliminate CAD, life expectancy would not be drastically altered. Consequently, it appears that we will eventually die of something, and perhaps the most important factor is how well we live until that time. This concept of the "compression of morbidity" puts the emphasis on prevention rather than cure and on postponement rather then palliation.[31] This approach yields a double benefit: first, it creates a vigorous, healthy individual able to contribute to society; and second, it could mean a dollar savings for the country as the onset of chronic

diseases is delayed. Delaying the onset of chronic disease involves decreasing one's risk. Many risk factors have been identified.[32] Factors such as cigarette smoking, lack of exercise, obesity, hypertension, saturated fat intake, alcohol intake, fiber intake, occupational toxins and environmental toxins are all associated with chronic disease. Prevention then can take one of two pathways: primary prevention or secondary prevention.

Primary. Primary prevention involves the measures taken to decrease an individuals risk of developing disease prior to the clinical manifestations of any symptoms.[33] This type of prevention would include preventive health services, health protection and health promotion.[17]

Secondary. Secondary prevention includes the measures that promote early diagnosis and prompt therapy to shorten duration of illness, to ameliorate the symptoms of the disease and prevent reoccurrence if possible.[33] Included in this area would be medical treatment as well as lifestyle changes and education.[17]

The appropriate prevention strategy according to the Department of Health and Human Services[17] will then depend on, "the relations among the incidence of diseases, their common risk factors, their common outcomes, and the extent to which the risk factors are related to the goals and expectations of the population." Nevertheless, prior to determining which, if any, prevention strategy should be used, it is necessary to explain the pathogenesis and

etiology of the disease in question. In this case, and for the remainder of this review, the focus will be on CAD. The terms CAD, ischemic heart disease, and atherosclerotic heart disease are more or less synonymous, they denote the clinical manifestations of atherosclerosis[25] and will be referred to as CAD from here on. CHD on the other hand would include CAD and refers to myocardial damage or symptoms caused by any type of the pathological changes in the coronary arteries sufficient to interfere with adequate blood flow.[33]

Pathogenesis and Etiology of Coronary Artery Disease

Pathogenesis

Since World War II an immense amount of effort, time and money has been spent attempting to define in specific terms how atherosclerosis develops and results in CAD.[25,34,35] As was pointed out by McGill,[34] research activities have generated volumes of information regarding the biochemistry of lipid metabolism, vascular physiology, endothelial and smooth muscle structure and function, and blood flow characteristics to name only a few areas. These cellular and molecular investigations have however produced numerous insights into the pathogenesis of atherosclerosis. One such insight is the fact that atherosclerosis is apparently a response of the vascular wall to injury.[36] For detailed information, one should consult the numerous reviews of this area of study[35,37-44] and review the Nobel Prize work of Brown

and Goldstein[45,46] on low density lipoprotein cholesterol (LDL-C) metabolism . Briefly, the "response to injury hypothesis" proposed by Ross[43,44] and reviewed by Steinberg[40] states that "injury" to the endothelium is the initiating event in atherogenesis. This "injury" then leads to the fundamental lesion of atherosclerosis, the fibrous plaque. This is the lesion that causes the narrowing of the artery, calcifies and predisposes to thrombosis.[36,40] Unfortunately, even with all this research, the exact relationship between the risk factors associated with increased incidence of CAD and what is taking place at the cellular level is still not clear.[36] However, the modified "response to injury hypothesis", as laid out by Ross,[38] offers a cogent explanation for some of the reasons for the progression of the disease (i.e. what genetic factors or environmental agents may be responsible).

Etiology

The etiology of CAD is very complex. While the public has unfortunately come to equate only cholesterol with CAD, there are many environmental and genetic factors that contribute to its development. Scientists and epidemiologists studying the causes of CAD have identified many factors that have a suggested positive or inverse association with CAD.[32] All of these various factors have popularly become known as "risk factors", and out of this large number of "risk factors", a few have emerged that are

consistently and so strongly associated with CAD, that they are commonly believed to be causal.[34] The three major risk factors which have emerged from epidemiological and medical studies are high blood pressure, cigarette smoking, and elevated plasma cholesterol.[35] The first two are beyond the purview of this review and the third, elevated serum cholesterol, will then be the focus.

Little interest was generated by the observations of 19th century pathologists that the fibrous tissue of intimal plaques in coronary arteries contained deposits of lipid. Likewise, the 1914 reports from a Dutch physician in Java, of the association between diet, serum cholesterol, and atherosclerotic disease in humans[47] drew little comment. However, after the identification, in 1932, of cholesterol and its structurally related sterols, the role of dietary fat and cholesterol in the development of atherosclerotic plaques in humans became one of the medical obsessions of the late 20th century. Interest in the relationship between diet and blood lipids progressed rapidly after Stamler and Dock[48,49] first suggested that elevated blood cholesterol concentrations were possibly one of the causes of CAD. The research community then saw these early suspicions confirmed by the first results of the Framingham heart Study.[50] Today, we can look back at seven decades of epidemiological, animal, and human research that has examined everything from Arctic Eskimos to New Guinea tribesman. No attempt will be made to review the vast amount of literature on serum

cholesterol and CAD. There are several excellent review articles[51-54] that I would recommend for those that are interested in an in depth look at this area of research. I will focus on the Framingham Heart Study (FHS), which has made the largest contribution to our understanding of the etiology of CAD.

The FHS[3] has been in continuous operation since 1949, following a representative sample of the adult population of Framingham, Massachusetts for the development of CHD. In all, 2,282 men and 2,845 women aged 30-62 were initially examined. Less than 2% of the population sample was completely lost to follow-up at the 14 year point. At that time 8% had died from all causes. At the 14 year follow-up, as at each of the biennial follow-ups, the lipid analysis continued to show a that the risk of developing CAD was proportional to the serum cholesterol level. The relationship was generally stronger in younger than in older persons. They also found that there was nothing to suggest that some level was critical, since risk rose in proportion to the serum cholesterol concentration. The researchers have observed that the net effect of cholesterol is "clearly apparent in men of all ages."[55] It is interesting to note that in the FHS there were 6 xanthomatous individuals in the population sample of 5,127. All had serum cholesterol levels greater than 400 mg/dl and a strong family history of CAD. Within the follow-up period, all six died from CAD prior to their 50th birthdays.

Findings published in 1971[3] reported that any one of the lipids or lipoproteins examined can be used effectively for assessing vulnerability to CAD, and that none however, appeared superior to the more convenient serum cholesterol determination. However, in recent articles the assertion has been made that age must be taken into consideration. In the 30 year follow-up from the FHS,[56] it was reported that after age 50 there is no increased overall mortality with either high or low serum cholesterol. Under age 50 however, cholesterol levels are directly related with 30 year overall and CVD mortality. Even when they examined cholesterol levels as low as 180 mg/dl, they found that there did not appear to be a threshold value. The studies conclude that, "overall death increases 5% and CVD death 9% for each 10 mg/dl increase in serum cholesterol." Their observation that the association between cholesterol and CHD becomes stronger with lengthy follow-up may explain the lack of statistical significant results in many of the relatively short intervention trials.

Levy et al.[57] and Wilson et al.[58] have determined from their analysis of the Framingham data that high density lipoprotein cholesterol (HDL-C) levels are a potent predictor of CHD death in both sexes. Individuals with HDL-C levels above 65 mg/dl were found to be at very low risk for CHD. These findings mean then that at a minimum, age, total serum cholesterol (TC) and HDL-C cholesterol levels should be considered prior to making a risk determination.

The FHS results have helped to answer the question "does hypercholesterolemia increase the risk of CHD." In fact, most epidemiologic studies have shown that the risk of heart disease increases continuously from a TC of 180 mg/dl upwards.[57,59-61] While the magnitude of the increase in risk from levels of 180 to 240 or 260 mg/dl is still of concern to some,[60] the evidence which has accumulated over the past few decades leaves little doubt that elevated levels of plasma cholesterol do contribute to the progression of CAD. Additionally, there is now also evidence to show that serum cholesterol levels are determined partially by the intake of dietary cholesterol, saturated fatty acids, polyunsaturated fatty acids, and total calories. Let us now take a look at that this evidence.

Evidence Linking Diet and Coronary Artery Disease

Epidemiological Studies

The evidence that links dietary lipids (fats and cholesterol) to CAD is found in epidemiological, animal, and human studies. From 70 years of epidemiologic studies, a dietary hypothesis concerning CAD has developed. This hypothesis states that high cholesterol, high saturated fat diets do relate to the occurrence of CAD for entire populations.[62] The first report of this association was made, as mentioned above, by deLangen in 1916.[47] Others suspecting that a person's regular diet may determine susceptibility to CAD were Snapper,[62] Stamler and Dock[48,49]

and Keys.[63] These investigators pointed out that there seemed to be a strong association between the occurrence of CAD and the consumption of dietary cholesterol, animal products, and total dietary fat. This assumption is supported by studies from the 1950s of migrants with low plasma cholesterol levels that integrate into populations with high cholesterol levels, only to see their cholesterol levels increase, along with their CAD rates.[64]

The strong relationship between diet and serum cholesterol was illustrated by Stallones[65] when he summarized the results of a large collection of population survey data. He reported that for both men and women, serum cholesterol values are strongly related to the percent of total calories derived from fat. More specifically, in the classic Seven Countries Study conducted by Keys et al.,[63,66] they reported an impressive association between dietary fat and the occurrence of CAD. In another comparison among 24 countries, Connor and Connor[67] looked at the death rates from coronary heart disease compared with the mean daily intake of cholesterol. They reported that the epidemiological evidence about the association of dietary cholesterol and coronary heart disease mortality is especially clear cut. The findings of Connor and Connor were not surprising because of the high correlation between cholesterol intake and saturated fat intake.[51] Perhaps one of the best demonstrations of the striking relationship between serum cholesterol and the incidence of CAD came from

the Pooling Project Report.[54] The eight studies included in this report revealed a steady increase in incidence of CAD as serum cholesterol levels increased. Long term follow-up in the Western Electric Study has revealed that a diet high in cholesterol and saturated fats does predict the 19 year risk of death from CAD.[54] Similar results have been reported in the Zutphen,[68] the Boston-Irish,[69,70] the Honolulu,[71] and the Western Collaborative studies.[72] Of further interest is the fact that while the positive association between the cholesterol and/or saturated fat intake, and the subsequent incidence of CAD seems to hold across studies, only the Western Electric Study showed an association between dietary cholesterol and CAD risk independent of dietary fats.[59] This raises another important question. Does dietary cholesterol have an independent effect on CAD risk? Several researchers hypothesize that, since 47% of the excess CHD deaths attributed to serum cholesterol levels above optimal (180 mg/dl) occur in individuals with cholesterol levels below 245 mg/dl,[60] the intake of dietary cholesterol may increase the level of atherogenic, cholesterol-rich chylomicron remanents.[73] If, in fact, this is true, then it would lend even more support to limiting cholesterol intake to less then 300 mg/day. However, while there is currently no evidence to link chylomicrons with atherosclerosis, research has not absolved them.[39]

In summary then, observational epidemiological research has examined the associations among diet, serum cholesterol and CAD. The conclusions drawn from this research are that diets high in fats and cholesterol are a characteristic of populations with high TC levels, and populations with high rates of CAD. For those that wish to look at this area in detail I would recommend the following articles and reviews: 51,52,54,62,65,74-77.

One word of caution, as discussed by Stallones,[65] is that what is true for populations or communities may not in fact be true for individuals. Just as correlation does not necessarily show cause and effect, measurements in individuals are often of no value in anticipating the subsequent occurrence of CAD. This is to be expected when dealing with a disease that only manifests itself after a prolonged period of incubation and which has such a complex etiology. It has been pointed-out that intraindividual variation and methodological error could easily account for the low correlations found on the basis of a single serum cholesterol measurement or one day diet record.[35,51,78] Additionally, when looking at individuals within a population, it should again surprise no one to find that genetic variability has produced individuals who are high responders, that is individuals with high serum cholesterol levels that are prone to CAD. Conversely, it would not be surprising to find low responders that consume the same diet

as high responders, but have low cholesterol levels and are not prone to develop CAD.[62]

Animal Studies

Another line of evidence that supports the link between diet and CAD comes from experimental animal studies. A review of animal studies by Glueck[52] revealed," an unequivocal causal relationship between dietary cholesterol or saturated fat, plasma cholesterol levels and development or regression of atherosclerosis." This conclusion was based on 80 years of animal research. Since Ignatowsky first induced atherosclerosis in rabbits by feeding them meat, milk and eggs,[52] a variety of animals have been shown to be susceptible to diet induced atherosclerosis.[38] Experiments with monkeys and baboons have produced research which is most applicable to man. The entire range of atherosclerosis in man has largely been reproduced in primates by the feeding of cholesterol and fat.[79] As was mentioned earlier about the human response to fat in the diet, some nonhuman primates are hyperresponsive to dietary cholesterol, while others are more resistant.[53] While studies in man are just beginning to show that regression is possible, a positive answer has been obtained by experiments with rhesus monkeys and baboons.[80,81] Blankenhorn and Kramsch[82] have recently published a review of the status of research on the reversal of atherosclerosis and conclude that the outlook for atherosclerosis control is promising.

Animal experiments have also supported the epidemiological findings that other risk factors such as cigarette smoking, hypertension, and obesity are contributing factors, not a primary factor like hypercholesterolemia.[62] For example, hypercholesterolemic animals developing atherosclerosis will develop more severe atherosclerosis when they suffer from chronically elevated blood pressure. The same features are seen when we look at a Japanese population that suffers from hypertension and smokes, but due to a low fat diet has a low incidence of CAD. For a detailed look at the atherosclerotic process in susceptible animals, reading the review by Ross[38] would be recommended.

Since studies have shown that humans are more resistant to dietary cholesterol and fats than most nonhuman primates, experimental trials in humans to verify animal findings are needed to help determine if the results are in fact transferable. In light of the numerous animal studies that reveal a causal relationship between serum cholesterol and atherosclerosis, and that suggest that the process may be reversible, human studies are certainly justified.[83]

Human Studies

To a substantial degree, most of the fat and cholesterol feeding experiments that have been carried out in animals, have now been replicated in humans. One of the first diet trials to be conducted was completed in 1922. In

this trial, the Dutch physician, deLangen, fed residents of the island of Java a high-fat European diet in place of their traditional, low fat diet of rice and vegetables.[62] He noted an increase in their serum cholesterol. His experiment was not typical of the high cholesterol feeding experiments that would occupy the next 30 years of research. It was only after World War II, when confronted with an apparent epidemic of CAD, that the hypercholesterolemic effect of dietary saturated fat was rediscovered.[84] Since then, there has been a determined effort to discover the effects of both dietary cholesterol and type of dietary fat on lipoprotein levels. This research effort has linked dietary cholesterol and the type of dietary fat to serum cholesterol levels, a link that bears directly on the incidence of CAD.

Early studies of the effect of dietary cholesterol on serum cholesterol levels found almost no positive or negative correlation between the two. Analysis of the Framingham data revealed the same findings.[85] For an excellent review of this research consult McGill[51] and Connor and Connor.[62] Their consensus was that these early experiments were incorrectly interpreted or designed, and that in fact dietary cholesterol does have an effect on serum cholesterol levels. A series of experiments conducted during the early 1960s clearly revealed this effect.[86-88] A 1982 study by Connor and Connor[89] repeated this early work and clearly demonstrated that dietary cholesterol does play

a role in the development of atherosclerosis. They showed that subjects initially consuming a 40% fat diet without cholesterol demonstrated an average increase of 34 mg/dl when 1000 mg/day of cholesterol was added to the diet. The majority of this increase was attributed to an increase in LDL-C. While this experiment seems very clear, as McGill's review[51] points out, the data really show that dietary cholesterol has only a modest effect in man. Keys[49] supports this contention and has shown that in humans, the change in serum cholesterol from only a change in dietary cholesterol is small. For example, cutting the intake of dietary cholesterol in half, eg. 300mg/1000 kcal to 150 mg/1000kcal, can on average be expected to decrease serum cholesterol about 7.6 mg/dl.

Additionally, this effect is variable. As was mentioned earlier, it has been demonstrated that about one-third of individuals show no response to dietary cholesterol, while another one-third respond with elevations several times the mean change of 10mg/dl for each 100 mg of cholesterol per 1000 Kcal in the diet.[34] This, coupled with the modest response mentioned above, demonstrates the futility of applying the Key's equation with respect to dietary cholesterol to individuals.

However, as was shown by Keys et al.[90] and Ahrens et al.[91] in the late 1950's, saturated fats raised, while polyunsaturated fats lowered serum cholesterol. Keys stated that, "saturated fatty acids have, per gramme, about twice

as much effect on serum cholesterol as do the poly-ethenoids which, moreover, act in the opposite direction." Hegsted et al.[86] expanded on the work of Keys, including dietary cholesterol in their equation. Their findings were basically similar to those of Keys with regard to saturation of fats, however Keys was to claim that the Hegsted equation overpredicted the effect of dietary cholesterol by 300%.[49] This charge was refuted by Hegsted,[92] who conceded that his equation did in fact overestimate slightly, but was not inconsistent with the literature. He concluded that the effects of dietary cholesterol are small compared to the effects of dietary fat and that in a free living population the equations predictive value is adequate within the range of usual cholesterol intake.

While there is agreement that the effect of increased dietary cholesterol is an increase in the levels of plasma cholesterol and LDL-C, it appears that there is both a threshold and ceiling amount for dietary cholesterol. Plasma cholesterol levels are increased little at cholesterol intakes above 300-500 mg/day.[93] However, as mentioned above, dietary fats have the major influence on serum cholesterol levels.

In fact, as reported by Anderson et al.,[94] the cholesterol-raising effects of saturated fats do not depend on increased absorption of dietary cholesterol. These workers added 291 mg of cholesterol to essentially cholesterol free diets of equal fat content, but with

different fatty acid composition- 97 grams/day of saturated oils versus 97 grams/day of polyunsaturated oils. They found a significant increase in serum cholesterol with each diet, but the increases were not significantly different from one another. Interestingly, when the saturated fat diet was substituted for the polyunsaturated diet, with or without added cholesterol, the rise in serum cholesterol was the same. These results did not match the results of one portion of the National Diet Heart Study, the Faribault Study.[95] In this study egg yolk added to diets rich in saturated fats (P/S=0.1) demonstrated a larger increase in cholesterol then when added to a diet rich in polyunsaturates (P/S=0.9), thus demonstrating an apparent interaction between the type of fat and the cholesterol. This same interaction was reported by Schonfeld et al.[96] While the issue still remains to be settled there is little doubt that increasing dietary cholesterol up to an individuals ceiling level and/or increasing saturated fats does in fact raise serum cholesterol levels.[53] Additionally, in natural diets there is apparently a further interaction between the type of dietary fat and dietary cholesterol.[97]

The National Diet Heart Study[95] was a collaborative feasibility study designed to assess the effect of diet on serum cholesterol levels. This 1960s trial was one of the earliest conducted specifically to test the effects of dietary modification on serum cholesterol levels. This

study found that a fat restricted diet could in fact reduce serum cholesterol and that the cholesterol reduction was proportional to dietary adherence. This study involved free-living men from the general population and long-term residents of mental institutions but did not look at hard end-points.

The consensus that has developed from these studies is that of all the dietary constituents, saturated fat has the largest influence on serum cholesterol.[89] Unlike dietary cholesterol which has only a small effect on HDL-C, altering mainly LDL-C,[98] saturated fat elevates both LDL-C and HDL-C. However, the increase in HDL-C is small, and any potential benefit is more than offset by a much larger increase in LDL-C. It has been estimated that saturated fats are responsible for approximately 60% of the change in serum cholesterol, while dietary cholesterol is accountable for 40% of the change.[99] It is this basic finding that has molded our attempts to prescribe diets to lower serum cholesterol. For a review the effects of dietary cholesterol and fat on serum cholesterol in humans, read the articles by McGill et al.[78] and Connor and Connor.[97]

Emerging from human nutrition research are several findings that may influence the dietary management of CAD. Even though early studies by Keys[100,101] and Hegsted[86] found that "monounsaturated fats have no effect on upon serum cholesterol", recent experiments by Grundy[102,103] have shown that a diet rich in monounsaturated fats appears to be at

least as effective in lowering plasma cholesterol as a diet low in fat and high in carbohydrates. In addition the monounsaturated fat diet, unlike a diet high in polyunsaturates, does not appear to lower HDLs.

Interest in a diet containing high amounts of fish oils was developed after reports of low CAD death rates in Eskimos that consumed large amounts of oil rich marine fish,[104] and results from the Zutphen Study that revealed an inverse relationship between fish consumption and 20 year mortality from CAD.[104,105] This area of study while promising, still needs further research before more than a recommendation to include more fish in the diet can be made.[104] For a complete review of this area, Herold and Kinsella's article[106] is recommended.

As this review has so far shown, there is abundant information linking diet and CAD. Specific risk factors have been identified and a proposed scheme for their actions identified. Further a host of animal and human experiments have clearly shown that dietary fats and cholesterol are directly responsible for raising the serum cholesterol levels, which in turn contributes to the development of atherosclerosis. Kris-Etherton et al.[99] have recently completed a lengthy, informative review of the role of diet on plasma lipids. While all this research strongly supports a casual link between diet, high serum cholesterol levels and CAD, causality can only be proven by intervention trials.[83]

Support for the "Lipid Hypothesis"

Overview

The direct evidence from clinical trials needed to support the "Lipid Hypothesis" has been accumulating for many years, but only recently has it reached "significance" in the minds of most scientists. This review will now look at the findings from the numerous randomized trials employing cholesterol lowering diets and/or drugs. I have chosen to look at primary and secondary trials separately and further divided each into diet only and trials that used primarily drugs, although dietary advice may have been included. A study was considered a primary one if the main focus was on individuals that did not have any clinical manifestations of CAD. A secondary study was then one in which the subjects were suffering from definite heart disease prior to entry into the study.

Due to the volume of studies available for review, I will concentrate on the studies that are, in my opinion, the most relevant to this examination and refer those who wish to begin an in-depth study of this area to reviews by Buchwald et al.[107] Connor and Connor,[89] Hopkins and Williams,[32] Glueck,[52] Leren,[108] and Tyroler.[109]

Primary Intervention Trials

Diet. Levy[110] reported that between 1955 and 1980, there had been 17 studies completed that attempted to verify the lipid hypothesis. One of these studies, The Diet and

Coronary Heart Disease Study Project, more popularly known as the Anti-Coronary Club[111,112] is an often referenced study that ran for 14 years from 1957 until 1972. This was one of the first studies to look at the use of a "Prudent Diet" (P/S greater than one and less than 350 mg cholesterol per day) for lowering serum cholesterol and the effect it had on the eventual development of CAD. Although this study suffered from problems with the selection and composition of the control group, the final results revealed that there was less heart disease in the diet group. This gave early support to the effectiveness of the "Prudent Diet".[113]

The findings were similar in The Los Angeles Veterans Administration (LAVA) Study[114] which was also known as the Wadsworth VA Trial Of Diet or the Dayton Study. In this study, the diets contained about 40% of their calories from either animal fats or vegetable fats. After eight years, Dayton et al. reported a 20% decrease in serum cholesterol for the experimental group, compared to 7.3% for the controls. The Veteran's Administration researchers claimed that the 12.7% difference in serum cholesterol levels was responsible for a 23% reduction in all CAD events in the experimental group.[48] However, there was no difference in the primary end-point sudden death or myocardial infarction and overall mortality was the same for both groups.

The Finnish Mental Hospital Study[115] was one of the first studies to attempt to look at how dietary changes affect mortality in a large population- approximately 7000

patients. This was not a true primary prevention trial since a portion of the original population had previous CAD. The normal diet in this study had a ratio of polyunsaturated to saturated fatty acids (P/S ratio) of 0.25, compared to a P/S ratio of 1.5 in the experimental diet. The exact fat level in the experimental diet was not given. The experimental diet was the "typical" Finnish diet, known to be at least 40% fat, that achieved its high P/S ratio mainly by substituting vegetable oils for milk fats. The diets were fed for six years and then reversed for another six years. When the pooled diet periods were compared with the pooled control periods, the findings showed reduced coronary heart disease mortality in the dietary treated groups (not significant), and no difference in overall mortality. The study concluded that, "the findings of this study justify the conclusion that a cholesterol-lowering diet reduces the incidence of CAD in men and women."[115]

These three studies, as all the others completed prior to 1980, failed to show a statistically significant difference in the development of definite CAD. Dietary modifications could not in fact be shown to prevent CAD, although the trends and analysis of subpopulations always indicated that dietary modifications were helpful.

Since 1980, several primary prevention trials have been completed that attempt to verify the "Lipid Hypothesis". The major diet trials were the Oslo Heart Study, the American MRFIT Trial, the European MRFIT Trials (Belgium,

and United Kingdom), and the North Kareila Study. The three major drug trials were the WHO Clofibrate Trial, LRC-CPPT, and the Helsinki Heart Study.

Presenting the strongest evidence to date for a beneficial effect of diet modification is the Oslo Heart Study or Oslo II.[116] In this study, members of the experimental group were given personal instruction on a low-fat diet and smokers were advised to quit. The results of this five year randomized trial revealed a reduction in total ischemic heart disease events of up to 47%; a reduction attributed to a decrease of 13% in plasma cholesterol and a 45% decrease in smoking. In an attempt to estimate the independent effects of cholesterol reduction and smoking, a multivariate statistical model was used to separate the effects. The model predicted that 60% of the reduction in CAD was attributed to the cholesterol reduction, while only 25% was explained by the reduction in smoking.

The American Multiple Risk Factor Intervention Trial (MRFIT)[4] was designed to test the effect on the incidence of CAD of interventions directed toward the reduction of serum cholesterol, cigarette smoking, and hypertension. While the National Diet-Heart Study[117] demonstrated that serum cholesterol could be lowered by fat-modified diets, no single clinical trial of diet modification had significantly reduced the rate of occurrence of CHD.[34] MRFIT was one of two major clinical trials designed to help answer the

question- should individuals undertake and maintain a dietary program to reduce serum cholesterol levels? In MRFIT, dietary counseling for cholesterol reduction was combined with vigorous attempts to control blood pressure and eliminate cigarette smoking in 12,866 high risk men aged 35-57.[4] The nutrition intervention specifically promoted a diet of no more than 10% of calories from saturated fat, 300 mg cholesterol per day and at least 10% of the calories from polyunsaturated fats. Three years into the study, the dietary advice was modified to recommend that saturated fat make-up only 8% of the diet and dietary cholesterol be reduced to less than 250 mg/day. The immediate goal of MRFIT was to obtain adequate reductions, through intervention, of the three modifiable CHD risk factors. While this large and complex trial was operationally successful, the overall results did not show a beneficial effect on CHD or total mortality.[118] Thus MRFIT ultimately did not provide a positive test of the hypothesis that interventions to modify diet and other risk factors would reduce the risk from ischemic heart disease.

For those interested in a detailed analysis of the differences between the inconclusive results of the MRFIT study and the successful Oslo study, the commentary by the Oslo Study Research Group[119] is recommended.

The UK[120] and Belgian[121] Heart Disease Prevention Projects like the North Karelia Study[122,123] are difficult to interpret due to the multiple nature of the interventions.

Interventions which were similar to those in the U.S. MRFIT, attempting to modify serum cholesterol, smoking, and high blood pressure. In the Belgian study, while they reported significant reductions in CAD incidence and mortality, how much of that reduction was attributable to cholesterol reduction was difficult to determine. Then once again, in both the UK and the North Karelia Studies, the final analysis showed that the intervention groups or counties fared no better than the controls.

Drug. Unlike the trials involving dietary modification, trials relying primarily on drugs to lower cholesterol levels have for the most part shown larger cholesterol reductions and are able to attribute decreases in CAD directly to the cholesterol reduction.

One of the largest and earliest trials was the World Health Organization (WHO) Clofibrate Trial.[124,125]. This three-center project found that after the first five years of the study that cholesterol levels were 9% lower in the treated groups and that there was a significant 20% decrease in the incidence of CAD. Unexplainably, there was not a significant difference in CAD deaths and the mortality rate from all other causes was higher in the clofibrate-treated group. As reported later,[125] the increased mortality rate in the treated group appeared to decrease to that of the control group after treatment was stopped. This has led scientists to comment that, "lowering cholesterol with clofibrate is difficult to recommend."[110]

Still another cholesterol lowering drug was tested during the Lipid Research Clinics Coronary Primary Prevention Trial (LRC-CPPT). The LRC-CPPT, unlike the WHO Trial, has in the opinion of many provided conclusive evidence of the benefit of cholesterol reduction in the prevention of CAD.[5,6,48,110] This study can not provide direct evidence of the effect of diet on the incidence of CAD, since in the LRC-CPPT study both the intervention and control groups were prescribed a diet to reduce cholesterol levels.[5] The diet prescription was relatively mild, calling for 400 mg of cholesterol each day and a P/S ratio of 0.8. Adherence to diet was assessed by means of semi-annual 24 hour recalls. Dietary adherence was determined to be the same for both groups throughout the study. Both groups were consuming a diet of about 38% fat at the beginning and at the end of the trial. However, the P/S ratio changed from 0.5 at the entry screening to 0.7 after diet counseling, which was reflected in decreases in TC of 11.1 mg/dl in the cholestyramine group and 12.6 mg/dl in the placebo group prior to the beginning of the trial. The decreased cholesterol values in the placebo group failed to provide evidence that dietary intervention alone would be successful in lowering CAD.[126] Two significant results of this trial that tested the effect of cholestyramine, a bile-acid binding resin that lowers serum cholesterol levels, were; 1) the treated group had a statistically significant reduction in the rate of development of CAD, and 2) these results

showed a "dose-response" relationship- the greater the reduction in serum cholesterol level, the greater the reduction in risk.

Specifically, the LRC-CPPT Study[5,6] involved 3,806 men, aged 35-59, with initial cholesterol levels greater than 265 mg/dl. All men were followed for an average of 7.4 years. The primary end-point was the combination of definite CHD death and/or definite nonfatal MI. The results revealed that treatment was associated with an average cholesterol fall of 8.5% beyond diet, and an average 19% reduction in CHD risk. Interestingly, more violent and accidental deaths occurred in the treated group. The researchers also claimed that there was an additional reduction in risk associated with an HDL-C increase of 3%, independent of LDL-C. The final conclusion was that, "the LRC-CPPT results give a clear and consistent picture of the relationship of its primary end point, CHD incidence, to changes in cholesterol levels."[5] The results of this study would most likely be even better if the participants had not been blinded and had been told their cholesterol levels and knew that their efforts were effective. This study is not without its critics.[126,127] One of these, Dr. Kronmal, wrote that "the LRC-CPPT was by proper standards, a statistically insignificant study that did not eliminate the null hypothesis."[126] He based his claim on the fact that the minimum test of significance agreed to at the beginning of the study was not used at the end, suggesting that it was

relaxed after the study only to show significance. Additionally, like the WHO study, the LRC-CPPT also did not show a reduction in total mortality.[127]

Helping to diffuse the criticisms mentioned above was the Helsinki Heart Study.[7] This was a primary prevention trial, using Gemfibrozil, randomized, double blind, and carried out over 5 years. The findings showed a 34% reduction in the incidence of CHD (95% confidence interval: 8.2-52.6) and a 26% reduction in CHD deaths due to an 8% decrease in cholesterol, 8% decrease in LDL-C and an increases of 10% in HDL-C. This works out to a 4% drop in risk for each 1% reduction in serum cholesterol. Unfortunately, as in the LRC study there was no difference in the total death rate due to an increase in the number of accidents and violent deaths. No reason for this finding was advanced.

Secondary Intervention Trials

Overview. While primary prevention receives the majority of the emphasis, secondary prevention of CAD received the earliest attention. While the early studies were really not experiments designed to test the lipid hypothesis, this area of investigation has over the years become more sophisticated, yielding information that has lent support for the diet-heart hypothesis. For a review of this research an article by Stallones[65] is recommended.

Diet. While some reports of secondary prevention trials appeared in the 1950s and early 1960s, the first serious trial would be Bierenbaum 's study.[128] His study reported on the 10 year follow-up of 100 men, aged 30-50, that after suffering heart attacks had been assigned to a 28% fat diet with either a P/S ratio of 2.6 or 0.3. When compared to a belatedly assembled control group, they found that the groups assigned to either diet had greater reductions in serum cholesterol and suffered fewer fatal heart attacks. While this study, like the Anti-Coronary Club investigation, was criticized due to not being a well controlled study with a preselected control group, it certainly encouraged further research in this area.

Many secondary diet studies have not supported the "lipid hypothesis". The results of the London Medical Research Council's Studies[129-133] did not find a significant effect of diet on the relapse rate, that is the number of individuals experiencing a reoccurrence of heart disease. The Oslo Diet-heart Study (Oslo I)[112,134] which was very similar in design to the London studies reported in a five-year follow-up that there was a significant reduction in CAD relapse rate in the diet group. Later in the eleven year follow-up, Leren[135] reported that a significant reduction in myocardial infarction mortality was evident in the diet group. Unfortunately, overall survival rates for the diet and the control groups were not significantly different. Still, Oslo I revealed that CAD mortality was correlated

with the Framingham risk factors: age, serum cholesterol, blood pressure, body weight, and smoking habits.

Another method of measuring success of an intervention is to actually measure the lesion growth or regression in the coronary arteries of a patient. One study that did this, using only dietary treatment to lower lipid levels was the Leiden Intervention Trial.[136] A vegetarian diet (P/S=2.0) was prescribed for patients with definite CAD and after two years they found that there was no lesion growth in 18 of 39 patients. It was also determined that coronary lesion growth was strongly correlated with the TC/HDL-C ratio. No coronary lesion growth was observed in patients who had a TC/HDL-C ratio less than 6.9. The lack of a control group makes it difficult to interpret the results of this intervention.

Drug. There was no difficulty interpreting the results of the Cholesterol-Lowering Atherosclerosis Study (CLAS).[137,138] This was a well designed study that included a control group. A 22% fat diet was prescribed to these middle-aged men, 40-59, with previous bypass surgery. The experimental group that then received Colestipol-Niacin therapy for two years were found to have lowered their TC by 26%, LDL-C by 43% and increased HDL-C by 37%. This led to a significant reduction in the number of lesions per subject and some regression of atherosclerosis. In the diet only group, LDL-C decreased only 4% and HDL-C did not change.

This study gave tremendous support to the efficacy of lowering serum cholesterol.

Additional support for the Diet-Heart hypothesis was gained from the long-term follow-up of the Coronary Drug Project.[129,140] The results have shown that the treated group had a significant decrease in overall morbidity and mortality, compared with the placebo group, in a long-term follow-up of men treated with nicotinic acid after myocardial infarction.

While mortality is the major endpoint examined in many of these studies, morbidity is another aspect that deserves mention. Recently, the MRFIT Research Group[141] published a report clarifying the result of their trial. These workers revealed that logistic regression analysis showed that the number of first major CHD events was reduced by the cholesterol and cigarette smoking interventions. Even more interesting was that, "all other cardiovascular endpoints specified at the outset of the trial, with the exception of stroke, occurred less often in the special intervention than in the usual care men." The results were statistically significant for angina pectoris, peripheral arterial disease and congestive heart failure. Similar reductions in morbid events (non-fatal CAD) were reported for the LRC-CPPT study.[5] These findings have not received much attention, but they are important because these cardiovascular endpoints occur throughout the population and not only degrade the life of the stricken individual, but require

large expenditures of medical resources. Fries[31] looks at the benefits to the individual and society from what he calls "the compression of morbidity", and claims that these benefits alone should justify nationwide focus on prevention. In any case, they add to the case supporting life-style modifications as a means of reducing/preventing CAD.

In summary, the studies reviewed above support the general conclusion that lowering TC and LDL-C levels will reduce the subsequent incidence of CAD events. Also, the pooled analysis of clinical trial findings suggests that intervention is as effective in secondary prevention as it is in primary prevention. For an excellent verbal and visual illustration of the effectiveness of 12 primary and secondary cholesterol lowering trials, see the Lipid Research Clinic's paper discussing the relationship of reduction in incidence of CAD to cholesterol lowering.[6] The direct evidence from clinical trials is strongest in middle-aged men with high initial cholesterol levels. However, the complete set of evidence, including the epidemiologic observation and animal experiments, strongly supports the generalization that reducing TC and LDL-C levels is also likely to reduce CAD incidence in younger and older men, in women, and in individuals with more moderate elevations of cholesterol.[142]

Dietary Guidelines

Just as our understanding of CAD has developed incrementally, so have the dietary recommendations for its prevention. The research of the 1950s led many scientists to believe that dietary fat and cholesterol had a probable role in the development of CAD. In line with this understanding the American Heart Association (AHA) issued cautious recommendations in 1961[143] that counseled weight reduction for the obese and dietary modifications for individuals with hypercholesterolemia. No general recommendations were made for the entire population. The Nutrition Committee of the AHA through three revisions of its basic statement[144] published in 1965, 1968, and 1973 gradually strengthened their recommendations. In 1965, the whole family was advised to make dietary changes which included substitution of polyunsaturated for saturated fats and a decrease in dietary cholesterol. In the 1968 revision, fat and cholesterol intakes were quantified. Americans were advised to maintain desirable weight and to restrict intake of fat to 30-35% of total calories, with a distribution of 10% saturated, 10% polyunsaturated, and 10% monounsaturated fats. A cholesterol intake of no more than 300 mg/day was suggested.[145] The 1973 statement was basically the same as the 1968 statement and then in 1978, a cautious recommendation of a "prudent diet" for all Americans was made.[146] The basic ingredients of the "prudent

diet" were, calories to maintain ideal body weight, fat calories at 30-35% with no more than 10% from saturated fat or from polyunsaturated fat, and again, a reduction in dietary cholesterol to less than 300 mg/day was recommended. There was however, not general agreement with this pronouncement. In a 1980 report, titled Toward Healthful Diets,[147] the Food and Nutrition Board of the National Research Council stated, "that it does not seem prudent at this time to recommend an increase in the dietary P/S ratio except for individuals in the high risk categories." Taking the opposite stand, 1980 saw the AHA publish another update to its recommendations.[148] This time they proposed that the population-wide adoption of the "prudent diet" should definitely be promoted. Still there remained individuals and groups that did not agree, and prior to 1984, even the National Heart Lung and Blood Institute (NHLBI), sponsors of the LRC-CPPT, believed that there was insufficient scientific justification for a concerted national cholesterol education effort.[149] Then in January 1984, with the publishing of the LRC-CPPT findings, summarized above, the NHLBI began to develop plans to ensure the widest possible dissemination of the LRC-CPPT results. Included in the Institute's plans was a national education program.

The publication of the LRC-CPPT results also prompted the National Institutes of Health (NIH) to hold a consensus conference to review the connection between blood cholesterol reductions and heart disease. The NIH reasoned

that in light of the total body of evidence supporting the benefit of lowering elevated blood cholesterol levels, there was a need to develop and refine treatment guidelines. The consensus, published as The Statement of the NIH Consensus Development Conference on "Lowering Blood Cholesterol to Prevent Heart Disease"[10] in December, 1985 included this key recommendation: "all individuals in this country beyond the age of 2 years should be advised to follow fat-modified diets in the interest of preventing coronary disease regardless of their sex, their age, or the presence of other risk factors". This added even more support to the NHLBI's National Cholesterol Education Program (NCEP) begun in November 1985. All of the NCEP's development activities were directed toward the dissemination and public acceptance of four basic tenets about cholesterol, namely 1) There is a clear association between cholesterol and CHD, 2) cholesterol is easy to measure, 3) everyone should know their cholesterol level, and 4) individuals with elevated blood cholesterol levels should do something about it.[149] After the NHLBI's report, the dominant opinion became that a "prudent diet" should be promoted and that the primary focus should be on the use of diet as the first line of treatment for individuals with moderate or high risk cholesterol levels.[10]

During the years since the beginning of the NCEP, the program has been scaled back and much of the initial enthusiasm lost. In October, 1987, the NHLBI announced the

start of a national cholesterol campaign designed to change American eating habits, a campaign that had been reduced in scope from that originally envisioned in 1984. Why? First, when it was discovered that Dr. Charles Glueck had apparently falsified some of his research with children, the American Academy of Pediatrics refused to endorse a fat restricted diet for children and young people.[150] Also, critics noted that the diet recommendations were extrapolated from only white, middle aged men and that there were frequent reports of increased total, noncardiac mortality in individuals involved in diet/drug trials.[151] As a result, the NCEP has modified the dietary recommendations. Individuals under 20 are now not included and the emphasis is on the treatment of "at risk" individuals.[142,127,152] This treatment in most cases should be diet, since it has been estimated that only about 5% of the population may actually need drugs.[8]

The public health strategy has, however, not been abandoned. The AHA has continued to present the public with dietary guidelines for healthy American adults.[153,154] In their 1988 statement,[155] the AHA notes that they have published guidelines for the diagnosis and treatment of hyperlipemia that were revised to be consistent with the guidelines of the NCEP. Then they offer a set of dietary guidelines for all healthy American adults, designed to help prevent heart and vascular diseases.[154] These guidelines are basically the Step-One Diet used for dietary treatment of

high blood cholesterol levels[142] and are really little changed from the 1978 guidelines.[148]

Dietary Guidelines:

1. Total fat intake should be less than 30% of calories.
2. Saturated fat intake should be less than 10% of calories.
3. Polyunsaturated fat intake should not exceed 10% of calories.
4. Cholesterol intake should not exceed 300 mg/day.
5. Carbohydrate intake should constitute 50% or more of calories, with emphasis on complex carbohydrates.
6. Protein intake should provide the remainder of the calories.
7. Sodium intake should not exceed 3 g/day.
8. Alcohol consumption should not exceed 1-2 oz of ethanol per day.
9. Total calories should be sufficient to maintain the individual's recommended body weight.
10. A wide variety of foods should be consumed.

As a result of the recommendations of the NCEP's Subcommittee on Dietary Treatment,[156] most health care professionals are using the AHA dietary guidelines detailed above, coupled with a pragmatic clinical approach.[78,157] For a historical look at the development of this position and the opposing viewpoints, see the articles by Stallones[65] and/or Wadden & Brown.[158] Hegsted[159] perhaps offers the best advice when he states, "that the dietary recommendations must consider food habits and be practical...and they need

to be presented in ways that are understandable and that can be put into practice by the consumer..."

Recommendations for Patient Care

While the debate over who should be treated and how to treat them continues, a consensus seems to be developing among both the national and the international medical communities.[156] They are adopting a practical approach to the management of high blood cholesterol. This approach is clearly detailed in the NCEP's Report on the Detection, Evaluation and Treatment of High Blood Cholesterol in Adults.[83] The program calls for the screening of all individuals for high blood cholesterol levels and treating those individuals at risk. The NHLBI, through its Consensus Conference on Lowering Blood Cholesterol to Prevent Heart Disease and the initiation of the NCEP has given the nation a standardized identification and treatment paradigm which is detailed below.

The findings of the Consensus Conference that elevated levels of serum cholesterol are a major cause of CAD led them to develop guidelines for the identification and treatment of "at risk" individuals.[10] They defined moderate risk as serum cholesterol levels between the 75th and 90th percentiles and high risk as levels above the 90th percentiles as appropriate for the age of the individual. General education programs were also suggested for the entire population. The Consensus Conference determined

that, "the first step in the treatment of persons with high-risk and moderate-risk blood cholesterol levels is diet therapy and caloric restriction for weight normalization in the overweight."[10] Diet therapy was based on the AHA Phase I and II diets.[160] These recommendations were subsequently modified during the deliberations of the NCEP's Adult Treatment Panel. Their report provided practical guidelines to use in measuring and reducing blood cholesterol in adult patients. Earlier recommendations had been confused by the age stratifications and lack of a definite treatment protocol. Numerous papers have been published on the NCEP's treatment plan. I would recommend reading the Panels actual report,[83,142] and then several of the papers that describe the effect of the recommended practices on individual practitioners.[99,152,156]

The need for standardized recommendations became apparent after surveys revealed that many physicians were not basing their patients treatments on the most current research information.[9,160] The new treatment recommendations for adults (men and women) use only three total cholesterol levels to define risk and then makes specific treatment recommendations over definite time intervals. Total cholesterol levels less than 200 mg/dl are defined as desirable, levels of 200-239 mg/dl are considered borderline-high, and levels of 240 mg/dl and above are considered high.[83] Recommended follow-up is based on the presence or absence of definite CHD and/or CHD risk factors.

See the Report of the NCEP's Expert Panel for the details of the treatment programs.[83]

Nutrition Education and Coronary Artery Disease

Theory

Nutrition education is atypical in that its focus is on behavior, not on just the mastery of information. This tells us that designing, implementing and carrying out a nutrition education project will require an extensive background in educational and behavioral change theory. In fact, in one paper that gave an example of how to choose and use a theory in nutrition education, the authors claimed to have reviewed studies in, "anthropology, behaviorism, cognitive psychology, education, human development, and social psychology in addition to the nutrition literature."[161] Obviously, this review will not be able to examine such an expansive field. However, when examining education theories, one must remember that individuals process, store and act on information presented to them according to a set of complicated rules that must be accounted for when designing programs of study. As educators, we must "understand the major processing mechanisms and design information inputs that can be effectively processed."[162] Individuals lacking a background in basic educational or learning theory will find many excellent texts on the subject. I would suggest a review of Bloom's taxonomy of educational objectives, cognitive

psychology, behavioral and neo-behavioral research, and then perhaps an examination of developmental and learning theories proposed by authors such as Lewin, Hull, Piaget, Kohlberg, Skinner, Jung, Bandura, and Maslow.[163-168]

Johnson and Johnson[169] reported on the results of their meta-analysis of 303 nutrition education studies in a recent supplement to the Journal of Nutrition Education. Their analysis attested to the efficacy of nutrition education. They showed nutrition education to have positive effects on knowledge, attitude and behavior. The major failing of nutrition education in their opinion was that it was often not based on theoretical models. Their opinion was supported by Nitzke and Athens[170] who found that only 19% of the 157 research projects in their study included theory in study designs. This failing has become the focus of present day research in nutrition education. Now many projects are in fact including theory in their design. This is important because according to Achterberg et al.,[161] "theory is the set of principles that tells us why concepts are related to each other." In other words theoretical concepts attempt to not only predict the outcome of a project, but to also explain the complex interactions that led to that outcome.

Theories may in fact be inherently transient,[161] but there is a core of theories that are frequently used in the design of nutrition education projects. According to Nitzke and Athens[170] the theories most frequently reported are related to meaningful learning, reasoned action, social

learning, and communications. In addition, the Health Belief Model, which was originally formulated to explain preventive health behavior,[171] deserves mention in this review, as does the Model of Acceptance of Change,[14] since this is the model upon which my research project is based. While there is often no distinction made between theories and models, there is a definite difference between them that should be understood. As Gillespie[172] points out, "while theories make useful contributions to nutrition education, sometimes it is difficult to apply them to a particular program." This is where the model comes in, since the model is in effect the application of a theory.

Meaningful Learning. Ausubel has looked extensively at the process of learning and has developed approaches that depend heavily on cognitive structures. His research has led to what many call a theory of meaningful learning which focuses on how individuals learn and how they create and share meaning. For a detailed description, one should refer to Educational psychology- A cognitive view by Ausubel et al.[173] and A theory of education by Novak.[174]

Theory of Reasoned Action. A theory that is perhaps better known to most investigators is Fishbein and Ajzen's Theory of Reasoned Action,[175,176] which is basically a behavioral change theory. Their theory presents a conceptual framework for the study of the relationships between attitudes, beliefs, behavioral intention and behavior. This model claims that an individual's intention

to perform a given behavior is a function of attitude toward performing the behavior and normative beliefs about what friends and significant others think should be done, weighted by motivation to comply with those others. This theory must often be combined with others, since it has difficulty explaining why attitudes, beliefs and intentions are often not predictive of overt behavior.[177]

Social Learning Theory. Social learning theory is thought to have been developed by Bandura.[178,179] He had an extensive background in psychology and learning research. Social learning theory views learning as a reciprocal interaction between the individual's environment, cognitive processes and behavior. This view is often referred to as "reciprocal determinism". The basic techniques used are modeling, con[illegible]acting and self-monitoring. These techniques are very familiar to followers of the nutrition education literature. The large volume of research data that already exists, makes this theory appealing to use.

Communication Theory. This is not really a single theory, since it incorporates several theoretical approaches in an attempt to study and explain the effects of communications. As may seem obvious, the main focus of communications research has been on persuasion or changing people's attitudes and behavior through the spoken and written word. This theory claims that there are six behavioral steps in persuasion: presentation, attention, comprehension, yielding, retention and overt behavior.[177]

Gillespie has written an excellent review of communication theories[172] and has also developed a nutrition communication's model[180] that can mainly be applied to short-term interventions.

Health Belief Model. The health belief model was developed during the early 1950s[181] and is especially useful because it can be applied to health, illness and sick role behavior. It was originally produced to explain health-related behavior at the level of individual decision making.[171] One aspect relevant to this review is the models attempt to explain preventive health behavior. For example, why do people not currently suffering disease choose to take actions to prevent health problems? Models of health related behaviors have six main concepts that may explain health behaviors:[177]

1. Access to health services.
2. Attitudes towards health care.
3. Perceived threat of illness.
4. Knowledge about disease.
5. Social factors.
6. Demographics.

Out of the 14 or so models of health related behavior mentioned in several reviews,[182,183] only the health belief model has been used with any frequency over the last 30 years. Accordingly, there is a large stock of research data available for those wishing to examine this model.

Model of Acceptance of Change. The Agricultural Extension Service of the U.S. Department of Agriculture formulated a model of acceptance of change.[184] This model divided the process by which adults adopt new ideas and practices into five stages: a) Awareness, b) Interest, c) Evaluation, d) Trial, and e) Adoption. See the chapter on Planning Learning[14] for a description of each of the five stages. Since this model attempts to describe the relationship between knowledge, attitudes and behavior, it is especially appropriate for nutrition education research. Nestor[185] looks at the conceptual framework surrounding this model and concludes that most nutrition education programs are directed at only the awareness level of the model. It then becomes apparent that if we hope to design nutrition education programs through which we hope to change behavior, we must consider the interest, evaluation and trial stages.

The theories examined in this review have been the ones most commonly used in nutrition research. There are many more theories and models out there that have seen limited use or are just coming into use. These are all available for study and/or use in future research projects. For a recent review of theoretical models used in health education, both well-defined theories and multiple theory models, I would suggest that you start with Parcel's 1984 article.[177] Then for a review of the various teaching models available for your use, I would recommend Models of Teaching by Joyce and Weil.[186]

Teaching Adults

Another important area to consider when planning a nutrition education project is the differences between teaching adults and children. Dailey[187] states that, "andragogy, the art and science of helping adults learn, is based on self-directedness, experience, readiness to learn and problem centeredness." This then would be differentiated from pedagogy which Knowles[188] defines as the "art and science of teaching children." So pedagogy is the usual teacher centered method of instruction with which we are all familiar, designed to prepare children for adulthood.[189] Note that the definition of andragogy reveals why the USDA model to promote adoption of innovations is an adult-oriented model.[184]

From research on adult learning there has emerged a theory of adult learning.[13] This theory has five basic principles that form its foundation:

1. Motivation for adults to learn comes through the recognition of needs and interests that learning will satisfy.
2. An adult's orientation to learning is problem-centered.
3. Adults learn best through analysis of experience.
4. Adults need to be self-directing.
5. Individual differences among people require differences in style, time, place and pace of learning.

With these principles in mind, nutrition educators that take advantage of the research in adult education may find that their research yields greater results.

For more information on adult education, I would suggest reading M.S. Knowles' The Modern Practice of Adult Education.[190] In addition, several papers that provide a nutrition education perspective are recommended: Adults as Learners,[13] The Use of Adult Education In Teaching Nutrition[191] and for the relationships between learning theories and adult education, an article by Dubin and Okun.[192]

Types of Preventive Programs

Overview. Nutrition education has been defined as, "the process of imparting to the public knowledge aimed at the general improvement of nutritional status through elimination of unsatisfactory dietary practices, promotion of adequate food habits and better food hygiene, and more efficient use of food resources."[193] Helen Guthrie[194] charges that it is the responsibility of the nutrition educator to, "translate this information into terms that are meaningful to the consumer... and to develop effective means of communicating this information." I will not delve into the controversy concerning at which level this nutrition education should be delivered: a high risk versus a public health strategy in regards to the prevention of CAD.[19,23,108,195,196] Instead, this section of the review will

concentrate on the interventions that deal with identifying and treating "at risk" individuals, individuals that are being influenced by a national, mass media public health campaign that is currently stressing general dietary advice and knowing your cholesterol levels.[9,83] Under these circumstances, the nutrition educator provides nutrition education to one or more of the following populations- the community, the social/worksite group and/or the individual. While there is obviously considerable overlap, this breakdown based on the setting and/or primary target audience allows us to examine the type of activities that characteristically take place under each category and gage their success.

Community. Community health education programs designed to reduce the risk of CAD use multiple strategies to achieve their ends.[197,198] On the surface they appear to be public health media crusades. While the use of the mass media is important it is just one of the approaches that must be integrated in a community intervention.[12,199] Community interventions also draw on the community organization model, the communication-behavior change framework and the social marketing framework.[197,200] We also see behavior modification strategies being applied with increasing frequency to community health education . This does make intuitive sense, since many of the risk factors for CAD are mainly behavioral. For an excellent review of

over 25 community intervention projects see the article by Elder et al.[201]

The interest in community-wide intervention developed in part from a review of epidemiological data, and in part from the realization that practical experience has shown that CAD risk factors can be reduced effectively and safely at group and community levels by direct education, motivation, and skills instruction in the promotion of health.[202] Another consideration that supports community interventions is that individuals were more likely to maintain reductions in risk factors when they had the support of their family, worksite and community.[203] These realizations have led to the development, and implementation of several programs designed to reduce the risk of CAD at the community level.

The major community interventions have been the North Karelia Project,[122,123] the Stanford Three Community Study,[204,205] the Pawtucket Heart Health Program,[206-210] and the Minnesota Heart Health Program.[202] While the Pawtucket and Minnesota studies are not yet complete, results from Stanford and North Karelia have shown that media campaigns are effective in modifying dietary behavior.[158,203] In these projects we are talking about mass media as part of a coordinated community media campaign These findings are of special interest because formerly, mass media alone had rarely been shown to cause individuals to adopt new behaviors.[211] As in many nutrition education studies, gains

made during the course of the study are usually lost without continued follow-up. For example, six months after the completion of the Twin Cities Extended and Second studies, serum cholesterol levels had returned to their baseline values.[95]

Group. A large percentage of the patient education on CAD risk factors, and their modification takes place in the health care providers office on a one-to-one basis.[12,212] Even though this type of patient education may constitute the foundation of a preventive cardiology program, a lack of time makes it very difficult for the health care provider to provide in-depth instruction or follow-up. As a result, organized classes for groups of patients are becoming more common. These group classes are taught by a variety of instructors, ranging from physicians, nurses, and dieticians to lay instructors.[213] The group environment can range from therapy sessions to the familiar basic classroom and includes families, friends, fellow workers, or perhaps strangers with similar concerns.[214]

The group environment can play a very important role in helping individuals change personal habits.[159,215] For a review of group therapy, I would refer you to two chapters in Helping People Change.[213,216] The conclusion that, "group therapy, thus far, has been demonstrated to be at least as effective as individual treatment and in most cases, much less costly," may indicate an appropriate approach to increasing nutritional compliance in individuals at risk for

CAD. Group therapy is already used for weight reduction, and in many programs dealing with addictive behaviors with varying degrees of success being reported.[217]

Group sessions for low fat diets are shifting from strictly knowledge transfers to programs that are utilizing the social psychology literature, counseling theory, and validated education principles, in addition to sound nutrition practices.[218,219] This new direction demonstrates the desire to integrate knowledge, attitude and behavioral considerations into nutrition education. For a review of group dynamics and how to direct group sessions from a dietetics perspective, the chapter on the Group Process[220] is recommended.

There have been many group studies published that involve instruction on adopting low-fat/cholesterol diets. While many of these studies have involved artificial groups, that is individuals brought together specifically for the class because they share a common risk factor, family groups or groups of fellow workers contribute an additional factor- social support to the group environment.[202] Social support has been shown to be extremely important to maintenance of behavior after the group sessions have ended.[221] An excellent review of nutrition education for risk factor reduction was published by Glanz.[203] A number of group studies are reviewed and synthesized in this paper. While most of the interventions demonstrated short term success, it was repeatedly found that, "nutritional status declined

to pretreatment levels after termination of the counseling."[204,222,223] This again points out the need to make provisions for long-term follow-up in any nutrition education project.[224]

The worksite is a natural arena for group programs. The worksite provides the social support mentioned above, a site for efficient delivery of information, and the opportunity for long-term follow-up.[225] However, until just recently there has not been very much information available on nutrition programs conducted at the worksite.[226] Bruno et al.[227] reported on a nonpharmacologic behavioral education program designed to reduce serum cholesterol. This eight-week worksite program showed that the treated individuals had a significant 6.4% reduction in total serum cholesterol as compared with the controls and a significant increase in nutrition knowledge.

The remainder of the 1980s however, has seen an explosion of interest in worksite programs of all descriptions.[203] Much of this change is due to the fact that industry has discovered that it is cheaper to prevent disease then it is to treat it.[228] Also, since a majority of the population never seeks professional help, the worksite provides an excellent opportunity to contact these individuals.[229] For an overview of the current status of worksite nutrition programs, including a section on cardiovascular risk reduction, read Nutrition at the Worksite: An Overview.[230]

The literature has begun to show certain strategies that are consistently found in successful worksite wellness programs. For a review of this area consult articles by R. Feldman[231] and L. Frederiksen.[232] Additionally, in a recent review of the frequency of worksite health promotion activities[225] from a random sample of 2,400 private corporations, nutrition education comprised 16.8% of the activity reported. Nutrition education experienced one of the largest percentage increases over the last five years. In response to this increase in interest in worksite nutrition programs, a guide has been prepared to show companies the benefit of offering nutrition programs.[182] This guide is of interest to anyone interested in worksite nutrition programs because it looks at strategic planning, implementation and gives examples of 16 successful nutrition programs.

Cholesterol reduction at the worksite has proven to be successful. The L.L. Bean program[233] used cholesterol screening at the worksite to encourage high-risk individuals to participate in their health promotion program. In this study, eight months after the initial screening, the high-risk individuals showed a 14% reduction in cholesterol levels. This relatively simple program contrasts with a multi-level nutrition program designed to lower the cholesterol levels of employees that was started at the Pawtucket Memorial Hospital (PMH).[234] They reported a 10.9% reduction in serum cholesterol, six months after the initial

screening, for individuals that had had the opportunity to take part in a variety of educational programs and work in an environment designed to promote cholesterol reduction eg. low fat menu in the cafeteria. These two studies which on the surface appear to be successful, provide excellent examples of a major problem in group studies- drop-out.

In the L.L. Bean study, 70% of the high risk employees returned for the recheck, and in the PMH study only 66% of the "at risk" individuals returned for a second measurement. The reported cholesterol reductions must be interpreted cautiously, since they are only applicable for the population of "at risk" individuals that chose to return. Drop-out is a major problem in many nutrition education projects. For example, in weight-control programs, dropout rates from 27% to 95%,[222] 33.8% to 57.5%[235] and from 31%-82%[236,237] have been reported. In the National Diet Heart Study, they reported a 10% dropout rate by the one-year point,[95] which is much better than the 20% to 80% reported in other studies of the treatment of hyperlipidemia.[227,238,239] Interestingly, dropout rates for worksite programs are up to four times higher than the dropout rate for the same program conducted in a clinical setting.[240] Brownell et al.[235] feel that the no-cost, convenient enrollment for most worksite programs may encourage marginally motivated individuals to join, the same individuals that would be very likely to dropout when any demands were placed on them. Monsen and Cheney[241] suggest that the dropout rate in studies could be

reduced by addressing the topic during study design. The dropout question aside, even as we continue to see a variety of group programs gaining in popularity, individual nutrition counseling is still the principal format used for delivering nutrition information.

Individual. The customary method used to teach adults about nutrition is personal instruction.[152] What immediately comes to mind is a picture of the dietitian at the patient's bedside, but that is no longer the case. Today, the nutrition educator is confronted by a diverse population that has been advised or feels the need to obtain nutrition information in hopes of reducing their risk for a host of nutritionally related diseases. This instruction may take place in a variety of locations ranging from an office to the home. This individualized instruction is important because the physicians often lack the time to provide in-depth coverage of the area of concern. Also the quality and content of the instruction done in the doctor's office will vary widely.[12] Although as mentioned earlier, a group approach is often promoted, many educators feel that individuals require careful evaluation and individually tailored therapies.[196] This is especially true when attempting to help individuals modify their serum lipid profiles, in an attempt to reduce their risk of developing CAD.

While individual instruction may be combined with a group or community program, working one-on-one with an

individual is considered to be nutrition counseling[203] as opposed to nutrition education. Just as working with a group has its own peculiarities, counseling an individual has its own set of required skills.[242] In either case, the educational theories discussed earlier remain the same and must be carefully considered when planning a nutrition education program for an individual.

Bartlett[243] disclosed eight fundamental principles that would, "enlighten and guide future patient education research and practice." Two of the principles focused on individualization being very important and on personalized approaches being more effective than pamphlets or video tapes. This viewpoint is supported in a review by Glanz[203] that looked at nutrition education for low-fat diets. She concluded that, "educational programs that included interpersonal methods were more successful." A similar conclusion was reached by Mazzuca[244] in a review of 320 articles dealing with the therapeutic value of patient education in chronic disease. Interestingly, he points out that, "patients need to know less about the pathophysiology of their disease and more about integrating new demands into their daily routine." This observation meshes nicely with the expected role of the nutrition educator, who would do well to leave the presentation of medical facts to the health care providers.

In the past, nutrition educators have found that there is not really much of a difference between teaching methods

when the measure of success is change in knowledge.[215] However, when the desired outcome is behavioral change both short term and long term, the instructional strategy does become important. As Kanfer and Goldstein[213] point out, to affect lifestyle changes the educator must carefully consider which treatment strategy/therapist/patient mix would be the most appropriate. Caggiula[245] recently addressed this area when she looked at the reasons for the lack of adherence to nutritional therapies designed to lower serum cholesterol. Stating that traditional programs "primarily rely on information exchange", she goes on to address the behavioral aspects that must be considered and the role that the educator must play if patients are to comply with dietary instructions. A similar position is taken by Bassler et al.,[246] who point out the importance of individuals taking an active role in the selection of the diet strategy they will choose to follow.

Evidence of this change in focus for nutrition education can be seen in the recent literature. Brush et al.[218] published a study designed to look at the relationship between knowledge, attitude and behavior in an affective-based adult nutrition education program. The need to consider a variety of instructional approaches when designing nutrition education programs was pointed out by Chery et al.[247] Crockett[248] used Fishbein and Ajzen's Theory of Reasoned Action in a study of adult's attitudes about attending nutrition classes as did Shepard and Stockley[249]

when they examined the relationship between nutrition knowledge, attitude and fat consumption. Additionally, there has been an increase in the number of authors from the social sciences conducting nutrition research,[170] giving the field an infusion of new ideas. An example of which would be the publication of an article describing an information processing approach to nutrition education.[250] Slowly then, the choices available to the nutrition educator when planning counseling or group sessions are increasing. All of these changes are helping to speed the shift from a "diet instruction" to a "diet education" mode. This is a shift that must take place if the nutrition educator is to provide the dietary therapy called for in the NCEP[83] and thereby help individuals lower elevated levels of serum cholesterol. Effective dietary counseling is perhaps the only way to overcome the inclination of many patients to come to a physician and be given a drug, rather than take personal responsibility for their own health.[110]

Evaluation

Even when an intervention is based on the best research from the psychological, the educational and the nutritional literature, procedures must be included to determine the effectiveness of the program. This aspect of nutrition education is often overlooked.[12,235,251] A complete evaluation will include both a formative and a summative evaluation if possible.[14] For information on evaluation requirements and

procedures, see Implementing and Evaluating Learning,[252] Evaluation of Nutrition Education,[253] and Educational Evaluation.[254] These sources will provide an excellent overview of the evaluation process.

One aspect of evaluation that I would like to look at in a little more detail is the use of serum cholesterol levels for diagnostic purposes and as an indicator of compliance with a low fat diet prescription.[255] While it is generally accepted that use of serum cholesterol values are valid for these purposes, there is some concern over the accuracy of these measures.[83] The Lipid Research Clinics Prevalence Study[256] investigated the retest reliability of plasma cholesterol and triglycerides and reported that it was not uncommon to see apparent cholesterol changes of 50 mg/dl and apparent random triglyceride changes as high as several hundred mg/dl. The results of their study suggested that the coefficients of variation were 8% for cholesterol and 25% for triglycerides. The data from their report has been interpreted to mean that for total cholesterol, the standard deviation of repeated measurements in an individual over time is 18 mg/dl.[256] For further information on the problems involved with cholesterol measurements, read Measuring Cholesterol is as Tricky as Lowering It.[257]

Since the problems with cholesterol measurement revolve around individual variation and inaccuracy in cholesterol testing, any study using serum cholesterol in its evaluation should include estimations of the precision (coefficient of

variation) and accuracy (bias) of the laboratory that performed the cholesterol analysis. As a guide, in the report from the Laboratory Standardization Panel of the NCEP,[258] they are looking for a bias of no greater than ±3%, and a CV of 3% or less. This information is important because if you had a client with an initial cholesterol level of 250 mg/dl, that reduced his cholesterol by 10%, the change may not be detected in a lab with poor precision.[258] Labs are able to maintain strict standards, as is evidenced by results achieved in the Air Force HEART Study.[259] They were able to maintain a bias of less than 1.2% and a CV of less than 0.7%. Not all laboratories are this accurate, so it is very important to consider the variation in cholesterol measurements when planning and evaluating studies. The standard deviation of repeated measurement is especially important when determining the sample size for a study.[260]

Self-Paced Nutrition Education Programs for Cholesterol Reduction

The question "how can nutrition education be delivered in the most effective way possible?" will receive a variety of answers.[211] Often, the choices are limited by the situation. Money, time, lack of trained instructors, motivation of clients, etc. all have an influence on the type of program that can be offered. This section will examine various aspects that should be considered prior to

choosing to use a self-paced nutrition education program for management of serum cholesterol.

In Relapse Prevention[217] Marlatt and Gordon compared the consumption of a diet high in fats and cholesterol to addictive behaviors such as smoking, and alcohol and drug abuse. If we think of the consumption of a high fat and cholesterol diet as a form of addictive behavior, then from a social learning perspective which claims that everyday behavior often consists of a chain of responses, we are dealing with a "bad habit."[217] As with many "bad habits", we can approach them using basic psychological principles that control behavior and behavior change. While in the past, attempts at behavior modification viewed the client as a passive participant in the effort, today, with our understanding of adult education, the adult client is seen as the one actively seeking a "solution" to his or her problem. The demise of the passive, accepting, and trusting client has reveled an individual that will accept responsibility for his or her own change program.[14] This type of individual is also one that adopts change along the lines of the acceptance of change model.[242] For a complete description of self-management and the role played by self-control in behavior change read the chapter on self-management by Kanfer.[261] While the theoretical basis may exist for a self-care program, is there any reason to implement one?

As discussed earlier, the problem of dropout from nutrition education groups was a significant factor in some studies. However, do we know much about the characteristics of the individuals that seem to drop out of or decline to participate in group studies? Research has shown that they may be individuals with jobs that require travel and, as a result, are not likely to attend classes regularly.[239] When the class is not located within a convenient distance from the individual's home, dropout is also probable.[15] Then there are individuals who just do not like to attend courses or are unwilling to talk about their health problems in front of others.[222] Also, since adults are oriented to solving "their" problem, they may not want to sit through counseling sessions or group meeting that are not focused on their particular needs. Other adults expect a class on healthy eating to make them feel guilty about their eating patterns, and so they are reluctant to attend.[248] In Control Data's Stay Well program,[15] the answer to concerns and situations such as those above was to develop self-study lifestyle change courses. While Control Data has not evaluated these self-help programs, others have been effective.

Behn and Lane[222] reported on the use of a self-help weight-loss program which combined nutrition education, behavioral modification, and self-control techniques. They found that for individuals unable or unwilling to attend classroom sessions, the self-help approach achieved results

similar to the classroom/manual method. Stritter et al.[262] showed that self-instructional materials that stressed frequent feedback and practice produced significantly higher test scores than the traditional lecture method of instruction. Buller[263] prepared a dietary education program for patients with hyperlipidemia that included an instructional booklet and the opportunity for free telephone consultation with a dietitian. The results showed that the program was successful in changing both attitude and behavior. Nowlin and Shortridge[264] reported that their self-instruction program which was developed because of a shortage of instructors, was as effective as the leader-led program in enabling adults to follow nutrition plans they had made for themselves. Another positive aspect of this approach was that individuals had a "choice" as to which program to complete.

In many situations, the individual may not have a choice. As mentioned in the section on group instruction, many doctors, physician assistants, nurses, and dietitians operate under severe time constraints.[12] Also, many of the health care providers that also provide most of the nutrition education may not possess the depth of knowledge or the counseling skills needed to provide appropriate diet counseling.[203] This means that unless specific ancillary programs have been developed to give special attention to individuals with unique problems, for example elevated serum cholesterol, clients are not likely to get the in-depth

coverage of their problem or the personalized follow-up they may need. Even when the channels to provide special treatment programs are in place, there may be a lack of trained instructors (vacation, pregnancy, illness, turnover) or the referral rate may be so great that individuals must wait an unacceptable length of time prior to receiving instruction/counseling.[265] Exactly for these reasons then, a program of self-instruction would be an acceptable alternative to physician counseling or to provide an alternative to returning some time in the future for specialized counseling or for a group session. In fact, Craighead et al.[265] suggest that self-help programs "may be used as screening devices to provide more cost-effective treatment... and that the persons response to the self-help program could be used to suggest appropriate further interventions."

The cost-effectiveness of self-help programs has not been addressed, but that is obviously one factor that should be considered.[266] Brownell et al.[235] discussed a number of weight control programs and concluded that the cost-effectiveness of work site weight reduction programs was good. One estimate was that for each 1% reduction in the percentage overweight, the cost was $3.00 for a self-help group compared to $44.60 for a university-based professional program. Behn and Lane[222] also pointed out the cost savings realized by using a self-teaching manual. In a study conducted by the Phode Island Group Health Association,

savings of $2.50 to $3.50 for every dollar spent, depending on the level of self-care intervention were reported.[267]

However, not all self-care programs have been shown to be cost-effective. Smith[268] has written an excellent article which looks at not only the costs of self-care programs, which by the way tend to be more expensive to develop than traditional courses, but lists five factors that should be considered when trying to choose between self-care and leader-led instruction. She incorporates these considerations into a questionnaire that can help one make the choice, and points out that there are circumstances where either type of instruction can be used.

For a complete review of the self-help area I would recommend Perspectives on self-help and bibliotherapy: You are what you read.[265] This review of written self-help materials begins with a look at how self-help approaches have developed, and includes an extensive look at self-help research in a variety of problem areas. As might be expected weight control programs are extensively reviewed. The summary provides support for the use of self-help in providing diet therapy. The authors conclude that self-help programs seem to be the most successful when they are designed for "a particular subtype of a problem or for a particular subset of the population." The reason for this being is that such programs give the appearance that they have been individualized for the client, just as a dietitian

would individualize a program, while they still retain the cost-effectiveness and convenience of self-help.

CHAPTER III

METHODS

Rationale, Research Objectives and Design

Rationale

Self-CARE for a Healthy Heart (Self-CARE). Self-CARE was designed to help adults reduce high cholesterol levels and thereby reduce their risk of developing CAD. It is a self-paced, nutrition education program that introduces the participants to eating pattern changes that it would be desirable for them to make. Implementation and evaluation of this project was carried out at two Air Force medical facilities.

The Air Force has a strong commitment to preventive cardiology, a commitment that was codified in January, 1982, when the Surgeon General of the Air Force published Air Force Regulation 160-18, establishing the Air Force Coronary Artery Risk Evaluation (CARE) Program.[269] The CARE program "identifies active duty Air Force personnel who are at increased risk of developing CAD and uses a multidisciplined approach to reduce that risk through counseling, education, therapy, and follow up evaluations." At the facilities used in this study, retired military members and dependents are not directly covered by the CARE regulation, but when they

are found to be "at risk", their treatment is no different from that given active duty patients.

The local Medical Facility Commander implements the CARE program and establishes a follow up evaluation program for all personnel at increased risk, who voluntarily enter intervention. The Flight Surgeon's office is usually responsible for the CARE intervention and conducts the health education programs. In my opinion, one program of particular importance is the cholesterol intervention program.

An individual that has been identified as at increased risk for CAD because of elevated TC or a TC/HDL-C ratio greater than 4.5, after counseling, by a credentialed health care provider (CHCP) may choose to participate in the cholesterol intervention program. A CHCP is a physician, physician's assistant, or nurse practitioner. Once the individual requests intervention, my observation was that in the military facilities along the Front Range, there is not a "usual" program or approach. Since each program is administered at the "base" level, the cholesterol intervention may be managed by a registered dietitian, a diet technician, or a registered nurse. Additionally, the constant turnover of personnel or absences due to pregnancy, vacation, or temporary duty at another location, leaves the intervention program at the mercy of whomever is available and as a result the programs may range from cursory to very thorough.

Successful programs are not recognized because the CARE program has no built-in evaluation system. Since there is not a standard with which to gauge the success or failure of the program, I was led to ask, "are the present cholesterol interventions effective?" Do they in fact reduce CAD risk by lowering serum cholesterol levels, raising HDL cholesterol levels or improving the TC/HDL ratio?

After reviewing the intervention programs at several facilities and consulting with the physicians in charge of the intervention programs, I concluded that there were two main problems. First, it was very difficult to assess the effectiveness of an individual program because of the lack of a comprehensive evaluation policy. Second, the numbers of "at risk" individuals choosing intervention place such a large workload on the system, that it is difficult to provide effective follow-up care.

Similar concerns were voiced in the only recent study carried out in an Air Force clinical setting.[212] At the conclusion of this study, Dr. Whitney who had developed a preventive cardiovascular risk reduction program, that had to deal with a large volume of patients and limited health professional resources, stated that "the backbone of any preventive cardiology program is patient education, which unfortunately is not easily accomplished in a physician's busy office." He goes on to point out that "one on one" counseling with a patient is prohibitive both in terms of time and money.

The proposed study will then serve a dual purpose. First, it will assess the effectiveness of a facilities "usual" intervention program (usual-care). Then it will determine if the self-paced nutrition education program, Self-CARE, provides an acceptable alternative to the usual-care, an alternative that would promote a more efficient use of the health care resources, addressing the concerns voiced by Dr. Whitney.

I chose a self-care approach for two reasons: 1) the time, money, personnel and/or the planning required for group sessions or one-on-one counseling is not an effective use of the available resources;[239,263,270] and 2) the typical individual identified as at risk is an adult, usually working full time, often at a job that requires a lot of time away from home or shift work. This type of individual traditionally is not likely to take part in organized group programs due to time constraints, travel, social stigma and/or motivation.[236,237,239,248] Even when these individuals do choose to participate, they are likely to drop-out. However, they are likely to complete a self-study program.[239]

While I feel that SELF-CARE may be an attractive alternative to the current programs, implementing this program is not the main focus of my project. Since evaluation is critical to determining the actual success of any project, the real substance of this study involves the ability to critically evaluate the results. This has been the major shortcoming of many of the previous work site

nutrition education programs.[230,241,271,272] Self-selection for participation and high dropout rates, in particular, often make assessment difficult.[15] Accordingly, the experimental design of this study is very important and it must insure that we are able to 1) evaluate the success of usual care, 2) evaluate the results obtained from using SELF-CARE and 3) compare the two programs.

Research Objectives

The specific goals and objectives for the SELF-CARE study are as follows:

1. To assess the effectiveness of "usual care" in modifying serum cholesterol levels at the two medical facilities in this study,

2. To assess the effectiveness of the SELF-CARE nutrition education program in modifying serum cholesterol levels at the two facilities in this study, and to

3. To determine if the SELF-CARE program can be considered a viable alternative to "usual" care.

With these three goals in mind SELF-CARE was developed to test the following hypothesis:

Individuals following the SELF-CARE program will experience an average reduction in total serum cholesterol, and/or improvement in the total cholesterol/HDL-C ratio that is equal to or greater than the improvements provided by "usual care".

If this hypothesis proves to be true, then the SELF-CARE program, because of its simplicity, low cost and

effective use of time and manpower, should be considered for expanded usage and long term evaluation.

Design

The specific design I have chosen to use is the "pretest-posttest control group design.[266] This design represents a true experimental design and can be described as follows:

R 01 Self-CARE Program 02

R 01 Usual-Care Program 02

Where the R's refer to the randomization of participants and the 0's represent the measurement of the participants. The program is designed to run for three months. The information collected at 01 will be sex, age, weight, height, TC, LDL-C, HDL-C, TG, and smoking status. At the end of the study, 02, weight and serum lipid values will again be collected. In addition, a diet worksheet will be completed and the % fat, % saturated fat, and % polyunsaturated fat in one day's menu will be determined. Each individual's prescription drug use, activity level, and consumption of oat products, alcohol and/or vitamins will also be recorded.

The research plan was approved on March 17, 1988 by the Committee on Human Research for Colorado State University and on April 19, 1988 by the Air Force Institute Of Technology. Copies of the project approvals are included in Appendix A.

Development of Materials

Self-CARE Package

The Self-CARE program was designed specifically to meet the needs of the adult learner, something that most nutrition education programs fail to consider.[187] Realizing that the adult learner is very practical, and wants immediate, useful information, Self-CARE was designed to dispense only essential knowledge, using practical examples, applications and analogies.[187] In designing the program, a list of problems to be considered was compiled as different articles were read and different classes on education completed. This list contained items such as motivation is critical to the success of the program, individuals have limited time and that humans are impatient by nature. Additionally, it was important to sift through the sea of information out there and think about what is really important to the individual with elevated cholesterol levels. This led me to realize that every effort must be made to narrow the focus of the program. Consequently, a single theme, "reduce your fat intake" was the main focus of this program . A single concept message was used because this type of approach has been successful in other nutrition education programs[273] and also, after reviewing a number of other nutrition eduction programs it became apparent to me that most of them provided so much information, that an individual not already trained in nutrition would be

overwhelmed. Since this program deals with changes in attitudes and is not just an information transfer, special attention was paid to the affective objectives of the intervention, in addition to the expected cognitive and behavioral considerations.

Steps. Each of the four "Steps" in the program were organized in accordance with the Model of Acceptance of Change.[184] This model contends that the process through which an adult will change behavior involves five stages: 1. Awareness, 2. Interest, 3. Evaluation, 4. Trial, and 5. Adoption or Rejection. Accordingly, each "Step" in Self-CARE attempts to create awareness, followed by an attempt to develop interest and to give the individual a chance to evaluate the new idea or concept. Once the new practice is evaluated, provisions for a trial period will hopefully lead the subject to permanently adopt the new practice. In addition, as the organization progresses, aspects of the Theory of Reasoned Action were integrated as I attempted to look at the relationship between attitudes, beliefs and behavior.[175] The Health Belief Model also contributed to this program by revealing the importance of social factors in promoting behavior changes.[177]

Specifically, page one, titled "WHAT CAN I DO?", develops "awareness" by introducing a new practice or idea using a quiz or example. Page two, titled "HOW CAN I DO IT?" is designed to increase the "interest" of the reader and help them to "evaluate" the suggested changes.

Specifically, this step compares the individual's present eating behavior with other healthier options and discusses changes the individual may want to make. Once the advantages and disadvantages of particular eating habits are known, the subject should be able to answer the question, "Can I do it?" Page three is titled "MORE THAT I CAN DO!" and provides the individual with the opportunity to examine the usefulness of the new practice on a "trial" basis. Finally, step four titled "FACTS...FOR SELF-CARE", is designed to reinforce the new diet habits the individual has "adopted". This step is especially important because the individual will not perform the new behavior(s) consistently, unless he or she is continually reinforced.

The organization outlined above served as the foundation of the SELF-CARE program. The information included in Self-CARE program was drawn from the Eating and Your Heart program[274] (used with permission- see Appendix B), the Pawtuckett Heart Health Program[275] (used with permission-see Appendix B) and the Family Foodstyle Nutrition Education Program[276] (used with permission-see Appendix B). The physical format, folder and inserts, was similar to that used in the Pawtuckett Heart Health Program and artwork from the Family Foodstyle Nutrition Education Program was used.

Specific cognitive, affective and behavioral outcomes were established for each "Step" of SELF-CARE program after seeing that most nutrition education programs dealt with

only knowledge transfers. The specific objectives for each step are as follows:

Step 1

1. Affective- The learner will become aware that a sensible diet based on the U.S. Dietary Goals is important in the prevention of lifestyle diseases.

2. Behavioral- The learner will use the Cardiovascular Disease Risk Chart to determine his cholesterol and lipid risk profile.

3. Cognitive- Given a list of coronary heart disease risk factors and a discussion of each, the learner will be able to list personal risk factors.

Step 2

1. Affective- The learner will be able to identify a need to reduce the saturated fat and the total fat content of his/her diet.

2. Behavioral- The learner will complete the "Eating Pattern Survey" which deals with the reduction of dietary fat intake. The learner will set a personal dietary objective.

3. Cognitive- The learner will be able to list the major sources of cholesterol, saturated, monounsaturated, and polyunsaturated fats. The learner will be able to recognize the sources of visible and invisible fat in his diet.

Step 3

1. Affective- The learner will be able to identify the need to increase the complex carbohydrate and fiber content of his diet.

2. Behavioral- The learner will complete the "Carbohydrate & Fiber Survey". The learner will set a personal dietary objective.

3. Cognitive- The learner will be able to determine whether a diet is providing the proper amounts of carbohydrate and fiber.

Step 4

1. Affective- The learner will understand the importance of including nutrient rich foods in his diet. The learner will become aware of the need to include exercise in a program designed to reduce cholesterol levels.

2. Behavioral- The learner will complete an analysis of two common snack foods in order to determine their nutrient density. The learner will set a personal dietary objective.

3. Cognitive- The learner will be able to define "nutrient density". The learner will be able to explain the relationship between exercise and weight control.

After the four steps were developed, the materials were reviewed (see validation of materials below), and with the assistance of a local graphic artist were prepared for printing. Copies of the four Self-CARE steps are in Appendix C.

Folder. The four "step" inserts and the materials listed below are contained in a folder. The simple pocket folder has a distinctive cover that utilizes the Colorado State University's Healthy Heart Program logo.[274] The inside flap was designed so that the individual's lab results could be listed. Headings for Total, LDL, HDL, Triglycerides, and TC/HDL-C ratio were printed. This feature not only provides the individual with a written record of his individual lab values, but the program participant is frequently referred to these values while completing the Self-CARE program. A copy of the folder cover is included in Appendix C.

Introduction Sheet. After the basic package was prepared, an introduction sheet was drafted that explained

why the program was designed and its basic organization. Basic instructions on how to approach the program were also included. One sheet was included with each package. A copy of this sheet is in Appendix C. These sheets could also be distributed individually for program promotion.

PRUCAL Diet Analysis . An integral part of the Self-CARE program is the PRUCAL diet analysis program.[277] Computer generated diet analyses were included so that each individual would have an idea of how their usual consumption compares to the prudent diet guidelines and how their current dietary practices compared to the recommendations.[194] The analysis also provides a means of maintaining contact with the subjects during the course of the study. This was important because it has been reported that self-instruction is not very successful unless periodic contact is maintained.[265] A four page insert on preparation of the PRUCAL Diet Analysis Worksheet was prepared. The first page reviews the purpose of the Self-CARE program and encourages the participant to complete each diet worksheet in a timely manner. The last three pages are an example of a PRUCAL computer print-out for a healthy heart diet. The example was included to reinforce the concept of a healthy heart diet and to serve as an introduction to the PRUCAL format. See Appendix C for a copy of this insert.

PRUCAL Diet Analysis Worksheet. An instruction sheet with blocks for personal information on one side, and a form on the other to record food intake for a 24 hour period was

prepared. This form was designed not only to record information, but to also serve as a reminder of the prudent diet guidelines. The worksheet was to be returned in the preaddressed, stamped envelope that accompanied the worksheet. A copy of the worksheet is included in Appendix C. Both the envelops and the worksheets were canary yellow. This was done so that participants would associate the yellow envelope with the Self-CARE program and not discard mailings.

Self-CARE Reference Sheet and Magnetic Logo. A SELF-CARE reminder sheet with magnetic logo was designed. The two and one-half by eight inch sheet contained some general reminders on how to reduce fat intake, and was designed to be personalized with the participants current cholesterol level, the individual's three month cholesterol goal and the date for a follow-up check. The three month cholesterol goal used for this study was a 10% reduction in total serum cholesterol. This was a reduction that could reasonably be expected to be achieved.[238] The sheet was laminated with plastic and a red, magnetic heart with SELF-CARE printed on it in white letters, attached with adhesive cement. A red marker was provide at each location to record personal information on the sheet. The reference sheet was designed to be attached to the refrigerator and serve as an additional source of reinforcement and reminder of the cholesterol recheck date. The magnetic hearts were

purchased from Magnetic Collectables, Ltd., Cape Girardeau, MO. A copy of the reference sheet is included in Appendix C.

Final Critique Form. A brief critique of the program is used to indicate program completion, reemphasize the main points of the program, evaluate the course and prompt the individual to complete the monthly PRUCAL analysis. This critique form was patterned after the one used in the Pawtucket program. A copy of the critique form is included in Appendix C.

Enrollment Forms

Computer generated enrollment forms were prepared. They were designed to collect the basic information that would be used in the analysis of this program. Two versions of the form were prepared. See Appendix D for examples of these forms. One version contained space for five individuals per page and was used where the individual received individual counseling and had a counselor available for assistance in completing the form. The other version had space for only one enrollment per sheet and included a example at the beginning of the form. This version was designed for group enrollments where there was not individual supervision of the enrollments.

Subject Information Sheets

Two Subject Information Sheets (A&B) were prepared, one for those enrolling in Self-CARE (A) and one for those

enrolling in usual-care (B). The sheets were basically identical, reviewing program goals and objectives, and the procedures to be used. The Self-CARE sheet, Subject Information Sheet-A, differed in that it attempted to reinforce program goals by making a brief introduction of the philosophy that the program stresses, that is the concept of the individual taking responsibility for his own diet decisions.[192,278] Copies of these sheets are included in Appendix D.

Consent Forms

A consent form was prepared and approved by the Colorado State University, Human Research Committee. The same form was used for all participants. A copy of the consent form is included in Appendix D.

Data Collection

Due to the variety of data, both personal and physiological, to be collected and analyzed during the course of this study, a computer file was designed. The design of this filing system allowed all suspense dates and data for a particular individual to be stored on one line. Each individual was identified by a number that represented the order of enrollment in the study. A similar form was used for individuals enrolled in Self-CARE and in usual-care. An example of a line entry for an individual enrolled in Self-CARE and in usual-care is included in Appendix D.

Collecting the data in this fashion allows direct transfer of the information to the computer for statistical analysis.

Validation of Materials

Preparation of materials for this study involved taking the best of several established programs, combining them in accordance with proven teaching strategies and preparing a package for actual use. The initial draft and evaluation of this project was completed as a project for a community nutrition class. Using the comments and suggestions from that endeavor, and consultation with the CHCPs at each military facility, a final draft of the Self-CARE package was completed. The package was reviewed by three registered dietitians and a specialist in adult education for content, clarity and utility. The final package design was then completed, and the graphic artist, Jean Comstock Graphic Design, prepared the final layouts which were reviewed by ten Air Force employees prior to the final printing and subsequent initiation of this study.

Implementation

Site selection

Site selection was an ongoing process throughout the planning and development stages of this project. Originally, all military facilities along the Front Range and in Cheyenne, Wyoming were considered for use in this study. Access to adequate numbers of patients and lab

analysis at no cost to the author were the features that made the military facilities attractive study sites. After initial inquiries, it became apparent that trying to use a mix of U.S. Army and Air Force facilities would not be practical. The effort required to gain approval for the study from both Services was not warranted, when sufficient subjects were available at the Air Force facilities. Plans to use Lowry AFB in Denver, Colorado were also dropped because their patients were treated at the Army hospital in Denver, not on the base. The final Air Force facilities selected for use in this project were the hospital at F.E. Warren AFB (FEW), WY. to be used for a pilot study and for the actual study, the hospital at the United States Air Force Academy (USAFA) and the clinic at Peterson AFB (PAFB), both of which are located in Colorado Springs, Colorado.

The study sites were excellent choices. The two facilities in Colorado Springs had a sufficient number of patients so that the study population could be enrolled within a four month period and the individuals enrolled were from the same geographic area eliminating a possible confounding factor. The individuals in charge of the cholesterol education programs were very supportive and extremely interested in discovering how successful their programs actually were. The laboratories at each facility use the same basic diagnostic procedures and are subject to the same quality control demands. Also, in addition to the major comparison of Self-CARE against usual-care, using

these two facilities allowed for the comparison of individual diet counseling versus group counseling, since at the USAFA a registered dietitian or diet technician counsels patients one-on-one, while at the PAFB clinic a registered nurse conducts weekly group sessions.

Size of Sample

From a statistical standpoint, this study was designed to test the following null hypotheses:

1. there will not be a change in the cholesterol levels for the individuals enrolled in Self-CARE or Usual Care over the course of the study and
2. there will be no difference in the level of cholesterol reduction between the Self-CARE and the Usual Care groups.

The sample sizes needed for an adequate test of these hypotheses were calculated based on the following considerations:

1) Coefficients of variation (CV) for repeated measurements of cholesterol have been reported in a number of articles.[256,258,279,280] The CVs were found to be close to constant across lipid levels[256] and a value of 8 per cent represents an appropriate value for planning purposes. The Laboratory Standardization Panel of the National Cholesterol Education Panel recommends that as a National goal, laboratories should initially achieve an overall precision consistent with a CV of 5% or less.[258] Cholesterol measurements from the Air Force HEART study consistently achieved an average day-to-day precision (CV) of <0.7%.[259]

Since the labs in this study are now using automated, enzymatic cholesterol measurement procedures similar to those used in the HEART study, the use of a CV of 8% for planning purposes should prove to be a conservative choice.

Based on a CV of 8% and an average entry cholesterol level of 260 mg/dl, a standard deviation (SD) of 20 mg/dl was used for sample size determination. This value agrees with that used in other studies measuring change in serum cholesterol in hyperlipidemic individuals[280] SD was calculated as follows:

CV= .08 = SD/260 mg/dl SD = .08 x 260 = 20 mg/dl

2) A difference to be detected of 13 mg/dl, based on detecting a minimum of a 5% change in cholesterol and an average cholesterol value of 260 mg/dl. δ = 13 mg/dl

3) A power of 90% (β = .10) and

4) A 5% level of significance (α = 0.05),two tail test.

Hypothesis #1 involves the comparison of paired data, since we are looking at data that involves the change in cholesterol level for a single individual. Sample size was calculated as follows:[281]

Power = .90

Level of Significance = 95% (α = .05)

$n = (Z_\alpha + Z_\beta)\ (\sigma_D^2/\delta^2)$ n= # of individuals/group

$(Z_\alpha + Z_\beta)^2 = 10.5$ [from table 4.13.1][281]

σ_D = 20 mg/dl

δ = 13 mg/dl

$n = 10.5\ (20^2/13^2) = 24.9 = 25$

Hypothesis #2 comparing Self-CARE to Usual Care involves non-paired data. Sample size was calculated as follows:[281]

Power = .90

Level of Significance = 95% (α = .05)

$n = (Z_\alpha + Z_\beta)^2\ 2\ (\sigma_D^2/\delta^2)$ n= # of individuals/group

$(Z_\alpha + Z_\beta)^2 = 10.5$ [from table 4.13.1][281]

σ_D = 20 mg/dl

δ = 13 mg/dl

$n = 10.5\ [2(20^2/13^2) = 49.7 = 50$

As shown above, the minimum number of subjects required in each group to compare Self-CARE to usual-care would be 50 individuals. Anticipating a dropout of 15% from the study groups, and after considering the gender distribution, the patient loads at each location and the time available, the sample size goal was set at 130 in Self-CARE and 130 in Usual Care distributed as follows:

Peterson Field Clinic- 70 in Self-CARE and 70 in Usual Care with approximately 45 males & 25 females in each group.

USAFA Hospital- 60 in Self-CARE and 60 in Usual Care with approximately 30 males & 30 females in each group.

These numbers will allow either location to stand alone during the analysis of results.

Screening of Subjects

Eligible participants were recruited from adult volunteers, men and women, aged 25 to 75 years, whose cholesterol values are above the 75th percentile (200 mg/dl)

or who have a TC/HDL-C ratio of greater than 4.5. Both sexes were recruited, since studies have found that any differences in cholesterol measurement between men and women at fixed lipid levels were negligible[256] and that the lipid responses of men and women at fixed lipid levels to dietary modification are the same.[280] Any subject that would not be available for the three month serum analysis was not enrolled in the study. Additionally, individuals with triglycerides above 500 mg/dl were excluded due to problems with accurate LDL-C and HDL-C measurements. Individuals with TG from 400-500 did not have their LDL-C level calculated[282] and the level was recorded as missing data. Individuals taking prescription medications, including cholesterol lowering drugs were admitted as long as there status did not change during the course of the study.

Clinical and Anthropometric Measurements

Height and Weight. The height and weight of each participant was obtained at initial enrollment by reference to the individual's latest physical examination form or directly from the individual if the exam form was not available. Each individual's height and weight data was used to calculate a body mass index (BMI) that would be used during analysis of the data. BMI is calculated by dividing the individual's weight in kilograms by height in meters squared and multiplying it by 100.[33] The individual's three month weight was self-reported on the diet worksheet.

Total Cholesterol, HDL-C, Triglycerides, and LDL-C. At both the USAFA and the PAFB laboratories, the blood for the lipid analysis was obtained after a 12 hour fast, with the patient seated and the tourniquet removed. Each facility is standardized and has continuous centrally supervised quality control. Both laboratories adhere to the guidelines established by the College Of American Pathologists.[283]

The USAFA laboratory used a Hitachi-737 Automatic Analyzer and enzymic methods (Boehringer Mannheim System Reagents, Boehringer Mannheim Diagnostics, Indianapolis, IN 46250) to measure TC[284] and TG.[285] They used an Abbott VP analyzer, and a Sclavo Precipitating Reagent (Sclavo Inc., Wayne, NJ, 07470) for the enzymatic measurement of HDL-C.[286]

At PAFB, the Abbott VP analyzer was used to determine TC,[284] TG,[285] and HDL-C[287] values. Reagents from Abbott Laboratories (Abbott Laboratories, Diagnostic Division, Irving , TX 75061) were used for all enzymatic determinations and a Baker Precipitating Reagent (Baker Instrument Corporation, Allentown, PA 18103) for the separation of HDL-C. The LDL-C values are calculated from the other lipid values, using the relationship that LDL-C = TC-(HDL-C + TG/5).[282] A TC/HDL-C ratio for each analysis is also calculated.

Dietary Evaluation

Participants in the Self-CARE program were to submit three diet worksheets during the course of the study. One

was to be submitted within two weeks of enrollment, the next by the six week point and the third during the tenth week of the study. Usual-care subjects submitted only one worksheet, at the time they came in for their three month cholesterol recheck. The worksheets (Appendix C) contained detailed instructions for recording one day's food consumption. Upon receipt of a diet worksheet, it was reviewed for completeness and every attempt was made to insure that the results of the analysis were returned in the next day's mail.

The worksheets were coded for computer processing by the author, using the PRUCAL diet analysis code list and then analyzed using the PRUCAL computer program[277] When a food portion was not given, a standard portion was used and a note to that effect included with the completed analysis. Each diet was analyzed with comparison to the "Prudent Diet" that recommends no more than 30% of calories as fat, with 10% each from saturated and polyunsaturated fat. Each completed analysis contained a personal note that highlighted a "good" aspect of the individual's diet.

A protocol was established for missing worksheets. Any individual that did not submit a worksheet within one week of the day it was due would receive one phone or post-card reminder. Any individual in the Self-CARE program that did not complete at least two diet worksheets was considered a program drop-out. Individuals in usual-care that completed their three month cholesterol check but did not turn in a

diet worksheet were retained in the program with their diet data treated as missing data. They were telephoned to obtain a final weight, activity level and to determine what prescription drugs they were taking.

Interventions

USAFA Recruitment. At the USAFA, individuals who had been identified as at risk for CAD due to elevated serum cholesterol and/or TC/HDL-C levels above 4.5 were advised by their CHCPs that they could enter an intervention program that was available through the dietitian's office. Interested individuals were then able to make an appointment for individualized diet counseling. The registered dietitian would review the individuals records and if the client met the requirements listed in the screening section, she would inform the individual that a Self-CARE program was available. If the individual expressed an interest, they were given a Self-CARE folder that had been personalized with their lipid levels on the folder flap and their cholesterol goal and recheck date on the magnetic reminder (Appendix C). While the package was being personalized, the individual completed an enrollment and a consent form, and was provided with a subject information sheet (Appendix D). The individual was instructed to start the program as soon as possible and send in the first diet worksheet within two weeks. The entire enrollment process takes approximately 10 minutes and then the individual is free to leave. All Self-

CARE enrollments at the USAFA were completed by a Registered Dietitian.

Individuals that had received the individualized diet counseling of approximately one hour duration were asked if they would participate in a study that was being conducted to determine the effectiveness of the nutrition education program. Individuals that agreed to let their serum cholesterol values be used and to complete one diet worksheet were enrolled in usual-care. They completed a consent form, were given a subject information sheet and then told that approximately two weeks prior to their recheck date, they would be mailed a diet worksheet and instructions for their cholesterol recheck. In some cases, individuals that declined to enter the Self-CARE program were enrolled in usual-care.

Recruitment at the USAFA began on August 1, 1988, and the last individual was enrolled on 6 Jan 1989. A total of 62 individuals were enrolled in Self-CARE and 61 subjects in usual-care. During the last 30 days of the enrollment, only men were targeted for enrollment to insure that 30 men would be enrolled in both portions of the study.

PAFB Recruitment. Individuals that were identified by their CHCP as at risk for CAD due to elevated TC levels or a TC/HDL-C ratio above 4.5 were advised that they could enter an intervention program offered by the clinic. The intervention program offered at PAFB involves a group lecture session on cholesterol and diet taught by a

Registered Nurse. During the one-hour class, a variety of handouts from the American Heart Association and the Air Force dealing with the "Healthy Heart" diet are available and there is usually an opportunity for questions at the conclusion of the session. Twenty to 25 individuals are usually in attendance at each of these diet classes.

Enrollment of individuals at PAFB was conducted by the author. A brief description of the Self-CARE program was presented to the group prior to the start of the class and then any interested individuals where taken to another room where they were enrolled as described for the USAFA and then allowed to leave. The enrollments at PAFB were completed by the author, since the nurse in charge of the program would be teaching the diet-class while the Self-CARE individuals were being enrolled.

Individuals were enrolled in usual-care at the conclusion of the diet-class. The program was explained to the diet-class participants and interested individuals were allowed to enroll. They were given a package containing an enrollment form, consent form, and a subject information sheet. Five to 10 individuals were usually enrolled at a time and their paperwork checked as they left the classroom. As at the USAFA, they were told to expect a letter with their diet worksheet and instructions for the three month recheck in the mail. An attempt was made to enroll individuals in Self-CARE one week and then enroll individuals in usual-care the next week, however, some

individuals had been told by their CHCP that the Self-CARE program was available and they were allowed to enroll regardless of the enrollment planned for that week.

Recruitment at PAFB began on September 29, 1988, and was completed on December 21, 1988. A total of 76 individuals were enrolled in Self-CARE and 76 were enrolled in usual-care. Toward the end of the enrollment period, an emphasis was placed on enrolling only men so that the planned numbers would be admitted to the study.

Self-CARE. The Self-CARE program after enrollment did not differ between the two bases. All individuals had identical packages and identical cholesterol reduction goals (10%). Individuals were followed for the three months, with reminders sent or called for diet worksheets that were not in on time. See the section on Dietary Evaluation for the protocol followed.

Usual-care. Usual-care differed significantly between the two locations. At each base the individuals were allowed to choose any of several pamphlets dealing with dietary management of serum cholesterol,[288,289] but none of these resembled an organized program. At the USAFA, the intervention consisted of a session lasting approximately one hour. During the appointment, the individuals were thoroughly familiarized with all the aspects of the "Prudent Diet". The dietitian was able to adjust the program depending on the clients background and concerns, but had no further contact with the client until the three month point.

The group sessions at PAFB examined the major aspects of the "Diet-Heart" question. The pathogenesis and etiology of CAD was reviewed by the nurse and the foods to eat and the foods to avoid were emphasized. There was usually time for questions at the end of the session, but the presentation itself, varied little from week to week. As at the USAFA, there is not any contact with the usual-care individuals until the end of the three month period.

Three Month Follow-up. For the individuals enrolled in Self-CARE, a note was attached to the last diet worksheet, reminding them of the three month recheck date. Individuals that were one week late for their cholesterol recheck were called and reminded that they had missed their recheck. If they still did not respond to the call, they were sent a post card. Those that still did not respond were dropped from the study.

Individuals enrolled in usual-care are sent a diet worksheet and a letter (Appendix D) in the mail that reminds them of their three month recheck date and the procedures to be followed. Subjects that miss their recheck, as in Self-CARE, receive a telephone call and then are sent a reminder. Since there has been no contact with the usual-care individuals during the previous three months, they are given a second call reminding them to get their recheck, if necessary. If the subjects still do not respond, they are dropped from the study.

For each program, at the three month check information on weight, activity level, prescription drug use, and cholesterol levels are collected. The Self-Care participants also turn in their critique form (Appendix C). For all individuals dropped from the study, they are interviewed, if possible, to determine the reason for their withdrawal or failure to complete the program.

Evaluation

Statistical Analysis

In the analysis of this study, I attempted to avoid the statistical problems, involving multiple P values and low statistical power, that have developed in other studies, when the data is "over" analyzed.[271] This analysis focuses on the two primary endpoints, the change in TC and the change in TC/HDL-C ratio specified during the design of this study. Analysis of multiple end points and subgroup analyses are reported but only interpreted in an exploratory manner. The statistical analysis first treats each location separately and then examines the combined results.

The data for the lipid analysis is presented as the mean $\pm$ the standard deviation at the baseline and at the three month point. Preliminary analysis involved assessing the comparability of the treatment groups at the start of the study and then determining if there was a significant difference between each group's response to treatment. A two sample "t" test was used to assess the significance of

these between group differences. A student "t" test for paired data was used to assess the statistical significance of changes in blood lipids among subjects. The level of statistical significance was determined using a two-tail test and a 5% level of significance for each test.

When the preliminary analysis revealed a statistically significant change in the mean level of a serum lipid, multiple regression analysis was used to look at the relationship between the changes and several independent variables. The dependent variables used in the analysis were the change in TC, HDL, TC/HDL ratio, LDL, and TG. For each dependent variable, the independent variables entered were location, treatment, sex, age, BMI, prescription drug use, oat product consumption, activity level, smoking status, fat intake and P/S ratio. Step-wise multiple regression analyses were performed by computer using the SAS/STAT Release 6.03.[290] The step-wise procedure was used so that the independent variable(s) remaining in the model would only be those that are statistically significant. Finally, analysis of covariance was used to determine if the significant differences detected by the two sample "t" tests were actually due to treatment or perhaps to factors such as the age and/or initial BMI of subjects in the study.

Noncompliance/Missing Data

Individuals that failed to comply with the program or requested to be dropped were interviewed to determine their

reasons. A record was maintained in an attempt to monitor the reasons for their actions. Noncompliant individuals were retained in the study for analysis purposes if at all possible. This was done in an attempt to avoid a selection bias and result in a report on how the treatments performed in actual practice. The same attempt was made for individuals that requested to be dropped from the study. A second analysis was completed and reported for individuals that actually completed the study.

Every effort was made to avoid missing data. In some cases, an individual may not have had a complete serum cholesterol analysis prior to the intervention or have moved prior to sending in a diet worksheet. In these cases, the missing data was reported, a sample size reported and an estimation of its effect on the analysis stated.

CHAPTER IV

RESULTS

Pooled Analysis

The data were first analyzed by pooling all the results and looking at the relationship between the dependent variables change in TC, LDL-C, and HDL-C, and the independent variables treatment, location, gender, age and initial BMI. The regression analysis revealed that there was a significant location effect for the change in TC ($F=34.42$, $P=0.0001$), the change in LDL-C ($F=26.40$, $P=0.0001$) and the change in HDL-C ($F=14.87$, $P=0.0001$). These findings served to reinforce the observation that the study sites were very different and that the findings were not in fact combinable and should be presented separately to facilitate the interpretation of the results of this project.

United States Air Force Academy Diet Clinic

Subject Profile

The planned sample size for this location was 120 subjects, with 60 to be enrolled in Self-CARE (SC) and 60 to be enrolled in usual-care (UC), equally divided between males and females (Table 1). The final enrollment was 62 in SC, 36 males and 26 females and 60 in UC, 28 males and 32

Table 1. United States Air Force Academy Diet Clinic - Sample Size: Goals, Actual, Dropout, and Final.

	Self-CARE (M/F)*	Usual-Care (M/F)
Goal	60 (30/30)	60 (30/30)
Actual	62 (36/36)	60 (28/32)
Dropout	7 (5/2)	5 (2/3)
Final	55 (31/24)	55 (26/29)

* M/F = Male/female

females. While the total sample size exceeded planning goals, the SC sample was 4 females short of the original goal and UC was 2 males below its goal. The main reason for this was that 2 males and 2 of the females were put on cholesterol lowering medications after enrolling in the study and consequently had to be dropped. The other two females were removed from the study when their complete lab work not available when they enrolled, revealed triglyceride levels above program limits. All subjects had at least a high school education.

As shown in Table 2, at the beginning of this study, there were no significant differences between the two groups in either the demographic factors recorded or blood lipid values measured. Subjects ranged in age from 25 to 79, with an average age of 53.6 in SC and 53.8 in UC. Total cholesterol levels averaged 258 mg/dl for SC and 259 mg/dl for UC, almost exactly the same as the 260 mg/dl used in planning. A BMI was calculated for each individual and the average values were almost identical for the two study groups, 25.3 (kg/m^2) versus 26.2 (kg/m^2). The initial LDL-C levels averaged 174.61 ± 35.17 for SC and 172.49 ± 30.80 for UC, levels well above the LDL-C level of 160 mg/dl that is used to indicate high risk.[83] Triglycerides were also elevated, averaging 196.89 ± 96.53 mg/dl for SC and 203.61 ± 110.76 mg/dl for UC.

The homogeneity of these groups attested to the success of the randomization of the subjects and supported the

Table 2. United States Air Force Academy Diet Clinic - Baseline Values* of Selected Variables for Self-CARE and Usual-Care.

Variable	Self-CARE	Usual-care
Number	62	60
Gender (M/F)	36/26	28/32
Smoke	9	7
Age (years)	53.60±13	53.80±13
BMI	25.25±3.3	26.15±3.79
TC (mg/dl)	258.16±38.24	258.85±36.45
LDL-C (mg/dl)	174.61±35.17	172.49±30.80
HDL-C (mg/dl)	46.21±11.28	47.95±15.19
TG (mg/dl)	196.89±96.53	203.62±110.76
TC/HDL-C ratio	5.90±1.5	5.80±1.6

* mean values ± standard deviation
# significant difference from Self-CARE, Student's t test $P<0.05$
BMI = body mass index (kg/m^2 x 100)
TC = total serum cholesterol
LDL-C = low density lipoprotein cholesterol
HDL-C = high density lipoprotein cholesterol
TG = triglycerides.

decision made during planning to not disrupt the normal education routine with baseline knowledge testing or extensive entry questionnaires that could influence the subjects and possibly confound the results.[291]

Retention of Subjects

The planned sample sizes were determined anticipating a 15% dropout rate. The actual dropout rates were 11.3% for SC and 8.3% for UC (Table 1). This meant that out of the 62 actually enrolled in SC, seven dropped out (5 males and 2 females), leaving 55 individuals in SC (31 males and 24 females). For UC, five individuals dropped out (2 males and 3 females) leaving 55 individuals (26 males and 29 females) enrolled at program completion. Analysis of the characteristics of the individuals leaving either program did not reveal any consistent characteristics that would predict which individuals would choose not to complete the study. Reasons for leaving SC included Grave's disease, a heart attack, and personal problems at home for three of the individuals. Of the remaining four, three expressed a general lack of motivation or desire to complete the program and the fourth could not be reached for comment despite repeated attempts. Reasons for not completing UC were similar to those given for SC. One individual suffered a stroke, one individual's husband died, and the remaining three just did not have the time or interest.

Program Completion

A log was kept that tracked the number of individuals in each program that did not complete their three month cholesterol recheck within one week of the recheck date. These individuals received additional reminders in accordance with the protocol established at the beginning of the study (see methods section). For individuals in SC, 15% or 9 individuals were contacted as compared to 45% or 27 individuals in UC (Figure 1). These results were not unanticipated, since there had not been any contact with the UC individuals during the three months of the program. The most common response given when asked why they had not completed their recheck was "I forgot all about it." At the USAFA, lab the individuals were able to have their blood drawn for their follow-up analysis anytime during the day or early evening. This meant that no one was able to use the lab's operating hours as an excuse for not completing their recheck.

Body Mass Index

While a large weight loss is not expected over only a three month period, both the SC and UC participants experienced an overall decrease in body weight, as evidenced by almost identical decreases in BMI of 0.45 ± 0.64 for SC and 0.41 ± 0.83 for UC. In each case as shown in Table 3, the changes for each program resulted in BMIs that were significantly different from the baseline values of

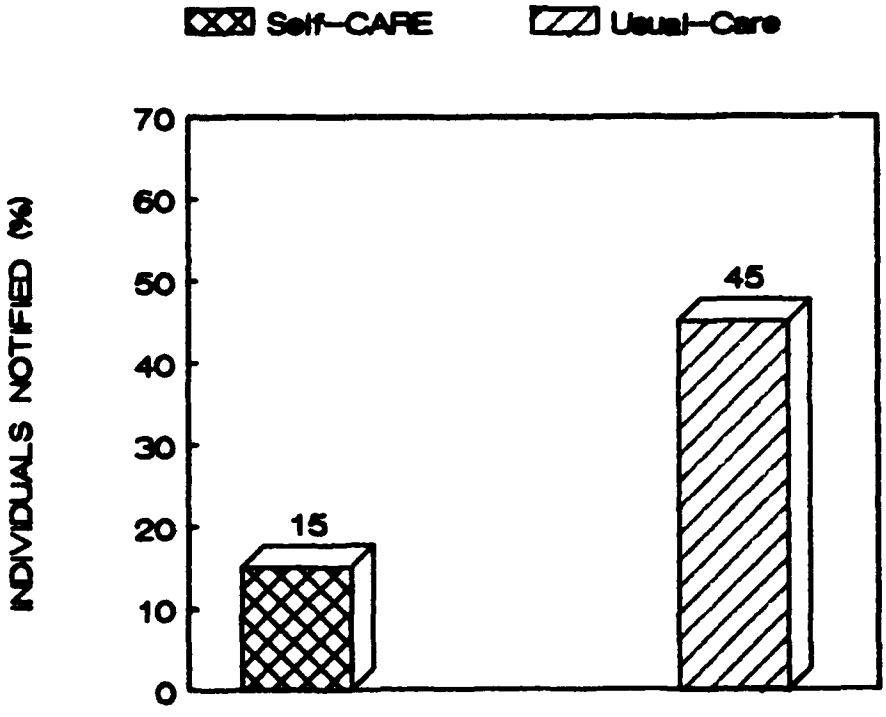

Figure 1. Percentage Requiring Follow-up (United States Air Force Academy).

Table 3. United States Air Force Academy Diet Clinic - Change in Values* of Selected Variables over three months.

Variable	N	Baseline	3-Months	Change
Self-CARE				
TC mg/dl	55	258.16±38.24	246.31±45.67	-13.27±26.29#
HDL-C mg/dl	55	46.21±11.28	45.18±11.99	-1.24±08.32
TC/HDL-C	55	5.90±01.50	5.80±01.60	-0.13±01.11
BMI	55	25.25±03.33	24.74±03.39	-0.45±00.64#
LDL-C mg/dl	52	174.61±35.17	165.02±41.15	-8.85±25.08#
TG mg/dl	55	196.89±96.53	181.44±81.41	-15.45±865.10
Usual-care				
TC mg/dl	55	258.85±36.45	247.04±34.71	-12.04±34.27#
HDL-C mg/dl	55	47.95±15.19	48.18±14.47	-0.32±06.09
TC/HDL-C	55	5.80±01.60	5.50±01.50	-0.37±01.16#
BMI mg/dl	55	26.15±03.79	25.65±03.98	-0.41±00.83#
LDL-C mg/dl	52	172.49±30.80	162.66±32.02	-8.19±33.71
TG mg/dl	55	203.62±110.76	187.00±98.04	-16.42±102.30

* mean values ± standard deviation
# significant difference by paired t test, $P<0.05$
$ significantly different from usual-care, Student's t test $P<0.05$
BMI = body mass index (kg/m^2 x 100)
TC = total serum cholesterol
LDL-C = low density lipoprotein cholesterol
HDL-C = high density lipoprotein cholesterol
TG = triglycerides.

25.25±3.33 for SC and 26.15±3.79 for UC, but not significantly different from one another (F=0.09, P=0.7698).

Behavioral Factors

The self-reported data collected on activity level, vitamin use, smoking, and alcohol and oat product consumption were treated as independent variables in the analysis of the results. Activity level was recorded as sedentary, light or moderate, and the remaining four categories as either "yes" or "no". There were no significant differences between the two groups in any of the categories. Oat product consumption was noted in the diets of 58.2% of the SC and 61.5% of the UC subjects. Only 27.3% of the SC and 33.3% of the UC participants reported alcohol consumption on their three month diet analysis.[292] These percentages are below the expected levels in the population at large, but are from only a 24 hour diet record and are self reported. However, the number of individuals reporting smoking was only 11.7% in UC and 14.5% in SC, percentages that are also well below the 28% average for this population.[292] The use of vitamins was not widespread, with only 10.9% of the SC and 11.8% of the UC subjects reporting their use. Analysis of the activity level data for SC and UC disclosed that 23.6% and 23.5% respectively considered themselves to be sedentary, while 38.2% of the SC and 45.1% of the UC participants chose "light" for their activity level. A "moderate" activity level was selected by 38.2% of

the SC and 31.4% of the UC followers. Statistical analysis of how these behavioral factors influence the serum lipids is included in the following section.

Blood Lipids

A complete cardiac risk assessment was completed both before the program and at the three month point. The values for TC, LDL-C, HDL-C and TG were recorded and the changes in these values used as indicators of the effectiveness of the SC and UC programs. Since, as mentioned in the methods section, the lipid responses of men and women at fixed lipid levels to dietary modification are the same,[280,293] and the work by Keys[294] which reported that in 39 studies of the effect of dietary cholesterol on serum cholesterol, that they found no difference in the response of the sexes, the data presented here is for the entire SC or UC group. Multiple regression and co-variate analysis was used to reveal any significant gender or age interactions, but the focus of this analysis was on the overall program effect on TC and the TC/HDL ratio.

At the USAFA, as shown in Table 3., the change in TC was significantly different from baseline for both SC and UC. The decrease was 13.27 ± 26.29 mg/dl for SC and 12.04 ± 34.27 mg/dl for UC, approximately a 5% decrease for each program. These decreases were not significantly different from one another ($F=0.05$, $P=0.8323$), but are in line with the 5-7% decrease in TC one would expect from

adoption of ıe AHA Step 1 Diet.[99] Also, the HDL-C levels in the SC grou, decreased 1.24±8.32 mg/dl, however, this decrease to a total HDL-C level of 45.18±11.99 mg/dl did not represent a significant decrease from the baseline value of 46.21±11.28 mg/dl. This contrasts with the small increase of 0.33±6.09 mg/dl in HDL-C shown by the UC group. As in the SC group, this small increase in HDL-C for UC was not significant and the three month HDL-C levels were not significantly different from baseline for either group. However, while the -0.13±1.11 decrease in the TC/HDL-C ratio for SC did not represent a significant decrease, the combination of a decrease in TC and the small but nonsignificant increase in HDL-C resulted in a significant decrease of -0.37±1.16 in the TC/HDL-C ratio for UC ($P<0.05$). This decrease was not significantly different from the SC change ($F=1.17$, $P=.2812$). Also as shown in Table 3, the decrease in LDL was almost identical for each group, significantly different from baseline values, but not significantly different from one another ($F=.01$, $P=0.9122$). Triglycerides also decreased for both the SC (-15.45±85.1 mg/dl) and the UC (-16.42±102.3 mg/dl) groups, but due to the large standard deviations, the decreases did not represent a significant change from baseline for either group.

Next, multiple regression analysis was used to examine possible relationships between the independent variables in this study and the dependent variables that had a

statistically significant change in mean level. For these dependent variables- TC, TC/HDL-C, and LDL, step-wise multiple regression did not show that any of the independent variables entered (treatment, gender, age, initial BMI, prescription drug use, oat product consumption, activity level, smoking status, or P/S ratio) had an independent and statistically significant relationship with the changes observed. Additionally, when the change in BMI was entered into the analysis, there were still no significant interactions noted. Note that only fat intake as a percentage of calories and smoking were even entered in the stepwise model, and that neither reached the 0.05 level of significance. There were no statistically significant results, so no other results from stepwise regression are presented. Overall then the analysis revealed that both SC and UC were associated with significant decreases in TC and BMI, and the SC group demonstrated a significant decrease in LDL-C, while the TC/HDL-C ratio decreased significantly only for UC. None of the changes in these dependent variables, that is the significant differences observed, were significantly related to any of the independent variables, nor did adjustments for the covariates age and initial BMI change this finding, see Table 4.

Dietary Intakes

While the reason for including the PRUCAL diet analysis was basically to increase compliance with SC by serving as a

Table 4. United States Air Force Academy Diet Clinic: Analysis of the Change in Total Cholesterol, TC/HDL-C Ratio, and LDL Cholesterol, Adjusted for the Covariates Age, and Initial BMI#. Degrees of freedom = 1.

Dependent Variable	TC		TC/HDL-C Ratio		LDL-C	
	F	P	F	P	F	P
Source of Variation						
Treatment	0.07	0.795	0.71	0.401	0.01	0.964
Gender	0.47	0.495	0.98	0.326	0.17	0.683
Treatment X Gender	0.39	0.534	0.01	0.949	0.24	0.629

@ Change in value - baseline to three months
# P<0.05
# BMI = body mass index (Kg/m^2 x 100)
TC = total serum cholesterol
LDL-C = low density lipoprotein cholesterol
HDL-C = high density lipoprotein cholesterol

periodic reminder of the program, the information on several dietary variables was recorded (Table 5). Since the main focus of the SC program was on reducing the fat content of the diet, the change in the fat in the diet as a percentage of calories was carefully examined. Individuals enrolled in SC submitted a 24 hour diet record soon after receiving the SC program and on average, they reported a 32.5±9.2% intake of fat as a percentage of calories and a P/S ratio of 0.62±.39. By the end of the program, their fat intake had decreased significantly to 28.0±7.9% of calories ($p<0.001$), and the P/S ratio had increased significantly to 0.76±.40 ($p<0.001$). Since only one analysis was submitted at the end of the program by the UC group, the assumption was made that the UC diets were similar to the SC diets at baseline. For UC at the three month point, a fat intake of 30.8±8.1% of calories and a P/S ratio of 0.88±.42 was reported. Neither of these values was significantly different from the SC levels, but would be significantly different from the assumed baseline values. It would be expected that a decrease in total fat intake and the improvement in the P/S ratio would lead to reduced cholesterol levels. The "stepwise" analysis did not reveal a significant association between total fat intake ($F=3.52$, $P=0.0638$) and changes in TC, or a significant association with the change in the P/S ratio ($F=0.04$, $P=0.8453$).

Of the other three dietary variables that were analyzed, total calories and cholesterol intake demonstrated

Table 5. Mean Dietary Intake* by 24 Hour Diet Record for Self-CARE and Usual-Care at United States Air Force Academy (USAFA) and Peterson Air Force Base (PAFB) Diet Clinics.

Dietary Variable	Self-CARE		Usual-care
	Entry	3-Month	3-Month
USAFA Diet Clinic			
Total Calories	1726±528[a,b]	1519±509[aa,c]	1645±633[b,c]
as a % of goal**	89	78	86
Cholesterol (mg)	210±108[a,b]	176±100[a]	165±127[b]
as a % of maximum goal#	70	59	55
Total Fat % of calories	32.5±9.2[a,b]	28.0±7.9[a]	30.8±8.1[b]
P/S ratio**	0.62±0.39[a,b]	0.76±0.40[a]	0.88±0.42[b]
Dietary Fiber (gm)	28.7±8	19.3±8	20.2±10
as a % of minimum goal@	65	65	75
PAFB Diet Clinic			
Total Caolories	2027±686[a,b]	1684±511[a]	1711±543[b]
as a % of goal**	97	80	81
Cholesterol (mg)	256±178[a,b]	182±105[a]	193±105[b]
as a % of maximum goal#	85	61	64
Total Fat % of calories	33.8±8.0[a,b]	29.0±8.0[a]	30.3±7.7[b]

Table 5 (Continued)

Dietary Variable	Self-CARE		Usual-care
	Entry	3-Month	3-Month
P/S ratio***	0.64±0.42[a,b]	0.78±0.51[a]	0.78±0.43[b]
Dietary Fiber (gm)	21.8±9	21.9±12	21.6±12
as a % of minimum goal@	70	72	69

* mean values ± standard deviation.
** From Prucal diet analysis-based on gender, desirable weight for height and frame size, and activity level.
*** Ratio of polyunsaturated fat to saturated fat.
\# 300 mg/day
@ 15 grams/1000 kcal
[a,b,c] Values in the same row sharing the same superscript are significantly different. $P<0.05$.

a pattern similar to that discussed above for total fat as a percentage of calories and the P/S ratio. Each was significantly different from baseline or assumed baseline value (Table 5) but could not be shown to be of value in predicting changes in cholesterol. Dietary fiber also did not have a significant relationship with changes in blood lipids and in addition its intake did not change significantly over the course of the study (Table 5).

Prescription Medications

Reported prescription drug use was not significantly different for the two groups (X^2=3.78 P>0.05)). There were however, nine more UC individuals than SC reporting prescription drug use. One female in SC and two females in UC were taking a cholesterol lowering medication, Gemfibrozil, a bile acid sequestrant, at the start of the study. Six individuals in SC and 9 in UC reported taking blood pressure medications, while 2 females in SC and 4 in UC were taking estrogen supplements. Three females in UC reported birth control pill use, while none of the females in SC claimed to be taking birth control pills. On final analysis, due to the small numbers using each type of prescription drug, the individuals were categorized as either "drug" or "no drug" for the statistical analysis. Analysis of the 12 individuals in SC that reported prescription drug use and the 21 in UC as mentioned above, revealed no significant relationship between serum lipid

changes and prescription drug use or any significant difference between the two groups.

Peterson Air Force Base Diet Clinic

Subject Profile

The planned sample size for this location was 140 subjects, with 70 to be enrolled in Self-CARE (SC) and 70 to be enrolled in usual-care (UC). The plan was to have 45 males and 25 females take part in each program (Table 6). The final enrollment was 74 in SC, 50 males and 24 females, and 73 in UC, 46 males and 27 females. The total sample size exceeded planning goals except for the SC sample which was 1 female short of the original goal. One female was lost because she was put on cholesterol lowering medication after enrolling in the study and consequently had to be dropped.

An analysis of the baseline demographic factors recorded and the blood lipid values measured (Table 7) revealed that the only significant difference between the two groups was that the initial BMI of the UC group was significantly higher than that of the SC group ($T=2.754$, $P<0.05$). Subjects ranged in age from 21 to 72, with an average age of 44.6 ± 12.8 in SC and 48.0 ± 11.8 in UC. Total cholesterol levels averaged 264.62 ± 41.2 mg/dl for SC and 259.67 ± 31.69 mg/dl for UC, almost exactly the same as the 260 mg/dl used in planning. A BMI was calculated for each individual and the average values were 24.8 ± 3.8 for SC and

Table 6. Peterson Air Force Base Diet Clinic - Sample Sizes: Goals, Actual, Dropout and Final.

	Self-CARE (M/F)	Usual-care (M/F)
Goal	70 (45/25)	70 (45/25)
Actual	74 (50/24)	73 (46/27)
Dropout	7 (5/2)	9 (4/5)
Final	67 (45/22)	64 (42/22)

* M/F = Males/Females

Table 7. Peterson Air Force Base Diet Clinic - Baseline Values* of Selected Variables for Self-CARE and Usual-care.

Variable	Self-CARE	Usual-care
Number	74	73
Gender (M/F)	50/24	28/32
Smoke	10	7
Age (years)	44.60±13	48.00±12
BMI	24.81±3.79	26.67±4.40#
TC (mg/dl)	264.62±41.18	259.67±31.69
LDL-C (mg/dl)	183.62±35.99	175.00±30.81
HDL-C (mg/dl)	46.15±12.53	46.48±13.00
TG (mg/dl)	177.27±76.78	195.30±86.26
TC/HDL-C ratio	6.10±1.8	5.90±1.4

* mean values ± standard deviation
# significant difference from Self-CARE, Student's t test $P<0.05$
BMI = body mass index (kg/m^2 x 100)
TC = total serum cholesterol
LDL-C = low density lipoprotein cholesterol
HDL-C = high density lipoprotein cholesterol
TG = triglycerides.

26.7±4.4 for UC, the UC value, as mentioned above, being significantly higher than SC's average value. LDL-C values at entry were not significantly different, with a value of 183.62±35.99 mg/dl for SC and 175.00±30.81 mg/dl for UC. The entry triglyceride levels were not statistically different (T=0.738, P=<0.05) although the entry level of 195.30±86.26 mg/dl for UC appeared to be much higher than the 177.27±76.78 mg/dl for SC.

As shown in Table 7, with the exception of BMI,the samples are relatively homogenous. This finding supports the decision made during planning to not disrupt the normal education routine with baseline knowledge testing or extensive entry questionnaires that could influence the subjects and possibly confound the results.[291]

Retention of Subjects

The planned sample sizes were determined anticipating a 15% dropout rate. The actual dropout rates were 9.5% for SC and 12.3% for UC (Table 6). This meant that out of the 74 actually enrolled in SC, seven dropped out (5 males and 2 females), leaving 67 individuals in SC (45 males and 22 females). For UC, 9 individuals dropped out (4 males and 5 females) leaving 64 individuals enrolled at program completion. Analysis of the characteristics of the individuals leaving either program did not reveal any consistent characteristics that would identify an individual likely to leave the study. Reasons for leaving SC included

a husband's death, mother in hospital, ulcers and an unspecified illness for 4 of the individuals. Of the remaining 3, two expressed a general lack of motivation or desire to complete the program and the third had a disconnected phone and could not be reached for comment, despite repeated written inquiries. Reasons for not completing UC were another case of ulcers and an extended vacation for two of the women. One of the males was unexpectedly reassigned to Italy, and the remaining three just did not have the time or interest.

Program Completion

A log was kept that identified the number of individuals in each program that did not complete their three month cholesterol recheck within one week of the recheck date. These individuals received additional reminders in accordance with the protocol established at the beginning of the study (see methods section). For individuals in SC, 34% or 25 individuals were contacted as compared to 58% or 42 individuals in UC (Figure 2). These results were not unanticipated, since there had not been any contact with the UC individuals during the three months the program ran. The most common response given when asked why they had not completed their recheck was: "I forgot all about it." At the PAFB lab, the individuals were able to have their blood drawn for their follow-up analysis only from 0730-0930- weekdays. This resulted in many individuals

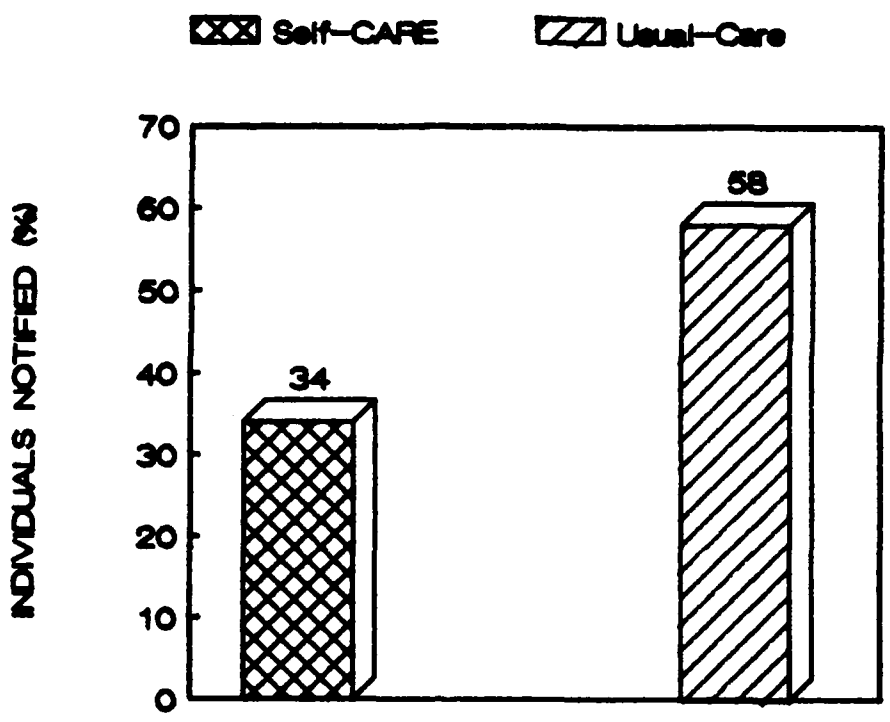

Figure 2. Percentage Requiring Follow-up (Peterson Air Force Base).

claiming that they were not able to get in for their rechecks due to the lab's limited hours.

Body Mass Index

While a large weight loss is not expected over only a three month period, on average, both the SC and UC participants experienced decreases in BMI. For SC the decrease was 0.42 ± 0.62 and for UC it was 0.34 ± 0.69 and in each case as shown in Table 8, the changes for each program were significantly different from baseline ($p<0.05$) but not significantly different from one another ($F=0.53$, $P=0.4684$).

Behavioral Factors

The self-reported data that was collected on activity level, vitamin use, smoking, and alcohol and oat product consumption were treated as independent variables in the analysis of the results. Activity level was recorded as sedentary, light or moderate, and the remaining four categories as either "yes" or "no". There were no significant differences between the two groups in any of the categories. Oat product consumption was noted in the diets of 62.3% of the SC and 48.4% of the UC subjects. Only 23.2% of the SC and 28.1% of the UC participants reported alcohol consumption on their three month diet analysis. These percentages for alcohol consumption are slightly below the expected levels in the population at large, but are from only a 24 hour diet record and are self reported. However,

Table 8. Peterson Air Force Base Diet Clinic - Change in Values* of Selected Variables over three months.

Variable	N	Baseline	3-Months	Change
Self-CARE				
TC mg/dl	67	264.62P41.18	218.18±32.29	-46.48±32.6#$
4HDL-C mg/dl	67	46.15±12.53	43.02±09.93	-3.05±09.51#
TC/HDL-C	67	6.10±01.80	5.30±01.30	-0.77±01.70#$
BMI	67	24.81±03.79	24.46±03.91	-0.42±00.62#
LDL-C mg/dl	67	183.62±35.99	143.06±32.13	-39.94±30.03#$
TG mg/dl	67	177.27±76.78	160.85±69.45	-16.42±65.50
Usual-care				
TC mg/dl	64	259.67±31.69	229.31±34.38	-29.13±34.86#
HDL-C mg/dl	64	46.48±13.00	41.92±09.79	-4.14±07.44#
TC/HDL-C	64	5.90±01.40	5.70±01.60	-0.21±01.03
BMI	64	26.67±04.41	26.34±04.17	-0.34±00.69#
LDL-C mg/dl	61	175.00±30.81	154.30±30.98	-21.49±32.59#
TG mg/dl	64	195.30±86.26	163.19±75.10	-32.21±70.20

* mean values ± standard deviation
# significant difference by paired t test, $P<0.05$
$ significantly different from usual-care, Student's t test $P<0.05$
BMI = body mass index (kg/m^2 x 100)
TC = total serum cholesterol
LDL-C = low density lipoprotein cholesterol
HDL-C = high density lipoprotein cholesterol
TG = triglycerides.

the number of individuals reporting smoking was only 13.7% in UC and 12.2% in SC, percentages that are also well below the 28% average for this population.[292] The use of vitamin supplements was not widespread, with only 20.3% of the SC and 25.0% of the UC subjects reporting their use. Analysis of the activity level data for SC and UC disclosed that 17.4% and 28.1% respectively considered themselves to be sedentary, while 53.6% of the SC and 45.3% of the UC participants chose "light" for their activity level. A "moderate" activity level was selected by 29.0% of the SC and 26.6% of the UC followers. Statistical analysis of how these behavioral factors influence the serum lipids is included in the following section.

Blood Lipids

A complete cardiac risk assessment was completed both before the program and at the three month point. The values for TC, LDL-C, HDL-C and TG were recorded and the changes in these values used as indicators of the effectiveness of the SC and UC programs. Since as mentioned in the methods section, the lipid responses of men and women at fixed lipid levels to dietary modification are the same,[280] and the report by Keys[294] that no difference was found in the response of the sexes to dietary cholesterol, in addition to sample size limitations, the data presented is for the entire SC or UC population. Multiple regression and co-variate analysis was used to reveal any significant

interactions between the dependent variables and independent variables and to adjust the data for age and/or BMI interactions , but the focus of this analysis was on the overall program effect on the primary endpoints, the change in TC and the TC/HDL ratio.

At the PAFB clinic, as shown in Table 8., the change in TC was significantly different from baseline for both SC and UC. The decrease was 46.48±32.64 mg/dl for SC and 29.13±34.86 mg/dl for UC, a decrease of almost 18% for SC and just over 11% for UC. These decreases were significantly different from one another ($F=8.53$, $P=0.0041$), and are in excess of the 5-7% reduction in TC one would expect from adoption of the AHA Step 1 Diet.[99] As would be expected in view of the TC decrease, the HDL-C levels in the SC group decreased 3.03±9.51 mg/dl, to a total HDL-C level of 43.02±9.93 mg/dl, a change that represents a significant decrease from the baseline value of 46.15±12.53 mg/dl ($T=-2.69$, $P<0.05$). This change was not significantly different from 4.14±7.44 mg/dl decrease in HDL-C shown by the UC group ($T=1.184$, $P>0.05$). As in the SC group this decrease in HDL-C for UC was significant, however the three month HDL-C levels were not significantly different from one another ($F=0.57$, $P=0.4501$). The 0.77±1.65 decrease in the TC/HDL-C ratio for SC did represent a significant decrease ($T=3.87$, $P<0.05$), but the decrease of 0.21±1.03 in the TC/HDL-C ratio for UC did not represent a significant change from baseline ($T=-1.65$, $P>0.05$). This decrease was

significantly different from the SC change (F=5.35, P=0.0223). Also as shown in Table 8., the decreases in LDL-C for both SC (39.94±30.03 mg/dl) and UC (21.49±32.59 mg/dl) were significantly different from the baseline values and significantly different from one another (F=10.97, P=0.0012). Triglycerides also decreased for both SC (-16.42±65.5 mg/dl) and UC (-32.21±70.2 mg/dl), but due to the large standard deviations, the decreases did not represent a significant change from baseline for either group (T=2.07 and 1.44 respectively, P>0.05), nor were the changes significantly different from one another (T=1.34, P>0.05).

Next, multiple regression analysis was used to examine possible relationships between the independent variables in this study and the dependent variables that had a statistically significant change in mean level. For these dependent variables- TC, HDL-C, TC/HDL-C, and LDL-C, step-wise multiple regression did show that of the independent variables entered (treatment, gender, age, initial BMI, BMI change, prescription drug use, oat product consumption, activity level, smoking status, dietary fat as a percentage of calories or P/S ratio) several had an independent and statistically significant relationship with the changes observed (Table 9).

The "stepwise" procedure for the dependent variable change in TC revealed as shown in Table 9, that both BMI change (F=10.15, P=0.0018), and treatment (F=6.50, P=0.0121) had statistical significant relationships with the observed

Table 9. Peterson Air Force Base Diet Clinic - Summary of the Stepwise Procedure for change in TC, TC/HDL-C Ratio, LDL-C, and HDL-C.

Variable	Partial R^2	F	P
Dependent Variable = Change in TC			
BMI	0.0762	10.1495	0.0018*
Treatment	0.0467	6.4961	0.0121*
Drugs	0.0191	2.7953	0.0971
Dependent Variable = Change in TC/HDL-C Ratio			
BMI	0.0622	8.1552	0.0050*
Treatment	0.0310	4.4104	0.0378*
Sex	0.0176	2.6016	0.1094
Dependent Variable = Change in LDL-C			
Treatment	0.0847	11.3857	0.0010*
BMI	0.0298	4.1014	0.0450*
Alcohol	0.0201	2.8072	0.0964
Dependent Variable = Change in HDL-C			
Sex	0.0611	8.0074	0.0054
BMI (Initial)	0.0187	2.4776	0.1181

@ Variables included were any that met the $P<0.05$, plus the next variable entered that met $P<0.15$ stepwise program default level of significance.
* $P<0.05$
BMI = body mass index (kg/m^2 x 100)
TC = total serum cholesterol
LDL-C = low density lipoprotein cholesterol
HDL-C = high density lipoprotein cholesterol
TG = triglycerides.

TC change. Decreases in BMI were associated with decreases in TC, while an increasing BMI was associated with an increasing TC. The least significant difference (LSD) pairwise comparison confirmed what the data seemed to show, that is that the SC treatment was responsible for greater TC decreases than UC. For the dependent variable change in HDL-C, there was not a significant treatment effect, but there was a gender relationship (F=8.01, P=0.0054), (Table 9). LSD comparison showed that UC females had a significantly greater decrease in HDL-C than the SC and UC males, but that their change was not significantly different from SC females. There was no significant difference between SC males and females, nor between SC an UC males.

The "stepwise" procedure for TC/HDL-C ratio change (Table 9), like the change in TC showed a significant relationship with the change in BMI (F=8.1552, P=0.005) and treatment (F=4.4104, P=0.0378). Again as with the TC change, the BMI and treatment variables exhibited similar relationships, that is SC was associated with greater effectiveness and BMI changes were directly correlated with ratio changes.

The treatment effect was even more pronounced for the change in LDL-C (Table 9). Treatment (F=11.3857, P=0.001) and BMI change (F=4.1014, P=0.045) again showed highly significant relationships with LDL-C change, with treatment having a larger F value than that for BMI change. LSD

comparison again show that SC is responsible for significantly greater decreases in LDL-C than UC.

It was interesting to note that the step-wise analysis revealed that there was a significant relationship between the change in BMI and the change in TC (F=10.15, P=0.0018), LDL-C (F=4.1, P=0.0450) and the TC/HDL-C ratio (F=8.155, P=0.0050) (Table 9). This result was not unexpected, since it has been recognized for many years that weight loss is usually accompanied by a decrease in TC and/or an increase in HDL-C.[58,99,295] However, when instead of change in BMI, the initial BMI was entered as an independent variable, there were no significant interactions noted with any of the dependent variables.

Finally, in order to determine if the differences revealed by the two sample "t" tests and the regression analysis were really due to treatment or if, in fact, the findings were due to other factors, analysis of covariance was employed. After examining the possible covariates, it was considered necessary to control for the effects of age and initial BMI. Two-way analysis of covariance was used with treatment, gender and treatment x gender, and the covariates age and initial BMI for the dependent variables change in TC, HDL-C, LDL-C and the TC/HDL-C ratio, (Table 10). For the dependent variable change in TC, after adjusting for the influence of age and BMI, there was still a highly significant difference between the adjusted mean changes in TC for SC and UC (F=5.64, P=0.0191).

Table 10. Peterson Air Force Base Diet Clinic - Analysis of the Change in TC, TC/HDL-C Ratio, LDL-C and HDL-C adjusted for the Covariates Age, and Initial BMI#. Degrees of freedom = 1.

Dependent Variable	TC		TC/HDL-C Ratio		LDL-C		HDL-C	
	F	P	F	P	F	P	F	P
Source of Variation								
Treatment	5.64	0.019*	4.50	0.036*	5.68	0.019*	1.30	0.257
Gender	0.20	0.656	2.44	0.121	0.01	0.927	5.07	0.026*
Treatment X Gender	0.01	0.929	0.04	0.836	1.46	0.229	0.50	0.479

@ Change in value - baseline to three months
* P<0.05
# BMI = body mass index (Kg/m^2 x 100)
TC = total serum cholesterol
LDL-C = low density lipoprotein cholesterol
HDL-C = high density lipoprotein cholesterol

Adjustments of the HDL-C change data did not alter the results; there still was not a treatment effect (F=1.30, P=0.2565) and UC females still showed a significantly greater decrease in HDL-C (F=5.07, P=0.0260) than any other subgroup. Looking at the adjusted means for the TC/HDL-C change showed treatment to still be significant (F=4.50, P=0.0359) with no other significant relationships. The same was true for the change in LDL-C, which even after adjusting for age and BMI, still showed a significant treatment effect (F=5.68, P=0.0187).

Overall then this analysis reveals that none of the changes in the dependent variables, that is the significant differences observed, were due solely to the age, gender or initial BMI of the subjects except for the HDL-C change in UC women (Table 10).

Dietary Intakes

The PRUCAL diet analysis was used to increase compliance with SC by serving as a periodic reminder or reinforcer for the program. However, information on five dietary variables was recorded (Table 5). Since the focus of the SC program was on decreasing the fat content of the diet, information on changes in the fat intake as a percentage of calories and the P/S ratio of the diets was analyzed in detail. Individuals enrolled in SC submitted a diet for PRUCAL analysis soon after receiving the SC program and on average, they reported a 33.8$\pm$8.0% intake of fat as a

percentage of calories and a P/S ratio of 0.64±0.42. By the end of the program their fat intake had decreased significantly to 29.0±8.0% of calories ($p<0.001$), and the P/S ratio had increased significantly to 0.78±0.51 ($p<0.001$). Since only one analysis was submitted at the end of the program by the UC group, the assumption was made that the UC diets were similar to the SC diets at baseline. For UC at the three month point, a fat intake of 30.3±7.7% of calories and a P/S ratio of 0.78±0.43 was reported. Neither of these values was significantly different from the SC levels, but both were significantly different from the assumed baseline values. While it would be expected that a decrease in total fat intake and the improvement in the P/S ratio would lead to reduced cholesterol levels, in the "stepwise" analysis total fat intake and the change in the P/S ratio never even met the default value of 0.1500 significance level for entry into the model for any of the dependent variables considered.

Two of the three remaining variables, total calories and cholesterol intake, decreased significantly from baseline but were not shown to predict changes in serum lipids (Table 5). The last dietary variable recorded, fiber intake, did not change significantly over the course of the study. The average intake remained at about 70% of the minimum goal and did not contribute to the regression equation for any of the dependent variables (Table 5).

Prescription Medications

Reported prescription drug use was significantly different for the two groups (X^2=9.3 P<0.05)). There were 16 more UC individuals than SC reporting prescription drug use. One male in SC and two males in UC were taking cholesterol lowering medications, cholestyramine or gemfibrozil, at the start of the study. Three individuals in SC and 14 in UC reported taking blood pressure medications, while 3 females in UC were taking estrogen supplements. Two females in SC reported birth control pill use, while none of the females in UC claimed to be taking Birth control pills. On final analysis, due to the small numbers using each type of prescription drug, the individuals were categorized as either "drug" or "no drug" for the statistical analysis. Analysis of the 11 individuals in SC and the 21 in UC that reported prescription drug use as mentioned above, revealed no significant relationship between serum lipid changes and prescription drug use, nor any significant difference between the two groups.

CHAPTER V

DISCUSSION

Characteristics of the Study Population

The average plasma lipid and lipoprotein cholesterol concentrations for the individuals enrolled in this study were less than the 290 mg/dl reported for those that enrolled in the LRC-CPPT study[5] but were higher than the average level of 254 mg/dl for those enrolled in the MRFIT study.[4] Still, by the NCEP's guidelines,[151] the subjects at both clinics in this study would definitely be classified as at high risk for developing CHD due to TC levels that averaged approximately 260 mg/dl and LDL-C levels in excess of 170 mg/dl. Comparison of the baseline values between SC and UC at each study location indicates that the randomization of subjects was very effective, since none of the differences were statistically significant at the 0.05 level for the USAFA participants and only the BMIs were significantly different for the PAFB subjects (Table 11). The mean BMI for the SC group at PAFB, 24.81 ± 3.79 was significantly lower than that of the UC group at PAFB 26.67 ± 4.41 ($T=2.755$, $P<0.05$). When also compared to the UC BMI at the USAFA, the difference indicates that the PAFB SC subjects were thinner than all UC participants, but their

Table 11. United States Air Force Academy and Peterson Air Force Base Diet Clinics - Comparison of the Baseline Values # of Selected Variables for Self-CARE and Usual-care.

	USAFA Diet Clinic		PAFB Diet Clinic	
Variable	Self-CARE	Usual-care	Self-CARE	Usual-care
Number	62	60	74	73
Gender (M/F)	36/26	28/32	50/24	28/32
Smoke	9	7	10	7
Age	53.60±13[a,d]	53.80±13[b,c]	44.60±13[a,c]	48.00±12[b,d]
BMI	25.25±3.33	26.15±3.79[b]	24.81±3.79[a,b]	26.67±4.40[a]
TC mg/dl	258.16±38.24	258.85±36.45	264.62±41.18	259.67±31.69
LDL-C mg/dl	174.61±35.17	172.49±30.80	183.62±35.99	175.00±30.81
HDL-C mg/dl	46.21±11.28	47.95±15.19	46.15±12.53	46.48±13.00
TG mg/dl	196.89±961.53	203.62±110.76	177.27±76.78	195.30±86.26
TC/HDL-C ratio	5.90±1.5	5.80±1.6	6.10±1.8	5.90±1.4

mean values ± standard deviation
a,b,c,d values in the same row sharing the same superscript are significantly different, $P<0.05$.
BMI = body mass index (kg/m^2 x 100)
TC = total serum cholesterol
LDL-C = low density lipoprotein cholesterol
HDL-C = high density lipoprotein cholesterol
TG = triglycerides.

average BMI was not significantly different from that of the USAFA SC group. There was no obvious explanation for this difference, especially since there were no other significant differences for the groups at each location. Further analysis of the BMI distributions did not reveal a significant age or gender interaction. That left the difference unexplained by the factors examined in this study.

While the ages of participants at each location were not significantly different from one another, there was a significant difference in age between locations (Table 11). The USAFA participants are on average 6-9 years older than the PAFB subjects. This difference is significant for both SC and UC and reflects a change in administrative policies at the USAFA Hospital. Prior to the start of this study, the USAFA Hospital made the decision to not provide routine care for retired military and their dependents that were not already receiving care at their facility. This meant that all newly retired personnel (younger) and their dependents now had to go to PAFB for care. This left a larger percentage of older individuals at the USAFA clinic, resulting in the age difference between locations. However, as revealed in the results section, no apparent age interaction was evidenced, which leaves one to conclude that the difference in age did not significantly influence the results.

No distinctions were made in this study on the basis of race. Race was not considered because the clinical management of elevated cholesterol levels should not differ according to race.[83] Minority participation was however less than 3% at the USAFA and less than 5% at PAFB. There were a considerable number of females enrolled in this study, and even though males have higher rates of CHD, individual females with elevated TC and/or decreased HDL-C levels still face an increased risk of developing CHD that approaches that of the male and are thus counseled to follow the same guidelines as males in reducing their risk.[99] Consequently, just as the guidelines of the NCEP with regards to recommendations for reducing elevated cholesterol levels and cutoff points to determine level of risk are intended for all adults 20 years of age and above,[99] this study looked at the effect of different educational approaches (SC and UC) on the entire "at-risk" population in hope of gauging overall program effectiveness. Gender was, however, considered as a dependent variable during the analysis and proved to have an independent effect only for the decrease of HDL-C for UC females. Even then the decrease for the UC females was not significantly different from that for the SC females, and there was no apparent gender interaction with any of the other parameters measured in this study. Consequently, the data was pooled for analysis.

Examination of the dietary intake values collected via a 24 hour diet record at the start of this study, at both locations, suggests that the program participants may not have been consuming a typical American diet, a diet which is said to consist of 37% of calories as fat and a P/S ratio of 0.5.[151] While a 24-hour diet record may not be representative of usual intake, the group average may give a better approximation of usual intake.[296] In both the LRC-CPPT and MRFIT studies, diet information was collected by means of a 24 hour recall.[4,5] In both of those studies, prior to any diet instruction, the individuals were consuming a diet of approximately 37-38% of the calories as fat with a P/S ratio of 0.48-0.50. In this study, the SC diets at the USAFA averaged 32.5% of the calories as fat, with a 0.62 P/S ratio, while at PAFB the SC diets averaged 33.8% of calories as fat and a P/S ratio of 0.64. These values are obviously different from the estimates for the typical American diet given above and may indicate that when the individuals enrolled in this study, they had already made some dietary adjustments.[4]

Comparison of the Results Obtained from the Self-CARE and Usual-Care Programs

Lipoprotein Analysis

At the USAFA, individuals that received UC, on average experienced a significant decrease in TC of 12.0 mg/dl or about 4.6%. A comparable reduction of 13.3 mg/dl was

achieved by the Self-CARE program. These results are interesting because they are very similar to the results reported for the pre-entry stage of the LRC-CPPT study which lasted approximately two months and for the one-year findings for the special intervention group in MRFIT. All subjects in the LRC-CPPT study, prior to randomization were instructed to adopt a moderate cholesterol lowering diet which consisted of no more than 400 mg of cholesterol per day and a P/S ratio of 0.8, and which was designed to lower cholesterol 3% to 5%.[5] In the LRC study, adoption of this diet resulted in the total fat in the diet decreasing about 3%, from 38% to 35% and the P/S ratio of the diets increasing from about 0.48 to 0.73 after two months. These changes resulted in a decrease in TC of 11.1 mg/dl (3.8%) in one group and 12.6 mg/dl (4.3%) in the other. Similar results were reported for the one-year diet changes in the MRFIT study.[4] The special intervention group in MRFIT was advised to adopt a diet pattern that recommended reducing saturated fat intake to less than 10% of calories, increasing polyunsaturated fat intake to 10% of calories and restricting dietary cholesterol to 300 mg/day; at the one-year follow-up, they had experienced a 4.3% reduction in TC. Since the fat content of the diets for the SC and UC groups at the USAFA decreased about 2-3% and the P/S ratios increased from 0.62 to 0.76 for SC and to 0.88 for UC, the results reported for the present study are not out of line with the LRC or MRFIT results. In fact, they tend to agree

with the observation that, "changing from a typical American diet to one with <30% calories from fat, 10% of calories from saturated fat, and <300 mg cholesterol will lower plasma TC levels by 5-7%."[99]

The decrease in TC for PAFB exceeded the LRC, MRFIT and USAFA changes by two to three times. At PAFB, individuals that received UC, on average experienced a decrease in TC of 29.13 mg/dl (11.2%), while an even larger average reduction of 46.48 mg/dl (17.6%) was achieved by the SC group. While considerably larger than the USAFA results, they are in agreement with the observation by Kris-Etherton et al.[99] that, "overall, plasma total cholesterol (and primarily LDL-C) levels can be lowered 10-20% with modifications in the saturated fatty acid and cholesterol content of the diet." The results are also not out of line with the studies that have suggested that changing from the typical American diet to one with <30% calories from fat, 10% of calories from saturated fat , and <300 mg cholesterol should reduce TC levels on average by 30 to 40 mg/dl.[86,151] In fact, for individuals that were serious about dietary changes, TC can be reduced another 15 mg/dl by further reducing saturated fat to <7% of total calories and dietary cholesterol to less than 200 mg/day. The results achieved at PAFB and the USAFA are certainly in line with these observations, with one exception. At the baseline measurement, the reported fat intakes as a percentage of calories for the USAFA averaged 32.5% (P/S ratio of 0.62), and 34.0% (P/S ratio of 0.64) at

PAFB, both below the 37% fat (P/S ratio of 0.50) that is typical for American diets. Since the TC decreases mentioned above were based on starting from a diet with 37% of calories from fat, what other factors could have an influence on the TC changes seen in this study?

In addition to controlling fat intake, total calories, and exercise, additional dietary modifications such as adding soluble fiber to the diet can lead to additional reductions in TC of from 1% to 10%.[99,280] At both of the study locations, over one-half of the participants reported consuming oat products on their 3 month diet record. While at the USAFA, 58.2% of SC and 61.5% of UC reported consuming oat products, the relationship between the change in TC and oat consumption never reached statistical significance ($F=2.95$, $P=0.0923$). At PAFB, 62.3% of the SC participants and 48.4% of those in UC reported consuming some form of oat products, but the relationship between TC and oat consumption never even approached statistical significance. These findings are not that surprising however, since any mention of oat products in a diet was recorded as oat consumption, some of those classified as consuming oat products may not be consuming levels of the product that would effect TC.[280] This observation receives some support from the fact that the total fiber intake did not change significantly at either location during this study and averaged only 65-75% of the dietary goal at the USAFA and approximately 70% at PAFB. A similar argument could be made

for the lack of correlation between dietary fat as a percentage of calories and/or P/S ratio and the change in TC. The dietary measurements may give an overall picture of the study population, but the 24 hour diet record is not an accurate enough reflection of an individuals usual intake to look at the relationship between an individual's changes in serum lipids and dietary intake.[297]

The typical response to decreasing saturated fat intake and increasing the polyunsaturated fat and carbohydrate intake, as is done when adopting the Step-1 diet, is for the TC, LDL-C and HDL-C to decrease.[302] Even though the HDL-C decrease is not desired, overall the change is beneficial because the decrease in LDL-C is much larger than that for the HDL-C. The end result is that TC and LDL-C levels are decreased, but the TC/HDL-C ratio will decrease slightly or remain the same. This is exactly what happened at PAFB with both SC and UC and to a lesser degree at the USAFA for UC and SC. At PAFB, while TC decreased 11.2% for UC, HDL-C decreased only 8.9%, the result of which was a small but nonsignificant decrease in the TC/HDL-C ratio. For SC, TC decreased 17.6%, and HDL-C decreased only 6.6%, resulting in a significant decrease from baseline in the TC/HDL-C ratio ($T=-2.36$, $P<0.05$) (Table 9). In both SC and UC at PAFB, risk status based on TC decreased from "high blood cholesterol" to "borderline-high", while the TC/HDL-C ratio stayed the same or decreased. The changes at the USAFA for SC and UC were not as large as those at PAFB, but showed the

same general trends as discussed above. Only the UC group at the USAFA had a significant decrease from baseline in the TC/HDL-C ratio ($T=-2.36$, $P<0.05$). The decrease was from 5.8 to 5.5 and while it was statistically significant, its practical significance is questionable.

While there is extensive information on the significance of elevated TC, what is the significance of the TC/HDL-C ratio? The NCEP does not directly address the question of TC/HDL-C ratios and levels of risk, but the Air Force does, and classifies individuals with a ratio greater than 4.5 as at increased risk.[269] The consensus developing in the medical community is that a ratio of 4.5 represents average risk, and above 6.0 high risk.[299] Levy and Kannel[57] claim that the Framingham data has made it clear that both TC and HDL-C levels must be examined when attempting to assess overall risk. They conclude that the Framingham data has shown that the TC/HDL-C ratio should be <4.5, unless the TC is <150 mg/dl.

At PAFB, for the SC and UC groups, the decrease from baseline for LDL-C was significant, for both SC and UC groups. At the USAFA, only the 5% decrease in LDL-C for the SC group was significant. The biological and behavioral factors affecting LDL-C are basically the same ones that affect TC, for example diet, BMI, activity level and individual responsiveness, since most of the dietary induced changes in TC are a result of decreased LDL-C.[99] In this case, the LDL-C decrease at PAFB of 21.4% for SC and 12.3%

for UC is slightly greater than the TC decrease, whereas the 5% decrease at the USAFA is approximately equal to the TC change. While the Framingham researchers claim that TC is as good as LDL-C for assessing CHD risk,[57] the NCEP after initial screening, bases treatment recommendations on a combination of LDL-C levels and risk factors present.[83] Although it is supposedly the LDL-C that exerts the atherogenic effect, in a majority of the cases, individuals with elevated TC levels will also have elevated LDL-C levels, and since the LDL-C values will have usually been calculated using the formula:

LDL Cholesterol= (TC)-(HDL Cholesterol)-(Triglycerides/5),

rather than directly measured, there is really little to be gained from using LDL-C values for risk assessment.[41] So for risk assessment with individuals, especially in men and women 50 and older, the focus on both TC and HDL-C levels is appropriate.[300]

Body Mass Index (BMI)

The BMI changed significantly from baseline at both locations and for both treatments. The BMI is an important variable, because of its role as an indicator of obesity, which has been shown to be an important contributor to CHD. Changes in relative weight have also been shown to be directly correlated with risk factors such as TC and TC/HDL-C ratio.[58,99,295] For example, on average each 2 pounds of excess body fat contributes 1 mg/dl of TC.[298] However, while

at the USAFA the analysis of the data showed no significant relationship between initial BMI or the change in BMI and any of the changes in the serum lipids, at PAFB, analysis of the data showed a consistent, significant relationship between the changes in BMI and changes in TC, LDL-C and the TC/HDL-C ratio. The findings at the USAFA can best be explained by considering the relatively small changes in serum lipids and weight changes that amounted to only 3-4 pounds coupled with the observation that while changes in risk factors follow the changes in BMI, linear trends do not become apparent until the weight change approaches 15 pounds.[57] Yet at PAFB, there was a strong consistent relationship in spite of the relatively small weight change of only 3-4 pounds. It appears that perhaps the relatively large changes in serum lipids were the difference because of a consistent pattern: namely individuals that experienced a 2-4 pound weight loss also seemed to experience a decrease in their TC. This pattern was supported by the highly significant relationship shown between BMI change and TC change (P=0.0018), LDL-C change (P=0.0450) and change in the TC/HDL-C ratio (P=0.0050). While the data from the USAFA does not show the strong relationship found at PAFB, the BMI as revealed by data from the Framingham Heart Study is an independent risk factor and it is important for individuals to maintain a BMI <22 (kg/M^2).[57] This may be very difficult for the majority of the "high-risk" individuals in this study. At PAFB, only 18.9% of the individuals in SC and

6.9% of the individuals in UC entered the study with a BMI less than 22, while, at the USAFA, only 16.1% of the individuals in SC and 15.0% of the individuals in UC entered the study with a BMI of less than 22.

Program dropout

The anticipated dropout rate of 15% for this study was an optimistic estimate to begin with, when compared to rates of 20% to 80% that have been reported in other studies of the treatment of hyperlipidemia.[227,238,239] The 15% figure was chosen after consideration of the military background of the subjects and the ability to locate and follow-up program participants. As it turned out, the retention rate at both locations was excellent. At PAFB, only 9.5% of the SC and 12.3% of the UC participants chose not to complete their program, compared to 8.3% of the UC and 11.3% of the SC participants at the USAFA. Of those that did not complete their program, there was almost a 50/50 split between those that had significant medical or personal problems and those that were just not interested or did not want to take the time.

The drop-out rates were low, but they must be examined realizing that built into each program was a follow-up procedure designed to encourage individuals that had missed their three month recheck to complete the program. For SC at the USAFA, 15% of the subjects or nine individuals required additional phone and/or mail notification. Of

those nine, four did not complete the study. Three times as many UC individuals, or 45% required additional notification. The 27 individuals that were notified responded well and only three choose not to complete the study. At PAFB, 34% of the SC subjects or 25 individuals required additional phone and/or mail notification. Of those 25, three did not complete the study. Almost twice as many UC individuals, or 58% required additional notification. The 42 individuals that were notified responded well and only five chose not to complete the study.

The large difference in the need for additional notification between SC and UC at each location only serves to reinforce the observation that, without periodic follow-up, many individuals will lose interest and may drop-out of intervention programs.[265] The monthly contact through the computer diet analysis with the individuals in SC seemed to result in better compliance with the program. In fact, the importance of continued follow-up is evident when you consider that without the additional follow-up that was built into the program, the dropout rates at the USAFA could have been as high as 21% for SC and 50% for UC and as high as 38% for SC and 63% for UC at PAFB if none of the delinquent individuals had returned for their follow-up check. Levels of this magnitude would be more in line with the dropout rates seen in other studies.

Dietary Intake, Exercise and Smoking

The data collected on the consumption of alcohol, fiber, fat and oat products, in addition to smoking and exercise was of interest because of the link between these behavioral factors and CAD. High intakes of oat products and other soluble fibers have been shown to decrease TC,[301] while a moderate intake of alcohol may increase HDL-C.[302] Exercise has been shown to be associated with lower levels of TC and increased HDL-C levels, although the contribution of exercise induced weight loss complicates the picture,[295] and smokers have been shown to have a twofold increased risk of CHD compared to nonsmokers.[57] In spite of these observations, no statistically significant relationships were found between any of these factors and changes in any of the dependent variables at either location. However, as mentioned earlier, this may be due in part to the low level of reliability in the dietary measurements used and the lack of quantitative measures of consumption.[297]

Location Characteristics

The large difference in results between the two locations dispensing essentially the same information is intriguing. If the SC program had not been included in this study, the conclusion that would have been reached from comparing the results of only the two UC programs would have been that group nutrition counseling produces significantly better results than does individual diet counseling.

Fortunately, the large difference in the results obtained using the SC at the two locations revealed that there were other factors at work. After a careful analysis of the locations, the laboratories, and a review of the procedures followed, several factors became apparent. The first of these is that there was a difference between the interest and involvement of the Health Care Providers (HCP) at each location. At the USAFA hospital, the HCPs knew little about the SC program or the experiment being conducted. Consequently, the patients knew nothing about this trial until they met with the dietitian.

Due to the smaller size of the staff and the direct involvement of several of the HCPs in the planning of the study at the PAFB clinic, individuals identified as at risk would often know in advance that a "special" nutrition education program would possibly be available. In effect, at PAFB the SC program had some publicity. The next major difference as detailed in the methods section involved the enrollment of the subjects.

At the USAFA, all direct contact was strictly between the dietitian and the subjects enrolling in the study. At PAFB, it became apparent as the results accumulated that perhaps the personal contact between the author and the individuals enrolling in the study had produced a sort of Hawthorne effect,[14] where these individuals felt that they were being given special attention and as a result worked harder at modifying their diets.

Close attention was also paid to laboratory measurements during both the planning and implementation of this study. Any cholesterol measurement is affected by biological, behavioral, clinical and analytical sources of variation.[303] Since this study was interested in the relative changes in cholesterol produced, and used UC as a control, accuracy of the measurements while important, was not as critical to the study as precision. Accepting 18 mg/dl as a good estimate of the standard deviation of repeated measurements over time for TC[83] and assuming that biological and seasonal variation, well important for determining individual cholesterol values accurately, would not have a significant effect on the results of this study, close attention was paid to the average day-to-day precision (coefficient of variation) or reproducibility of the measurements in the two laboratories in this study. Poor precision would mean that it would be difficult to detect small changes in cholesterol levels in the study population. The average coefficients of variation reported by the USAFA laboratory were 2.21 for TC, 1.79 for HDL-C, and 2.35 for TG. The USAFA lab exceeded the ideal goal of 3% for TC established by the Laboratory Standardization Panel (LSP).[258] The laboratory at the PAFB clinic reported coefficients of variation of 4.8 for TC, 9.2 for HDL-C, and 4.3 for TG. While not as precise as the USAFA lab, the PAFB lab exceeds the acceptable precision goal of 5% for TC established by the LSP, and both labs had a CV of less than

the 8% that was used in determining the sample sizes for this study.

Summary

The study at PAFB revealed that both the SC and UC programs produced a significant improvement in the average lipid profile of the participants (Table 9). In addition, there was a clear treatment effect, with SC producing significantly greater overall changes in TC (P=0.0121), TC/HDL-C (P=0.0378), and LDL-C (P=0.0010) than those produced by UC. The treatment effect remained significant throughout the statistical analysis. Adjustment for the sex, age, and initial BMI of the subjects did little to diminish the effect. The initial BMI was examined carefully because of the significant difference in BMI between the SC and UC groups. The initial BMI did not appear to have a significant influence on changes in any of the serum lipids that were found to be significantly different between treatments. On the other hand, the change in BMI as mentioned earlier showed a strong and consistent relationship to the changes in serum lipids independent of treatment. This observation would be expected, since if the treatment was effective and individuals both decreased their fat consumption, decreasing caloric intake, and improved their P/S ratio, they would be expected to both lose some weight and improve their serum lipid profile.[151,302]

The significant changes noted at PAFB in both of the major endpoints, TC and TC/HDL-C ratio, that were the focus of this study, on average moved the participants out of the high-risk category. While both programs were effective, the SC approach produced significantly larger changes in TC and the TC/HDL-C ratio. This then would indicate that in this type of clinical setting, a self-care approach could be successfully substituted for group diet instruction as a part of a preventive cardiology program.

At the USAFA, the only consistent change noted in the major endpoints specified at the beginning of this study was the decrease in TC for both SC and UC (Table 3). The changes were significantly different from the baseline values and represented changes of 5.1% for SC and 4.7% for UC. In any case, these results demonstrate that individual diet counseling contributes to the reduction of TC in an at risk population, and that a self-instruction method coupled with periodic feedback appears to be equally effective. These findings would support continued diet counseling for at risk individuals and would also support the use of a self-instruction diet education program where needed.

The benefits to be derived from a preventive program become apparent when you consider the costs involved. At PAFB, the changes in TC represented decreases of 17.6% for SC and 11.2% for UC, and at the USAFA, TC decreased 4.7% for UC and 5.1% for SC. Using the paradigm developed from the LRC-CPPT study that a 1% reduction in TC would equal a 2%

reduction in CHD rates,[83] these decreases if they were to be maintained would translate into a decrease in the CHD rate of from approximately 9.2% to 10.4% for the USAFA and from 22% to 36% for PAFB. If decreases of this magnitude were to be attained in the active duty force, "where the annual cost directly attributable to CAD is approximately $70 million per year",[292] the economic savings would be substantial.

CHAPTER VI

CONCLUSIONS AND RECOMMENDATIONS

Conclusions

The specific changes in lipid values and the significance of the changes have been explained and discussed in detail. Table 12 provides a review of the results for the two locations. All changes are presented as percentages, to allow easy comparison of the major findings, and is appropriate since the baseline values of the serum lipids were not significantly different. This also helps to summarize the results of this study. Basically, at the USAFA clinic, SC and UC were equally effective, producing a small but significant change in TC. At the PAFB clinic, both SC and UC produced significant changes in the lipid profile, changes that substantially reduced the risk of CAD. These changes were for the most part significantly greater than the changes at the USAFA.

With these results in mind, it is time to reexamine the specific objectives of this study. The objectives for the SELF-CARE study were as follows:

1. To assess the effectiveness of "usual care" in modifying serum cholesterol levels at the two medical facilities in this study,

Table 12. Summary: Percentage change in Selected Variables for Self-CARE and Usual-care at United States Air Force Academy (USAFA) and Peterson Air Force Base (PAFB) Diet Clinics.

	USAFA Diet Clinic		PAFB Diet Clinic	
Variable	Self-CARE	Usual-care	Self-CARE	Usual-care
TC	-5.1#[a,b]	-4.7#[c,d]	-17.6#[a,c,e]	-11.2#[b,d,e]
HDL-C	-2.7[a]	+0.7[b,c]	-6.6#[c]	-8.9#[a,b]
TC/HDL-C	-2.7[a]	-6.3#	-12.6#[a,b]	-3.6[b]
LDL-C	-5.1#[a,b]	-4.8[c,d]	-21.8#[a,c,e]	-12.3#[b,d,e]
BMI	-1.8#	-1.6#	-1.7#	-1.3#
TG	-9.3	-16.5	-7.9	-8.1

significant change from the baseline value, $P<0.05$.
a,b,c,d values in the same row sharing the same superscript are significantly different, $P<0.05$.
BMI = body mass index (kg/m^2 x 100)
TC = total serum cholesterol
LDL-C = low density lipoprotein cholesterol
HDL-C = high density lipoprotein cholesterol
TG = triglycerides.

2. To assess the effectiveness of the SELF-CARE nutrition education program in modifying serum cholesterol levels at the two facilities in this study, and to

3. To determine if the SELF-CARE program can be considered a viable alternative to "usual" care.

With these three objectives in mind, this study tested the following hypothesis:

Individuals following the SELF-CARE program will experience an average reduction in total serum cholesterol, and/or improvement in the total cholesterol/HDL-C ratio that is equal to or greater than the improvements provided by "usual care".

After reviewing the data, the conclusion is that this hypothesis has been proven to be true, and that the SELF-CARE program because of its' simplicity, low cost and effective use of time and manpower, should be considered for expanded usage and long term evaluation.

Trying to estimate what effect this program would have in another location would depend on many factors. It is obvious that the degree of involvement by the program coordinators and the support provided by the medical staff plays an important part in this program's success. The results from the USAFA would in my opinion provide an indication of the results that could be expected if the Self-CARE program were to be put into general use. The results from the PAFB clinic are excellent and would have external validity, but only in an environment where the

health care providers, dietitian and administration had made an enthusiastic commitment to preventive cardiology.

Recommendations

1. The SELF-CARE program because of its simplicity, low cost and effective use of time and manpower, should be considered for expanded usage and long term evaluation.

2. Distribution of the Self-CARE program should be handled by the HCPs. This would make the process much more timely and improve efficiency. Individuals learning that they are at risk for CAD would not have to make an appointment and then another trip to the clinic days or weeks later to receive diet instruction. Issuing of the program would be considered a prescription for diet counseling, providing a natural path for follow-up.

3. The best use of the Self-CARE program would be as a targeting tool. The program could help target individuals, directing them towards the type of follow-up that would be most effective for their particular needs.

4. Further research should include a one-year follow-up to determine the extent of long term changes made as a result of the Self-CARE program and possibly to compare those long-term changes to those for the usual-care programs. Additional study is needed to determine what is the most effective method of providing reinforcement during the study; for example, would a phone call be as effective as a diet analysis? Finally, both the Self-CARE program and the various types of usual-care should be compared to "no-care" groups to determine the true effectiveness of these interventions.

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APPENDIX A

PROJECT APPROVAL

COLORADO STATE UNIVERSITY

Committee on Human Research

PROJECT APPROVAL FORM

Project Title: Self-Care for a Healthy Heart - Lowering Cholesterol in an "At Risk" Population

Principal Investigator: Dr. G. Richard Jansen

Co-Investigator: Jeffrey M. Johnston

Department: Food Science & Human Nutrition

Agency: N/A

Agency Deadline Date: N/A

Date of Project Initiation: March 1, 1988

The above project was examined by the Committee on Human Research on 3/17/88 (Date) with the following recommendation:

☐ Project approved with no conditions.

☒ Project approved with the condition that an approved consent form must be used.

☐ Project conditionally approved if the following conditions are met:

_____

_____

_____

_____

Alan Tucker

Chairman
Committee on Human Research

3/17/88

Date

DEPARTMENT OF THE AIR FORCE
AIR UNIVERSITY
AIR FORCE INSTITUTE OF TECHNOLOGY
WRIGHT-PATTERSON AIR FORCE BASE OH 45433-6583

19 Apr 88

REPLY TO ATTN OF CIS

SUBJECT Research Proposal Approval

TO Major Jeffrey M. Johnston
10980 Thomas Road
Black Forest CO 80908

Your research proposal is approved and the $200.00 research fee will be processed for payment.

JAMES H. PARSONS, Lt Col, USAF
Chief, Special Programs Division

APPENDIX B

PERMISSION LETTERS

Colorado State University

Cooperative Extension
Colorado State University
Department of Food Science and Human Nutrition
200 Gifford Building
Fort Collins, Colorado 80523
(303) 491-7334

May 17, 1989

MEMO

I give Major Jeffery Johnston (USAF) permission to use any Healthy Heart materials, including the logo, for the Healthy Heart Self Care Program.

Jennifer Anderson

Jennifer Anderson, Ph.D., R.D.
Assistant Professor and
Food and Nutrition Extension Specialist
Department of Food Science and Human Nutrition
Colorado State University

Colorado State University

Department of Food Science
and Human Nutrition
Fort Collins, Colorado 80523
(303) 491-6535

To: Dr. T. Lasater 24 February 1988

From: Jeffrey M. Johnston (303-491-7322)

Subject: Permission to reproduce material

I am preparing a program entitled SELF-CARE for a Healthy Heart as a part of my dissertation project. This program will be compared to "usual care" at three Air Force clinics.

I wish to have your permission to include in my program the following material from the Pawtucket Heart Health Program Nutrition Kit (Steps 1-4):

1. Selected questions from the "rate your present eating pattern" and the "where do you stand" sections.

2. Selected questions from the "check the boxes when you have made the change" sections.

If permission is granted for the use of this material, the Pawtucket Heart Health Program will be credited as the source.

Sincerely yours,

JMJ Johnston

Permission Granted:

(Signature)

3-11-88

(Date)

UNIVERSITY OF GUELPH
COLLEGE OF FAMILY AND CONSUMER STUDIES
Department of Family Studies

GUELPH ONTARIO CANADA N1G 2W1
Telephone (519) 824-4120

Rerum
Cognoscere
Causas

February 3, 1987

Jeffrey M. Johnston
Department of Food Science and Nutrition
214 D Gifford
Colorado State University
Fort Collins, CO 80523
U.S.A.

Dear Mr. Johnston:

I am enclosing a copy of the research instrument and conceptual framework we used for the study reported in the Journal of Nutrition Education 18(6):253, 1986 as you recently requested.

If you do use or adapt this instrument we ask that you acknowledge its source.

Thank you very much for your interest in our work.

Sincerely yours

Donna M Woolcott

Donna M. Woolcott, PhD
Associate Professor
Applied Human Nutrition

encl.

APPENDIX C

Self-CARE PACKAGE

WHAT CAN I DO? 1

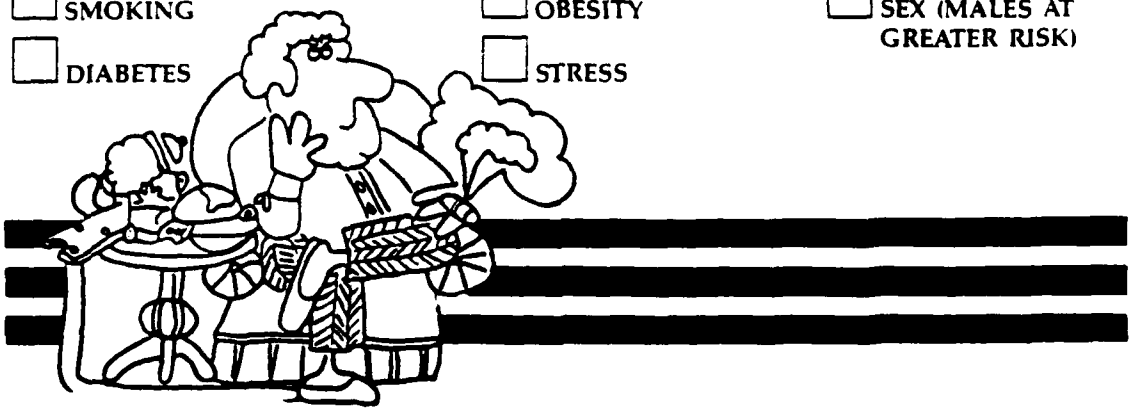
R I S K F A C T O R S

Are you at risk for developing coronary artery disease?

Check the risk factors below that you think apply to you:

These you *can change* . . . . . > These you *can not* change

(1) Preventable or controllable	(2) Contributing factors	(3) Not preventable
☐ CHOLESTEROL LEVELS	☐ FOOD HABITS	☐ FAMILY HISTORY
☐ HIGH BLOOD PRESSURE	☐ LACK OF EXERCISE	☐ INCREASING AGE
☐ SMOKING	☐ OBESITY	☐ SEX (MALES AT GREATER RISK)
☐ DIABETES	☐ STRESS	



How many risk factors applied to you?

See *"Facts. . .for Self-CARE"* (last page of Step-1) for a description of these risk factors and how they are especially dangerous in combination.

Now let's focus on the reason you are completing the *Self-CARE* program — your cholesterol is high and you want to do something about it. Fortunately, many individuals can reduce their cholesterol to a safe level by making a few changes in what and how they eat. The *Self-CARE* program was designed to help you make these changes.

Turn the page to get started

HOW CAN I DO IT? 1

F O O D R I S K F A C T O R S

You can usually decrease your cholesterol levels by making *just a few changes* in what and how much you eat.

WHAT ARE THE FOODS YOU SHOULD MODIFY:

Fats: Where do you find them? In meat, dairy products, and processed foods like crackers, cakes, and chips.

Too much fat in the food you eat can end up as fatty deposits in veins, arteries, and the heart.

Cholesterol: Where do you find it? Found only in foods of animal origin.

A high level of cholesterol in the blood has long been associated with heart disease. One factor that contributes to high serum cholesterol is diet — *eating a lot of fat.*

Sugar: Where is it found? Naturally in fruits and vegetables and hidden in many processed foods.

Sugary foods provide mainly calories without vitamins or essential nutrients and contribute to obesity and elevated levels of fat in the blood.

When you make modest dietary changes, your risk for heart disease should decrease.

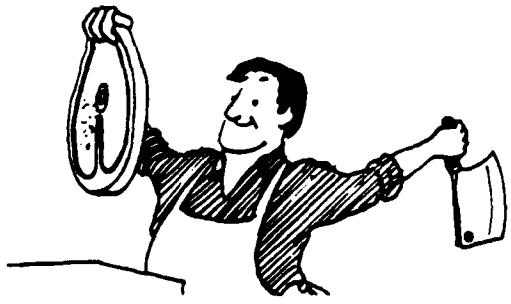
☐ On the risk chart below find your age and then circle your Total, LDL, and HDL Cholesterol levels; and your Total Cholesterol/HDL ratio. (See the inside cover of this package for your levels.)

Heart Disease Risk Chart for Cholesterol Levels

		EXCELLENT PROTECTION	INCREASED RISK	HIGH RISK
LEVELS	Desirable			
Total*	20-29	< 200	201-220	> 220
Cholesterol	30-39	< 220	221-240	> 240
mg/dl	40+	< 240	241-260	> 260
LDL**	20-39	83-117	118-137	> 144
Cholesterol				
mg/dl	40-59	104-141	142-162	> 183
HDL**	20-39	> 51	45-51	< 37
Cholesterol				
mg/dl	40-59	> 45	35-45	< 31
Total**	20-39	2.7-3.6	3.7-4.5	> 5.0
Cholesterol				
HDL ratio	40-59	Total 2	4.3-5.0	> 5.1

* Total levels from ABC Inc.[illegible]

** Total levels adjusted from Coronary Cholesterol [illegible] K.H. Cooper, Aerobics, Inc., NY



The diet changes suggested in this program should move you toward the **Excellent Protection** category.

At your 3 month follow-up, check this chart again to determine how much progress you have made in reducing your risk of coronary artery disease.

Healthy Heart Eating does not mean you have to give up what you enjoy!!!

Read on for more information.

MORE THAT I CAN DO 1

"DO NOT GIVE UP . . . MODIFY"

This program will show you several ways that you can change your eating style so that heart risk factors are *decreased* and heart health factors are *increased*.

This means that you *do not give up* what you enjoy eating but, instead, substitute something that is more beneficial to heart health. The key phrase is —

"DO NOT GIVE UP...MODIFY"

HERE ARE SOME SUGGESTIONS:

What You Eat:

1. Eat only the *calories* required to meet your individual energy needs.
2. Reduce *total fat calories*.
3. Reduce *cholesterol* rich foods — if your intake is high.
4. Meet your *carbohydrate* needs with starches rather than sugar.
5. Increase *nutrient-dense* and *fiber-packed* foods.

Good suggestions, but do you have any idea of how many calories you need or how much fat you are eating?

What is your carbohydrate or fiber intake?

LOOK TO PRUCAL FOR HELP

☐ To help make you aware of what you are eating and to provide a baseline from which you can judge your progress, please complete the yellow *prucal* diet analysis forms that you will find in the front pocket of this package.

All the instructions are on the form — but please remember the analysis requires candidness. The analysis is an aid for you, not a report card. The only time your record will even be seen by anyone else is when the program leader does your computer analysis.

PRUCAL

Developed and Copyrighted
by
Colorado State University
Department of Food Science and Human Nutrition

Diet Analysis for:	Healthy Heart
Age:	32
Height:	6 feet 0 inches
Recommended Weight:	162 pounds
Sex:	Male
Frame:	Medium
Activity:	Moderate

For some more information on "risk factors," turn to the last page, then move on to Step-2 so you can learn how to cut down on fat & cholesterol.

FACTS FOR SELF CARE 1

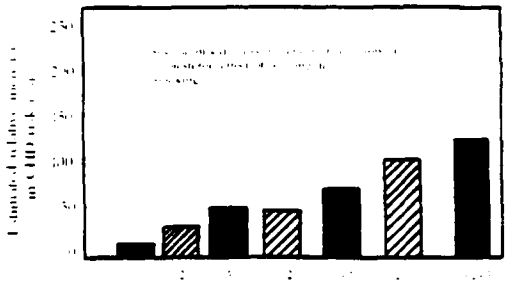
H E A R T R I S K F A C T O R S

Changing your risk means lowering it, not eliminating it.

Imagine that in the highest risk group, 7 out of 10 men will have heart disease, and in the lowest risk group only 1 in 10. There will always be some men who do all the wrong things but never develop heart disease. And there will always be that one man who seems to do everything right but has heart disease. It is a question of statistical chance, as in Russian roulette. If you had to put a 10 chamber gun to your head and pull the trigger once, would you rather play with *one* chamber filled, or with *seven?*

The following chart illustrates how your risk can decrease as you change the combination of risk factors present.

SYNERGISTIC INTERACTION OF RISK FACTORS



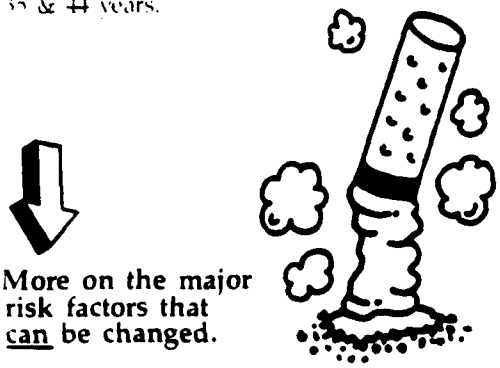
(THE FRAMINGHAM HEART STUDY, 1957)

More on the major risk factors that cannot be changed.

Heredity — It appears that a tendency towards heart disease is hereditary. A family history of heart disease increases your risk.

Sex — Men have a greater risk of heart attack than women.

Age — 60% of the USAF cardiovascular disease occurs in persons between the ages 35 & 44 years.



More on the major risk factors that <u>can</u> be changed.

Blood cholesterol levels — Too much cholesterol can cause buildups on the walls of arteries, narrowing the passageway through which blood flows, leading to heart attack and stroke.

High blood pressure — A major risk factor of stroke and heart attack, high blood pressure usually has no specific symptoms.

Cigarette smoking — The heart attack death rate among people who do not smoke cigarettes is considerably lower than for people who do smoke.

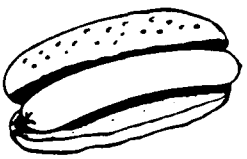
Now on to Step-2, so you can learn how to cut down on dietary fat and cholesterol, and lower your risk for heart disease.

WHAT CAN I DO? 2

DECREASING FATS & CHOLESTEROL

This step will help you cut down on saturated fat and cholesterol.

"For healthy people, *moderation* in fat intake should become the rule of thumb!! Fats, irrespective of their source, are of high caloric value."



First, rate your present eating pattern and check the most appropriate column:	MOST DAYS	EVERY 2 OR 3 DAYS	SELDOM
How often do you eat fatty meats like hot dogs and luncheon meats?	☐	☐	☐
How often do you eat or drink high fat dairy products like whole milk and/or butter?	☐	☐	☐
How often do you eat hard cheeses like swiss and cheddar by themselves or in sandwiches and sauces?	☐	☐	☐
How often do you eat egg yolks, including those used in cooking (cakes, custard...)?	☐	☐	☐
Do you eat snacks and processed foods without reading the ingredients and nutrition labels?	☐	☐	☐

The checks in the *MOST DAYS* column will show you where you should begin your efforts to reduce the fat and cholesterol in your diet.

The questions above illustrate that fats and cholesterol are found in a variety of foods, and are often hidden, especially in snacks and desserts.

What is your fat intake and how does it compare? Find your fat intake on page 2 of your PRUCAL diet analysis —

Your Fat Intake (%)	_____	Goal	30%
Saturated Fat (%)	_____	Goal	10%
Polyunsat. Fat (%)	_____	Goal	10%

Turn the page and you will find information that can help you determine the best way for you to modify your fat intake.

HOW CAN I DO IT? 2

MEAT FATS & CHOLESTEROL

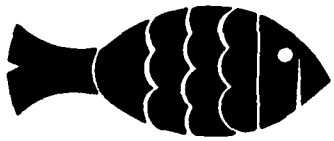
The first & most basic step to heart health is to *eat in moderation.*

On page-2 of your PRUCAL diet analysis you were able to get a general idea of how your total fat and cholesterol intake compared to your goals. Most Americans get about 40% of their calories from fat, while 30% would be a healthier level.

A good way to begin to *reduce* your fat and cholesterol intake is to *reduce the serving size* of the meat in your diet.

Check the boxes below for the changes you plan to adopt to reduce your cholesterol and meat fat intake:

☐ Buy or order the leanest cuts of meat. Trim off visible fat. Broil meat rather than fry. *(Hint: Chicken and turkey are very lean once you remove the skin. Fish has almost no saturated fat.)*



☐ Eat only lean meats and use smaller portions. *(Hint: All meats that have been breaded and deep-fried are high in fat.)*

☐ Cut down on luncheon meats, bacon, & sausage. *(Hint: Use sliced beef or turkey in place of cold cuts and decrease the serving size.)*

☐ Limit egg yolks to three a week, including those used in cooking. *(Hint: Egg whites contain no cholesterol. Try using two whites and one yolk for your egg dishes.)*

Trimming Fat — Can it really help??

As an example, if you were a man consuming 3000 calories/day, and as is typical 40% of that from fat, you could reduce your fat intake to 35% by simply trimming the fat, which contains 150 calories, from an average size pork chop.

*******Trimming the fat helps, but Watch Out* for the fat you *CAN NOT SEE:* The fat in foods such as hot dogs, mayonnaise and deep-fried chicken and fish products.

CHANGE COMES S-L-O-W-L-Y. Use the four changes suggested in this section to help cut down on some of the high fat foods you and your family are used to eating.

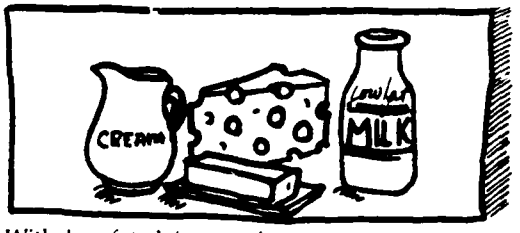
Moderation is the key — make a change in your approach to food selection, a change you and your heart can live with.

Turn the page and you will find several more changes you may want to adopt to help you lower your fat and cholesterol intake.

MORE THAT I CAN DO 2

DAIRY FAT & HIDDEN FAT

All dairy fats are highly saturated. (see "Saturation Facts" on the next page) and can raise the levels of cholesterol in your blood stream. Luckily, it's easy to buy dairy products in a low-fat form, or to find substitutes.



With low-fat dairy products you get fewer calories, less saturated fat, and cholesterol without loosing the nutrients.

Check the boxes below as you make each of these changes:

☐ Switch from whole milk to low-fat milk (1%-2%). *(Hint: Take 2 or 3 weeks for this step. Start by mixing whole milk with low-fat; then drink low-fat alone. If you are already drinking low-fat, terrific, read on for some other ways to cut down on fats.)*

☐ Cut down on high-fat cheeses. Most cheeses are high in fats (except low fat cottage, ricotta, and mozzarella made with part skim milk). Use only occasionally and do not add cheese to sandwiches made with meat. *(Hint: Use sharp flavored cheeses so you can use less cheese for more flavor.)*

☐ Cut down on cream, ice cream, sour cream, etc. Frozen low fat yogurt, sherbet, and ice milk are all much lower in fat than ice cream. *(Hint: Plain low-fat yogurt is an excellent substitute for sour cream in cooking.)*

Fats in processed foods are prime examples of *hidden fats.* While most vegetable fats are naturally unsaturated, those in processed foods are often saturated — particularly palm oil, coconut oil, and liquid oils which have been "hardened" or "hydrogenated." (See "Saturation Facts" on the next page.)

to avoid hidden saturated fats. Ingredients are listed in order of weight in a product. Avoid products whose labels list as the first ingredient an oil that has been "hardened" or "hydrogenated," and those made with coconut or palm oil. *(Hint: Choose products listing a liquid oil first, e.g. soy, corn, or sunflower.)* See Step-3 for more information on labels.

You now have most of the information you need to reduce your cholesterol.

Step 2 concludes with a description of the fats in our foods and will help you understand the terms you see on labels and in the news.

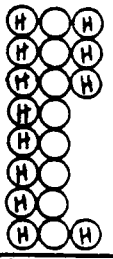
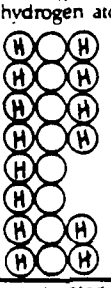
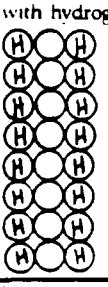
Please turn the page.

FACTS FOR SELF CARE 2

S A T U R A T I O N F A C T S

"Saturated" and "unsaturated" are chemical terms that describe the fats in our foods. They refer to how much hydrogen is attached to the fat molecule.

HERE IS A RUNDOWN ON FATS AND OILS AND HOW THEY AFFECT YOUR HEALTH

	Polyunsaturated fats (oils) are missing many hydrogen atoms	Monounsaturated fats (oils) are missing only two hydrogen atoms	Saturated fats are those that are filled with hydrogen
Different fats have different amounts of hydrogen:			
How they may affect our health:	In most people monounsaturated & polyunsaturated fats tend to *lower* blood cholesterol		In most people, saturates tend to *raise* cholesterol
At room temperature:	Polyunsaturated and monounsaturated fats are liquid (so we call them oils)		Saturated fats are usually solid or firm
Where they come from:	Mostly from plants	Mostly from plants	Mostly from animals but also some from plants
Examples:	Safflower oil Corn oil Sunflower oil Soybean oil Cottonseed oil Sesame oil	Olive oil Erucic oil or Rapeseed (found in Puritan oil)	Fat in meat Butter Lard Cheese Whole milk Cream	Coconut oil Palm oil Hydrogenated vegetable oil

CONGRATULATIONS — You have completed the most difficult part of this program and you are now on your way to lower cholesterol levels.

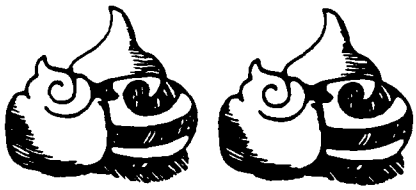
Please *DO NOT RUSH ON* — review the changes suggested in Step 2 and make the applicable ones a permanent part of your eating style

Now on to Step 3 for some suggestions on how to cut down on the "empty" calories in your diet.

WHAT CAN I DO? 3

REDUCING CALORIES

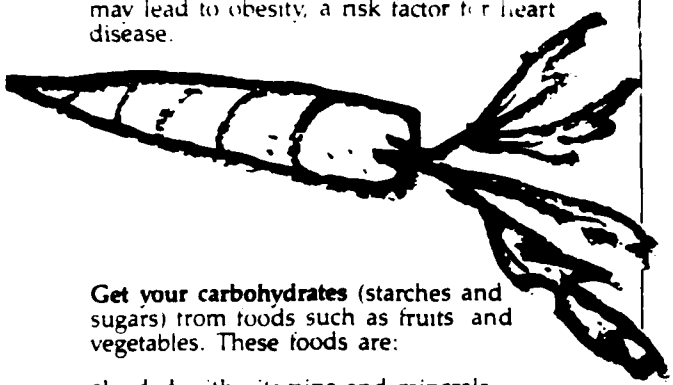
This step will show you how to cut down on the "empty" calories from sugar and increase the proportion of complex carbohydrates and fiber from starchy foods, vegetables, and fruits.



Simple carbohydrates, such as sugar and honey, have little nutritional value, providing only sweetness and calories — no minerals, vitamins, or fiber — *Empty Calories.*

♥

Extra sugar in your diet is stored as fat and may lead to obesity, a risk factor for heart disease.



Get your carbohydrates (starches and sugars) from foods such as fruits and vegetables. These foods are:

- loaded with vitamins and minerals
- filling without many calories
- a good source of fiber.

DOES THE CARBOHYDRATE & FIBER CONTENT OF YOUR DIET MEASURE UP?

	YES	NO
Do you often eat pre-sweetened cereals?	☐	☐
Do you often drink regular soft drinks?	☐	☐
Do you avoid bread, potatoes, rice?	☐	☐
Do you often skip vegetables at dinner?	☐	☐
Do you often eat cookies, pastries, or candy?	☐	☐
Do you eat a sugary dessert more than twice a week?	☐	☐
Do the "Total Sugars" on your PRUCAL diet analysis exceed 20% of calories?	☐	☐

Any checks in the YES column show where you can improve.

Step 2 showed you how to reduce the fat in your diet. Now go on to the next page. There you will find some suggestions on how you can satisfy your calorie needs using complex carbohydrates.

HOW CAN I DO IT? 3

C A R B O H Y D R A T E S

First, check your PRUCAL diet analysis. Your present carbohydrate intake is _____% (goal 57%), your total sugar intake is _____% (goal < 20%).

Most diets are high in *Total Sugars*, but low in *Total Carbohydrates*. How is yours?

For help in this area, check each box below when you feel confident that you have made the change.

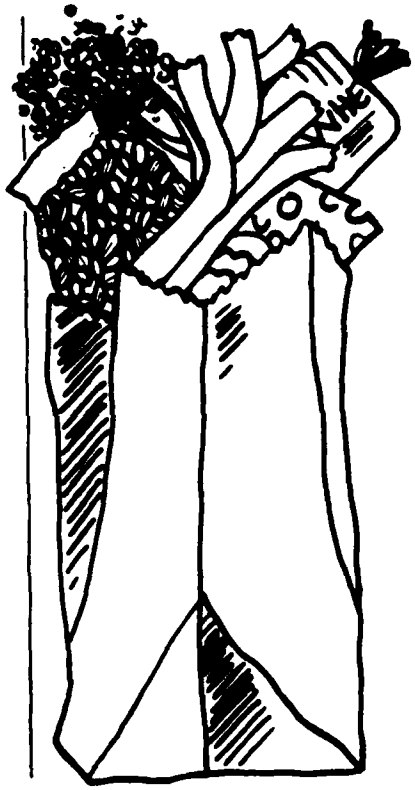
☐ Eat unsweetened cereal for breakfast or some other grain foods such as whole wheat bread, bagels, or muffins.

☐ Buy bread or grain products listing "whole wheat" first on the list of ingredients not just "wheat flour".

☐ Increase the amount of vegetables you eat. Eat two servings of vegetables each day in addition to your potatoes or other starchy food.

☐ Serve fresh or frozen fruit or some other low-sugar alternative for dessert.

☐ For snacks, replace cookies & chips with breadsticks, unbuttered popcorn, and raw vegetables.



S-L-O-W but steady — pick one or two ideas that you think will work for you. Get used to them before starting new ones.

You are moving steadily toward your goal. Small changes and moderation on your part will move you toward that excellent protection category.

Turn the page for some more information on sugars that you might want to use.

_____ _____

_____ _____

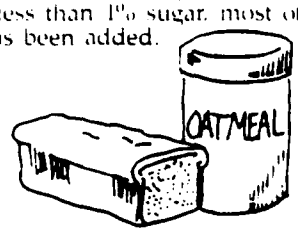
MORE THAT I CAN DO 3

SUGAR FACTS · FIBER FACTS

??? *GRANOLA — A low sugar breakfast alternative?* Not necessarily; according to the USDA, most of the nationally distributed granola contains around 25% sugar.

Other dry breakfast cereals contain varying amounts of sugar. Granola (25%) falls about in the middle of a range that begins at less than 1% sugar and tops out at 56% sugar.

Since wheat and other grains naturally contain less than 1% sugar, most of the sugar has been added.



This illustrates once again that you must read labels if you want to avoid extra calories. (See the next page for "Label Logic.")

You might be surprised at the "hidden" sugar content of some prepared foods. See Below —

	Sugar (%)
Quaker 100% Natural Cereal	23.9
Russian Wishbone Dressing	30.2
Sara Lee Chocolate Cake	35.9
Shake 'n Bake (Barbecue)	50.9
Coffee-mate	65.4
Jell-O	82.6

As you avoid prepared foods with high sugar contents and increase your intake of fruit and vegetables, you will also be increasing your fiber intake.

Fiber, often called "roughage," is also found in unrefined cereals and grains. Many claims have been made concerning fiber. Any generalized statements about the use of dietary fiber as a drug to cure specific diseases should be looked at with reservations.

The best advice is to exercise moderation. A variety of whole grain products, fruits, and vegetables will ensure a good mixture of the different types of fiber and make a positive contribution to the overall nutritional value of your diet.

This time, check your PRUCAL analysis for an estimate of your current fiber intake.

Fiber (gm) _____
Goal (15gm/1000 kcal) _____

If your intake is low, you will find suggestions on your PRUCAL print-out on how to increase it.

You have completed the 3 basic steps that, if adopted, will help you to reach your cholesterol reduction goal.

The final page of Step 3 presents some good information on food labels. If you are already a label reader, then go right on to Step 4 — *"Exploring Changes."*

FACTS FOR SELF CARE 3

L A B E L L O G I C

Ingredient Labeling — When ingredients are listed on the label, they must appear in *descending order according to weight*. This list serves as a fairly good source of information about the general quality of the product; however, you should watch out for the practice of separately listing each type of added sugar. For example, corn sweetener, dextrose, and sucrose will be listed separately so that the product will appear to be lower in sugar than it actually is. The following is a good example:

INGREDIENT LABEL from a box of Honey Graham Crackers:

INGREDIENTS: Enriched Flour, Graham Flour, Vegetable Shortening, *Sugar, Brown Sugar, High Fructose Corn Syrup, Honey,* Leavening, Salt, Artificial Flavor.

Nutrition Labels — A nutrition label may be added voluntarily, but is required when nutrients have been added or the product makes a nutritional claim. The example below, from a jar of "Natural" peanut butter, illustrates the required information that you will find. The "*" indicates additional information that may be included.

NUTRITION INFORMATION PER SERVING		PERCENTAGE OF USRDA	
Serving size 2 tbs.	32GM	Protein	10.0%
Serving per container	14	Niacin	20.0%
Calories	199	Iron	2.0%
Protein	8.6 GM		
Carbohydrate	5.5 GM	Contains less than 2% the USRDA	
Fat	16.3 GM	of Vitamin A, Vitamin C,	
Sodium	3-7 MG	Thiamine, Riboflavin and Calcium.	
% of calories from fat*	73		
Polyunsaturated*	5 GM		
Saturated*	3 GM		
Cholesterol*	0 MG		

This nutrition label includes optional listings for types of fats and cholesterol. It also illustrates the fact that most foods contain a variety of polyunsaturated, monounsaturated, and saturated fats. Peanut oil is usually classified as a monounsaturate, but as seen from this example actually contains 50% monounsaturated (8.3 gm), and 50% saturated (3 gm) and polyunsaturated fat (5 gm). Meat, pork, fish, and chicken also contain a mixture of the saturated, unsaturated and polyunsaturated fats.

Still have a question about labels or the fat content of foods? **Contact your Self-CARE coordinator.**

You have completed Step 3 and are now ready to develop your *"own"* new eating style using the information that has been presented so far.

WHAT CAN I DO? 4

HEALTHFUL FOOD CHOICES

Up to this point you have:

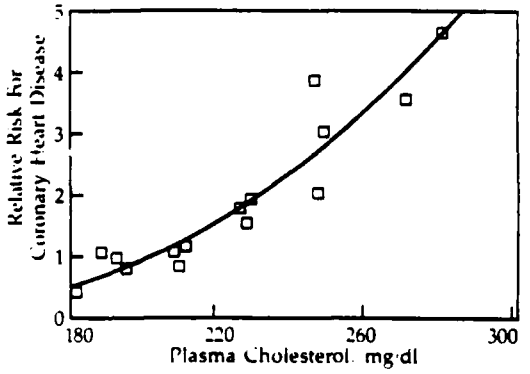
- Identified your personal "risk factors" (Step 1).
- Discovered how to decrease the fat and cholesterol in your diet (Step 2), and
- Eliminated some of those "empty calories" (Step 3).

***Step 4* will conclude this introduction to *heart healthy eating* by:**

- Reviewing your personal dietary goals.
- Providing information on nutrient dense foods.
- Discussing the importance of exercise in a healthy heart program, and
- Defining some of the terms that deal with fats and cholesterol.

Remember, the primary reason you are completing this program is because you want to decrease your cholesterol level to reduce your risk for heart disease.

Cholesterol Levels & the Relative Risk for Coronary Heart Disease



There are many approaches you may choose to take — but in general, the dietary principles for cholesterol reduction are not complicated.

They call for *restricting total fat, saturated fat,* and *cholesterol,* and *increasing complex carbohydrates.*

No single food is the culprit —
No single food is perfect.

What really matters is the *total fat,* the *% of saturated fat,* and the *ratio of polyunsaturated to saturated fat,* or P/S ratio. (See the last page of this Step for an explanation of P/S ratio.)

When the cook in your household understands what you are trying to do, and serves foods that are consistent with "Healthy Heart" eating, you are more likely to be successful in lowering your cholesterol.

Remember to have your spouse go through this program with you. If either of you have additional questions on healthy heart menus, diets, or programs, call your Self-CARE coordinator.

Go on to the next page for some good ideas on increasing *nutrient rich* foods in your diet.

HOW CAN I DO IT? 4

NUTRIENT RICH FOODS

A nutrient-rich food gives you the most nutrients (vitamins, minerals, and protein) for the number of calories consumed.

✔ Check the following nutrient analysis and decide whether snack #1 (apple) or #2 (candy bar) is the nutrient-rich snack.

NUTRIENTS	SNACK #	
	#1	#2
Calories	80	150
Protein (gm)	–	2
Total Sugars (gm)	16	15
Dietary Fiber (gm)	1	–
Fat (gm)	1*	9**
Saturated Fat (gm)	–	5
Polunsat Fat (gm)	–	2
Cholesterol (mg)	–	6
Sodium (mg)	–	30
Potassium (mg)	150	113
Vitamin A (IU)	125	75
Vitamin C (mg)	10	–
Vitamin B6 (mg)	1	–
Calcium (mg)	10	65
Iron (mg)	4	3

* 7% of calories
** 54% of calories

Maybe there is some support for the proverbial *"Apple-A-Day."*

Nutrient richness or density, in the simplest terms, means getting the most nutrition you can with your calories. (See page 1 of your PRUCAL analysis for your daily calorie Goal.)

Your calorie goal _____

The bar graph in your PRUCAL analysis illustrates the nutrient density concept.

Are you spending these calories wisely?

Using information from Steps 1-3, read the following suggestions, and implement those that you think can help you increase your intake of nutrient rich food.

☐ **Cut down on the portion size and the frequency that you serve meat, poultry, and fish.** Choose dishes that use these meats for flavoring, rather than the bulk of the meal. *(Hint: This means choose dishes like spaghetti with meat sauce, seafood chowder, or stir fry dinners.)*

☐ **Eat morning meals without breakfast meats or eggs.** Cereals, breads, bagels, and muffins are filling and delicious. *(Hint: Remember to use low-fat milk, whole grain products, and margarine instead of butter.)*

☐ **Substitute or modify your food choices to improve your heart health.** In the commissary or at the fast food stand, develop the habit of being *fat conscious. (Hint: A dish of Wendy's chili, green salad and a low fat milk (430 calories), instead of a double cheeseburger, french fries and a frosty (1520 calories) saves you over 1000 calories, most of them fat calories.)*

Diet is not the only weapon at your disposal in your battle against high cholesterol.

MORE THAT I CAN DO 4

D I E T & E X E R C I S E

The National Cholesterol Education Program emphasizes that the first step of any cholesterol lowering program is "diet." Not "diet" in the sense of a drastic change in lifestyle, but rather a new approach to eating which stresses *substituting* tasty new items for old favorites, choosing *low fat* substitutes when available, and consuming *smaller portions* of those foods you do not want to give up.

One result of this new approach to eating would hopefully be maintenance of a reasonable weight.

Weight control is important because "overweight" people frequently have *higher cholesterol, higher blood pressure,* and are more likely to develop *diabetes* than adults at their proper weight.

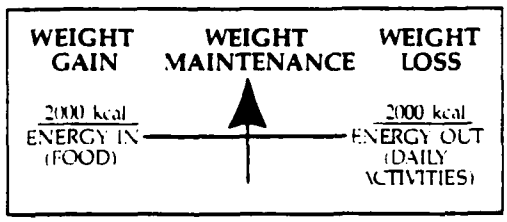
Remember that in "Step 1" these three factors were "Risk Factors" for developing coronary artery disease.

EXERCISE CAN HELP — Air Force personnel are encouraged to participate in regular physical activity.

Regardless of the activity you choose, regular exercise assists in weight control and plays a role in regulating your cholesterol level.

- exercise often helps you control your appetite
- exercise burns up calories
- exercise may increase the levels of your "good cholesterol" (HDL)
- exercise & weight reduction can help reduce your "bad cholesterol" (LDL)

Your weight, as you well know, is controlled by balancing the calories you take in (food) with the calories you use (physical activity).



All calories count the same. If you take in 500 more calories per day than you use, in a week you will gain 1 pound. The reverse is also true.

Increased physical activity can then complement your dietary changes, helping you to reduce your risk for developing heart disease.

The last page of Step 4 defines some commonly encountered terms & concepts, and summarizes what you have learned from Self-CARE.

FACTS FOR SELF CARE 4

T E R M S A N D F A C T S

Atherosclerosis — A disease of the arteries in which deposits of fat and cholesterol on the inside of the artery walls (plaques) gradually block the flow of blood.

Calorie — The amount of energy a food contains. Kilocalorie. Calorie and kcal stand for the same amount of energy. Carbohydrates and proteins contain approximately 4 Calories per gram and fat contains about 9 Calories per gram.

Cholesterol — A fatty substance required by the body to make several hormones and as a component of all your body's cells. Found only in foods of animal origin such as egg yolk, meat, and liver.

Fiber — General name for several types of indigestible carbohydrates found in cereals, grains, fruits, vegetables, legumes, nuts, & seeds.

Gram — A metric unit of weight. 30 grams is about equal to an ounce.

HDL Cholesterol — The "good" cholesterol found in the blood that seems to help lower serum cholesterol.

LDL Cholesterol — The "bad" cholesterol found in the blood that appears to deposit cholesterol in the artery walls.

P S Ratio — Represents the ratio of the polyunsaturated and saturated fats in your diet. The recommended ratio is 1.0. A diet containing 15 grams polyunsaturated fat & 30 grams saturated fat would have a P S ratio of .5.

Polyunsaturated, Saturated, & Monounsaturated Fats — See Step 2 — "Saturation Facts."

Simplesse — An all-natural substitute for fat made by finely grinding the protein from fresh egg whites or milk. Cuts calorie content by up to 80%. Not to be used in cooking. Not commercially available.

Sucrose Polyester (Olestra) — A fat substitute made by attaching part of the fat molecule (the fatty acid) to a sucrose molecule. It retains the taste and feel of fat, but can not be digested. Can be used in cooking. Not commercially available.

Triglycerides (TG) — One of the blood lipids measured during your cholesterol test. Not thought to be an independent risk factor for heart disease. Used by the lab to calculate your LDL level. (LDL = Total Cholesterol-HDL-(TG/5)).

TO SUM UP

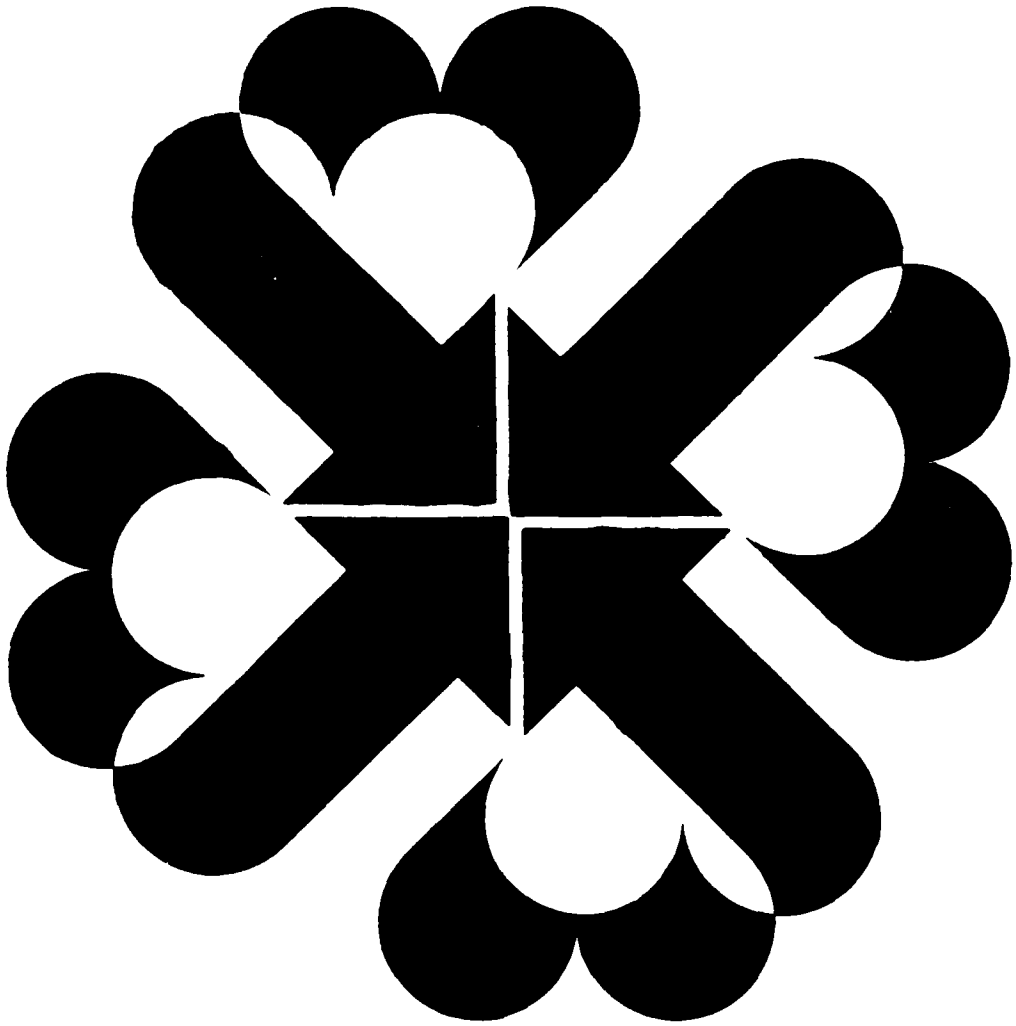
The 4 Steps in "Self-Care" were meant to give you some explanations and recommendations from current research on 5 major areas:

***Heart Risk Factors (decrease) * Cholesterol & Fats (decrease)**
***Calories From Simple Sugars (decrease)**
***Calories from Starches and Grains (increase)**
***Nutrients from vegetables, meats, grains, fruits, milks (increase)**

With this information, you should find it easier to decide what part of your eating style, if any, you need to change. If you need more information to help you make changes, or if you just have a question of general interest ....

CONTACT_____. Thanks for your participation.

SELF CARE



FOR A HEALTHY HEART

SELF CARE FOR A HEALTHY HEART

Self-CARE for a Healthy Heart is designed for people who want to reduce their risk for heart disease by decreasing their cholesterol levels, but are not sure about the easiest ways to accomplish it. *Self-CARE* will lead you through a series of 4 Steps. These steps will suggest changes that you can make in your eating habits over a period of weeks or months, depending upon your motivation.

The contributions to *Self-CARE for a Healthy Heart* by the following programs are gratefully acknowledged:
FAMILY FOODSTYLE NUTRITION EDUCATION PROGRAM (University of Guelph)
HEALTHY HEART PROGRAM (Colorado State University)
PAWTUCKET HEART HEALTH PROGRAM (The Memorial Hospital, Pawtucket, Rhode Island)

By the end of the program you and hopefully your family will:

- Understand which food factors are associated with heart disease.
- Decrease the fats and cholesterol in your diet.
- Decrease the "empty calories" in your diet.
- Make changes that become a permanent part of your eating style.

It is up to you to decide how long you spend on each step of the program. Start by reviewing all parts of the program and, if at all possible, have your spouse work through the program with you. Everyone should proceed at their own pace. It may take weeks or even months to change certain lifelong eating habits.

Each "Step" has 4 sections entitled: *What Can I Do?; How Can I Do It?; More That I Can Do; and Facts... For Self-CARE.*

- *What Can I Do?* — read this section first and answer any questions the best you can.
- *How Can I Do It?* — this section gets you started and should be kept where you can refer to it easily while working on this step.
- *More That I Can Do.* — this section complements the previous one and presents additional dietary alternatives you may want to pursue.
- *Facts...For Self-CARE* — this section will give you more detailed information on selected topics introduced in *Self-CARE*, or on topics that will help you adopt some of the changes suggested in the program.

Concentrate on one step at a time! Remember:

- Do not rush yourself! Gradual changes are generally the longest lasting.
- When you are confident that you have "mastered" one step, read and post the next step. Use the magnetic heart you will find in your kit to post each step or to post your plastic coated *Self-CARE* reminder sheet. The refrigerator is a convenient place for posting *Self-CARE* materials.

PRUCAL DIET ANALYSIS

The purpose of the "Self-CARE" program is 1) to provide high quality information on coronary heart disease, basic nutrition and dietary risk factors, and 2) to provide support and assistance for those who wish to make changes in their eating patterns.

To support these purposes and your efforts to control your cholesterol, you will receive a PRUCAL diet analysis during each of the months you are in the "Self-CARE" program. This diet analysis is designed to help you plan and evaluate your diet according to the "Prudent" diet guidelines presented in the "Self-CARE" program. Each month you will be asked to complete a PRUCAL diet analysis worksheet detailing one day's menu. Once submitted, your data will be computer analyzed and the results quickly returned to you.

Why go to the trouble you may ask? It is one thing to read about a healthy heart diet, but it is quite another to put it into practice. The PRUCAL Diet Analysis Program allows you to evaluate your current diet against prudent guidelines, then suggests foods to consume or avoid to more closely meet your goals (see the attached sample PRUCAL analysis). A monthly analysis will allow you to monitor the success you are having in adopting a healthy heart diet.

SEND IN THAT FIRST ANALYSIS ASAP- YOU WILL REFER TO IT THROUGHOUT THE "Self-CARE" PROGRAM.

NOTE-- ALL THE INSTRUCTIONS ARE ON THE FORM- BUT PLEASE REMEMBER THE ANALYSIS REQUIRES CANDIDNESS (INCLUDE ALL SNACKS, SNEAKS, BEVERAGES, AND CONDIMENTS SUCH AS BUTTER, MARGARINE OR SUGAR). THE ANALYSIS IS AN AID FOR YOU, NOT A REPORT CARD. THE ONLY TIME YOUR RECORD WILL EVEN BE SEEN BY ANYONE ELSE IS WHEN THE PROGRAM LEADER DOES YOUR COMPUTER ANALYSIS.

1- READ THIS COVER SHEET

2- LOOK OVER THE SAMPLE PRUCAL DIET ANALYSIS

3- COMPLETE THE DIET ANALYSIS WORKSHEET (YELLOW- TWO SIDES)

4- RETURN WORKSHEET IN THE PREADDRESSED ENVELOPE-- THANKS

SELF-CARE FOR A HEALTHY HEART **PRUCAL DIET ANALYSIS WORKSHEET**

PLANNING DIETS TO MEET PRUDENT GUIDELINES [ANALYSIS #]

NAME_____

MALE ____ FEMALE ____

AGE _____ (NEAREST YEAR)

HEIGHT ____FEET ____INCHES

BONE STRUCTURE ____SMALL ____MEDIUM ____LARGE

ACTIVITY LEVEL (Most people are sedentary, light or moderate)(You may select one level higher if you have a vigorous daily exercise program of more than 30 minutes)

_____ SEDENTARY: Most of the time is spent sitting.

_____ LIGHT: Much time is spent standing, while engaging in light activity or office work.

_____ MODERATE: Activities which require walking at moderate speed occupy most of the time.

** Current Weight _____** Please return critique form!!
*** What prescription drugs (including vitamins) are you taking?_____
_____.

This program will calculate your energy needs based on your age, activity level, and the average recommended weight for an individual your height and frame size.

Your nutrient intake will be analyzed for one day. The calorie goal is distributed so that no more than 30% of total calories come from fat, with 10% each from saturated and polyunsaturated fat. (see "Saturation Facts", Step-2, in your "Self-CARE" program for an explanation of saturation) The cholesterol goal is set at 300 mg/day, dietary fiber at 15 grams/1000 calories (for example if you required 3000 calories/day, your fiber goal would be 45 grams), and vitamins and minerals at 100% of your individual Recommended Daily Allowance set by the National Academy of Sciences.

PLEASE TURN THIS PAGE OVER AND RECORD YOUR FOOD INTAKE FOR ONE DAY.

When completed, return this form, via base distribution/mail/or in person, in the preaddressed envelope provided in your Self-CARE folder.

FOOD INTAKE LIST

Record the foods you consume for ONE DAY in the columns headed "FOOD ITEM & PORTION". When recording food intake, be sure to include all snacks, condiments such as butter, margarine and sugar, and all beverages except water. The computer has many of the food items from the national fast food chains, so you can put down "Big Mac" or "Wendy's french fries" etc. Please note: The column you use and the order you list food items is not important, but listing all that you consume is!!

Be specific when listing foods and portion sizes- FOR EXAMPLE:

1 CUP 2% MILK; 1 CUP COFFEE; 1TSP POWDER CREAM SUBST; 1TSP SUGAR; 12 OZ LIGHT BEER; WENDY'S FROSTY, 9OZ.

2 SLICES WHOLE WHEAT TOAST; SOFT MARGARINE, 2 TSP; JAM, 1TBL; CORN FLAKES, 1 OZ; 2 LARGE EGGS, POACHED.

HAM SANDWICH, WHITE BREAD,1 TSP BUTTER & MAYO, LETTUCE; 1 CORN DOG, MUSTARD (BROWN) 1TSP; 1 ORANGE.

CHICKEN POT PIE 8OZ; GROUND BEEF, LEAN, BROILED, 3OZ ; OPOSSUM, ROASTED, 3OZ.; 15 RITZ CRACKERS.

Food Item & Portion BREAKFAST	Food Item & Portion LUNCH	Food Item & Portion DINNER

"SELF-CARE"

Your Total Cholesterol: mg/dl

Your Goal: mg/dl by

You Can Reach Your Goal!!

1) Reduce your intake of fat
2) Eat smaller portions
3) Choose low-fat substitutes
4) Exercise

These Ideas May Help!!

Eat more of these	and less of these
•**Vegetables**	•Fried Foods
•**Lean meats, fish, poultry**	•Fatty meats lunch meats bacon hot dogs sausage
•**Vegetable protein sources** **peas** **beans** **grains**	•Fatty desserts ice cream pastries pies
•**Breads & Cereals**	
•**Fruit for dessert & snacks**	

We Need Your Help!

We would like to know what you think of the "Self-CARE" program! Please take a minute to answer the questions below. Return the completed form with one of your PRUCAL diet analysis forms.

I completed the "Self-CARE" program on _____.
Day--Month

How many of the 4 "steps" did you complete? _____.

Did you find the kit understandable? _____.
(yes, somewhat, no)

How much did you learn?

A lot _____.

A little _____.

Nothing new _____.

Good review _____.

Did you find the kit motivating?

Very motivating _____.

Somewhat motivating _____.

Not at all motivating _____.

What eating habit changes have you made (are you making)?

_____

_____

_____

Suggestions/Comments _____

_____

_____

_____

Your name (optional) _____

THANKS for your help!

APPENDIX D

MISCELLANEOUS FORMS AND LETTERS

UNITED STATES AIR FORCE ACADEMY (1-30 December)

ENROLLMENT SHEET FOR SELF-CARE PROJECT (complete a subject information sheet and a consent form for each individual)--

DATE/NAME	ADDRESS	ZIP	PHONE	PROGRAM?
5 OCT/ SMITH, BILL	10980 THOMAS RD. COLORADO SPRINGS,CO	80908	H-495-2307 W-472-1122	SC or UC

AGE	WEIGHT	HEIGHT (inches)	CHOL	LDL	HDL	TG	SMOKE?
30	190	70	280	160	45	375	Y or N

//

DATE/NAME	ADDRESS	ZIP	PHONE	PROGRAM?
				SC or UC

AGE	WEIGHT	HEIGHT (inches)	CHOL	LDL	HDL	TG	SMOKE?
							Y or N

//

PETERSON AIR FORCE BASE (1-30 DECEMBER)

ENROLLMENT SHEET FOR SELF-CARE PROJECT (complete a subject information sheet and a consent form for each individual)--

EXAMPLE: SEE BELOW

5 OCT/ SMITH, BILL	10980 THOMAS RD. COLORADO SPRINGS,CO	80908	H-495-2307 W-472-1122	SC or UC

DATE/NAME-XXXXXXXXXXXXXXX-ADDRESS-XXXXXXXXXXX-ZIP-XXXXXXX-PHONE-XXXX-PROGRAM?

30	190	70	280	160	45	375	Y or N

AGE-XXXX-WEIGHT-XX-HEIGHT (inches)-X-CHOL-XXX-LDL-XXX-HDL-XXX-TG-XX-SMOKE?

//

YOUR INFORMATION: ENTER BELOW PLEASE

SC or UC

DATE/NAME-XXXXXXXXXXXXXXX-ADDRESS-XXXXXXXXXXX-ZIP-XXXXXXX-PHONE-XXXX-PROGRAM?

Y or N

AGE-XXXX-WEIGHT-XX-HEIGHT (inches)-X-CHOL-XXX-LDL-XXX-HDL-XXX-TG-XX-SMOKE?

//

INSTRUCTIONS TO PRINCIPAL INVESTIGATOR: A signed Subject Information Sheet must be given to the subject for his records.

COLORADO STATE UNIVERSITY
(Subject Information Sheet-A)

Project Title: Self-CARE for a Healthy Heart

Principal Investigator: Dr. G. Richard Jansen
Co-Investigator: Major Jeffrey M. Johnston

Contact person and phone number for questions/problems:
Maj Jeffrey Johnston- Air force Academy-- 472-5071
Peterson AFB-- 554-4572

Purpose/Objective of Research: The purpose of Self-CARE for a Healthy Heart is to:

1) to provide high quality information on coronary heart disease, basic nutrition and dietary risk factors,

2) to provide support and assistance for those who wish to make changes in their eating patterns, and

3) to determine the effectiveness of using a self-study program.

The primary objective of this study is to have individuals that follow the Self-CARE for a Healthy Heart program acheive an average reduction in their serum cholesterol that equals or exceeds the reduction acheived through "usual care".

Procedures to be Used: Self-CARE for a Healthy Heart will present information that will allow you to decide what part of your eating style, if any, you may need to change to decrease your elevated cholesterol level. The information will be provided in a take home format and it will supplement and reinforce the verbal counseling you received from your Health Care Provider. Your participation in this program will require you to read the program, complete the monthly computer diet analyses required to monitor your progress and complete a 3 month progress check.

Risks: The only risks associated with SELF-CARE for a Healthy Heart are those you would normally encounter with routine medical care.

Assurance of Confidentiality: Confidentiality procedures as covered by DD Form 2005, Privacy Act Statement- Health Care Records will be followed. The data collected for this project will not be reported on an individual basis, only aggregate data will be used for evaluation purposes.

I agree that the subject has the right to terminate participation in this research project at any time.

_____
Investigator Date

INSTRUCTIONS TO PRINCIPAL INVESTIGATOR: A signed Subject Information Sheet must be given to the subject for his records.

COLORADO STATE UNIVERSITY
[Subject Information Sheet-B]

Project Title: Self-CARE for a Healthy Heart

Principal Investigator: Dr. G. Richard Jansen
Co-Investigator: Major Jeffrey M. Johnston

Contact person and phone number for questions/problems:
Maj Jeffrey Johnston- Air Force Academy-- 472-5071
Peterson AFB-- 554-7345

Purpose/Objective of Research: The purpose of Self-CARE for a Healthy Heart is to:

1) to provide high quality information on coronary heart disease, basic nutrition and dietary risk factors, and

2) to provide support and assistance for those who wish to make changes in their eating patterns.

Procedures to be Used: Your participation in this program will require you to attend one scheduled CARE briefing, and then at your three month cholesterol check, you will complete one 24 hour diet questionaire for computer analysis (the results of which will be returned to you).

Risks: The only risks associated with SELF-CARE for a Healthy Heart are those you would normally encounter with routine medical care.

Assurance of Confidentiality: Confidentiality procedures as covered by DD Form 2005, Privacy Act Statement- Health Care Records-- will be followed. The data collected for this project will not be reported on an individual basis, only aggregate data will be used for evaluation purposes.

I agree that the subject has the right to terminate participation in this research project at any time.

_____
Investigator Date

INSTRUCTIONS TO PRINCIPAL INVESTIGATOR:

A signed consent form must be given to the subject for his records and a copy retained for your files.

COLORADO STATE UNIVERSITY

Consent to Serve as a Subject in Research

I, _____(print name), consent to serve as a subject in the research investigation entitled:

Self-CARE for a Healthy Heart

The nature and general purpose of the experimental procedure and the risks have been made known to me by Lt. Col. J.M. Johnston.

He or she is authorized to proceed on the understanding that I may terminate my service as a subject in this research at any time I so desire.

I understand the known risks of participation in "Self-CARE for a Healthy Heart" are only those normally associated with routine health care.

I understand that it is not possible to identify all potential risks in an experimental procedure, but I believe that reasonable safeguards have been taken to minimize both the known and the potential, but unknown, risks.

Signed_____
Subject Date

If a subject is injured in the course of the research investigation and he contends that Colorado State University or an employee thereof is at fault for the injury, the subject must file a claim within 180 days of the date of the injury with the State Attorney General and the State Board of Agriculture. The University's legal and financial responsibility, if any, for such injuries is controlled by state law. Your claim will be referred to the Risk Management Liaison Office for review, and you should direct your inquiries to that office (303-491-5257). The University cannot otherwise compensate subjects for their injuries, and subjects must depend on their own health and disability insurance for compensation for injuries sustained in the course of the research investigations which are not the fault of CSU or its employees.

Anthropometric and Laboratory Data Collection

The following data categories have been included in each file for analysis:

@-	SEX	AGE	WEIGHT		HT	CHOLE		LDL		HDL		TGS		SMK	@-	BMI		%FAT	%SF	%PUF	P/SR	TC/HDL		DGS	OAT	ALC	V	A
1	1	35	170	165	70	220	210	160	150	55	50	160	150	Y	1	24.5	22.5	30.0	10.0	10.0	1.50	5.00	4.50	1	2	1	2	3

EXPLANATION- (MISSING VALUES DENOTED BY ".")

@- PATIENT NUMBER (NUMBERS REPEAT FOR SELF-CARE AND USUAL CARE AT EACH BASE AND TREATMENT)

SEX- 1=MALE 2=FEMALE

AGE- NEAREST WHOLE YEAR

WEIGHT- NEAREST WHOLE POUND AT START AND END OF STUDY (MISSING VALUE FOR ENDING WEIGHT DENOTES DROPOUT)

HT- HEIGHT IN INCHES

CHOLE- CHOLESTEROL IN MG/DL AT THE START AND AT THE END OF THE STUDY- EG. 300 250

LDL- LOW DENSITY LIPOPROTEIN IN MG/DL AT THE START AND END OF THE STUDY- EG. 160 140

HDL- HIGH DENSITY LIPOPROTEIN IN MG/DL AT THE START AND END OF THE STUDY- EG. 55 45

TGS- TRIGLYCERIDES IN MG/DL AT THE START AND END OF THE STUDY- EG. 150 130

SMK- 1=NO 2=YES

BMI@- BODY MASS INDEX AT THE START AND END- EG. 24.5 23.5 THESE NUMBERS MUST BE CALCULATED.

%FAT- PERCENT FAT IN DIET AT THE END OF STUDY- EG. 30.0

%SF- PERCENT SATURATED FAT IN DIET- EG. 10.0

%PUF- PERCENT POLYUNSATURATED FAT IN DIET- EG. 10.0

P/SR@- POLYUNSATURATED FAT TO SATURATED FAT RATIO- EG. 1.00 THIS NUMBER MUST BE CALCULATED.

TC/HDL@- TOTAL CHOLESTEROL TO HIGH DENSITY LIPOPROTEIN RATIO- EG. 5.00 4.50 THESE NUMBERS MUST BE CALCULATED.

DGS- PRESCRIPTION DRUG USE- 1=NONE, 2=BIRTH CONTROL PILLS, 3=BLOOD PRESSURE, 4= CHOLESTEROL LOWERING, 5=HEART MEDICATION, 6=[illegible], 7=OTHER

OAT- OAT PRODUCT CONSUMPTION 1=NO 2=YES

ALC- ALCOHOL CONSUMPTION 1=NO 2=YES

V- VITAMIN CONSUMPTION 1=NO 2=YES

A- ACTIVITY LEVEL 1=SEDENTARY, 2=LIGHT, 3=MODERATE, 4=ACTIVE

Usual-Care Three Month Reminder Letter- USAFA

Hello,

Three months have passed since you agreed to help us evaluate our Nutrition Education Program. Would you please complete the enclosed dietary analysis worksheet (yellow paper)- On one side you will find your name- please complete the items that follow your name. Then turn the page over and complete the food intake list-- just follow the instructions.

Next (1) turn in the yellow diet worksheet at Major Sutay's office, (2) get your lab slip from her, and (3) then get your cholesterol rechecked at the laboratory.

You can accomplish this from 0730 - 1800 hrs.; Monday through Friday.

Please try to complete this by _____ 1988.

Thanks for your help--

P.S. Any questions or problems please call Major Sutay at 472-5074.
If you forget to turn in your worksheet, please mail it to:

Lt Col J.M. Johnston
10980 Thomas Rd.
Colorado Springs, CO 80908

Usual-Care Three Month Reminder Letter- Peterson AFB

Hello,

Three months have passed since you agreed to help us evaluate our Nutrition Education Program. Would you please complete the enclosed dietary analysis worksheet (yellow paper)- On one side you will find your name- please complete the items that follow your name. Then turn the page over and complete the food intake list-- just follow the instructions.

Next (1) turn in the yellow diet worksheet at Ms. Simpson's office, ROOM 214C (2) get your lab slip from her (your slip number is _____, and (3) then get your cholesterol rechecked at the clinic's lab.
You can accomplish this from 0730 - 0930 hrs.; Monday through Friday.

Please try to complete this _____.

Thanks for your help--

P.S. Any questions or problems please call Ms. Simpson at 554-7345.
If you forget to turn in your worksheet, please mail it to:

Lt Col J.M. Johnston
10980 Thomas Rd.
Colorado Springs, CO 80908