

**COMPARATIVE PROFILES OF CURRENTLY ACTIVE AND FORMERLY ACTIVE
PARTICIPANTS IN A CARDIAC RISK REDUCTION PROGRAM**

by

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(ABSTRACT)

Selected anthropometric (body weight and BMI), dietary (kilocalories, carbohydrate, protein, total fat, saturated fat, linoleic acid, oleic acid, dietary cholesterol, and P/S ratio), blood pressure, and blood lipid parameters (total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C), triglyceride (TG), and TC/HDL-C and LDL-C/HDL-C ratios) were assessed in 67 males from the Cardiac Therapy and Intervention Program at Virginia Tech. Several studies have found strong correlations between these variables and the incidence of coronary heart disease. The group (cardiac or intervention), status (active or inactive), time (1982-83 baseline period, 1983-84 short-term follow-up period, and 1986 long-term follow-up period), and the group and status combination (cardiac active (CA), cardiac inactive (CI), intervention active (IA), and intervention inactive (II)) were chosen for statistical analysis to determine if there were significant differences due to these effects.

The P/S ratio (< 1.0), the dietary cholesterol intake (> 250 mg), the level of blood cholesterol (> 200 mg/dl), and the TC/HDL-C and the LDL-C/HDL-C ratios ($>$ average risk) were identified as areas which needed improvement in all groups. Compared to the dietary guidelines proposed by American Heart Association (AHA), all combinations of comparisons across three time periods exhibited higher percentages of kilocalories provided by total fat, saturated fat, and protein, and lower percentages of kilocalories provided by linoleic acid and carbohydrate. The HDL-C levels were below the fiftieth percentiles relative to the Lipid Research Clinics Population Study data. Blood pressures were under good control.

The four subgroups exhibited significantly different mean body weights and TC/HDL-C and LDL-C/HDL-C ratios. The II group had the highest values for all these variables, the lowest mean body weight was seen in the CI group, and the IA group had the lowest mean values for the latter two ratios. There was a trend toward the lowest mean dietary intake and blood lipid levels occurring at the short-term follow-up period; however, only the mean intakes of total calories and carbohydrate and the blood LDL-C levels were significantly different among the three time periods. The lowest mean values for these three variables occurred at the short-term follow-up period while the highest mean values occurred at the long-term follow-up period. The group effect was seen in the mean intakes of total fat, saturated fat, linoleic acid, oleic acid, and the percentage of kilocalories as fat and the mean levels of systolic and diastolic blood pressures. The intervention group exhibited the higher mean values for these variables. The major difference relative to status was in the mean values of the TC/HDL-C ratio. The inactive participants had the higher mean value. The results of a discriminant analysis procedure which was used to determine which combination of risk factors was most influential in distinguishing the cardiac group from the intervention group indicated that abnormal electrocardiogram test results and age were the most influential factors of those studied.

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INTRODUCTION

Coronary heart disease (CHD) continues to be a primary cause of death and disability in the United States, although overall age-adjusted mortality rates have declined in all age groups since 1968 (1-3). Atherosclerosis, the most common form of arteriosclerosis (hardening of the arteries), is the major nutritionally related cause of CHD. Atherosclerosis is a disease of the tunica intima of the large and medium-sized arteries and is characterized by localized fatty deposits and thickening of the arterial wall. The fat deposits distort the arteries and make them rigid. This condition, in turn, interferes with the flow of blood to the heart or brain.

Results of animal, epidemiological, and clinical studies give supportive evidence that the concentrations of blood lipids are closely related to the development of atherosclerosis (3). Researchers have tried to induce atherosclerotic plaques in several mammalian species by feeding high fat and high cholesterol diets at different levels and composition of fat. The results of these studies (4-16) suggest that blood lipids can be altered by changes in fat composition in the diet and that high dietary saturated fat is about twice as potent in elevating blood cholesterol levels as polyunsaturated fatty acids (PUFAs) are in reducing it (17-19). Since PUFAs have this hypocholesterolemic effect, it is important that a high cholesterol diet also be high in PUFAs. It has long been accepted that the total cholesterol concentration in the blood is a good predictor of the level of risk for atherosclerosis (20-23). The higher the level, the higher the risk. The predictive potential diminishes with advancing age (20,24-25), and total cholesterol (TC) shows little relationship with atherosclerosis after age 65 (20,24).

Recently more attention has been paid to the amount of cholesterol attached to particular protein carriers because researchers have found that CHD is more accurately predicted by measuring these subfractions of total cholesterol concentration (9,20-21,26). Specifically, risk of atherosclerosis is inversely related to the high density lipoprotein cholesterol (HDL-C) concentration (9,20,26-27). In turn, high levels of HDL-C have been correlated positively with exercise (3,24,26,28-43), higher intake of polyunsaturated fat, and lower total and saturated fat intake.

Furthermore, low levels of HDL-C were inversely related to obesity (3), cigarette smoking (3,20), and hypertriglyceridemia (3,36,38). The reasons for these findings remain uncertain but have been attributed to the major function of HDL-C. Specifically, HDL-C is thought to remove cholesterol from arteries and transport it back to the liver where it can be cleared from the body. In addition, HDL-C may compete with low density lipoprotein cholesterol (LDL-C) for binding in tissues, thus interfering with the LDL-C uptake by the cells (44). On the contrary, LDL-C is thought to transport cholesterol to the artery walls (20). Thus, the tendency towards atherosclerosis appears to increase with increasing amounts of cholesterol attached to LDL-C carriers. Low density lipoprotein cholesterol receptors play a critical role in regulating blood cholesterol levels in mammals and in humans. Lack of normal function or a decreased number of receptors may explain abnormally high levels of blood LDL-C level. Very low density lipoprotein cholesterol (VLDL-C) and the triglyceride (TG) associated with it may play an indirect role relative to incidence of CHD (20,27). The changes in the composition and distribution of the blood lipoproteins are attributed mainly to the type of dietary fat and the ratio of polyunsaturated fat to saturated fat (P/S ratio) (3-4,8-11,13).

Numerous studies have been conducted to identify potential risk factors for CHD. Among these, the Framingham study, begun in 1951, has drawn tremendous attention to the relationship between environmental factors and CHD (45). Thereafter a number of studies (46-56) were undertaken to confirm the most important risk factors proposed by this study (45). The most important environmental factors related to CHD are the following: dietary cholesterol and fatty acid intake, blood cholesterol concentration, high blood pressure, cigarette smoking, physical activity, and body weight (3,56). Genetic potential is thought to be an unmodifiable factor (57).

The causes of CHD are multifactorial (3,20-21). However, if any one risk factor is reduced or eliminated, then theoretically the incidence or mortality of CHD should be reduced. Cardiac therapy and intervention programs have been initiated to provide a systematic intervention strategy aimed at changing habits of the participants which may predispose them to CHD. The Cardiac Therapy and Intervention Center at Virginia Tech is an example of one such program. Although

the positive effect of exercise and the benefit of adopting dietary habits recommended by the American Heart Association (AHA) (53) have been stressed, the effectiveness of this cardiac risk reduction program has not been clarified fully. This study was undertaken to assess the impact of life-style changes as intervention strategies on the participants in this program. Specifically, those participants who continue to be active in this program were compared with those who have discontinued participation relative to status of the following risk factors:

- Dietary components (i.e., kilocalories, protein, carbohydrate, total fat, saturated fat, oleic acid (monounsaturated fat), linoleic acid (polyunsaturated fat), cholesterol, and P/S ratio)
- Blood lipid levels (i.e., total cholesterol, LDL-C, HDL-C, triglyceride, and the ratios of TC/HDL-C and LDL-C/HDL-C)
- Changes in body weight, BMI, and blood pressure

Profiles of participants were compared over three time periods: a 1982-83 baseline period, a short-term follow-up period 6 to 9 months later, and a long-term follow-up period in 1986. The rationale for these comparisons across time was to examine the degree to which the life-style changes influenced these coronary risk factors and to ascertain the most frequent combination of risk factors. The results of this study will be beneficial to the program staff by providing information relative to environmental factors influenced by life-style changes in these high risk participants.

LITERATURE REVIEW

Pathogenesis of Atherosclerosis

The major cardiovascular diseases (e.g. ischemic heart disease or heart attack, hemorrhagic cerebrovascular disease or stroke, hypertensive heart disease, atherosclerosis, and hypertension) account for almost one-half of all deaths in the United States (1), although the age-adjusted overall mortality associated with them has declined each year for the last two decades (2,3). Moreover, the additional costs for work-loss, disability, and health expenditures make these diseases the number one leading health expense in this country (57). In particular, atherosclerosis and hypertension, either separately or in combination, recently have contributed most to death from CHD, and they also underlie other causes of death such as diabetes (57-59) and renal disease (60).

Arteriosclerosis is a group of arterial diseases characterized by degeneration, thickening, and hardening (sclerosis) of one or more layers of the arterial wall (61). The walls of arteries contain three coats (or tunics): intima, media, and adventitia. The intima is the inner layer which consists of three parts: the endothelium, the connective tissue layer, and the elastic tissue layer. The media is the middle layer which contains smooth muscle. The adventitia is the outermost layer which contains connective tissue.

Atherosclerosis is a type of arteriosclerosis in which the fatty materials, cholesterol, and dead cells are deposited in the large or medium-sized arteries (61). The detailed mechanisms causing the damage of the arterial wall remain unclear; however, the following description represents the process by which the damage may occur. The earliest stage in the development of these lesions is thought to be caused by injury to the endothelial and subendothelial intima cell. The injury may be mechanical, biochemical, or immunologic in nature and may give rise to physical abrasion and rupture of the vessel wall (23). Once the cell injury has occurred, the smooth muscle cells will proliferate and migrate from the media into the lesion site. The process is followed by the precipitation of

cholesterol esters, phospholipids, triglycerides, and the formation of atheromatous plaques. In advanced stages of lesion formation, fibroblasts infiltrate the degenerative areas, adding protein, collagen, elastic fibers, and sulfated polysaccharides to the atheromatous plaques (62). The process leads to progressive sclerosis, eventually resulting in fibrosis and necrosis. The necrotic tissue is hard, thick, and rigid after calcification. It protrudes into the lumen, narrows the blood vessel, and reduces the blood flow. It may even form a thrombus which occludes the blood vessel. It has been proposed that fatty streaks, fibrous plaques, and advanced plaques (or complicated lesions) are the three histological features of an atheroma (63). They are thought to be permanent lesions. The development of fatty streaks, resulting from an abnormal accumulation of cholesteryl esters in the form of intracellular liquid or liquid crystalline droplets in the intima of blood vessel (63), is regarded as the first signal of atherosclerosis, even though it usually is clinically asymptomatic. Clinically, fibrous plaques are more important and permanent lesions than are fatty streaks. The plaques contain high levels of extracellular lipids, mainly cholesteryl esters with a composition similar to that for cholesteryl ester of LDL-C (63).

Cholesterol is a sterol and is insoluble in water. Thus, cholesterol usually is combined with protein to form lipoprotein, a major blood cholesterol carrier. About 70 percent of the blood cholesterol in the lipoprotein is esterified. The remaining non-esterified cholesterol is on the surface of the lipoprotein (64-65). The liver is the most active organ involved in cholesterol synthesis. The mechanism is under negative feedback regulation. That is, a higher intake of cholesterol may suppress the synthesis of cholesterol by the liver.

Most of the blood cholesterol is carried in LDL-C (21,66-68); therefore, LDL-C plays an important role in the development of atherosclerotic lesions and in the regulation of cholesterol metabolism (3). LDL-C must first be taken up by specific receptors for its degradation. This happens mostly in the fibroblast cells (64-65,69). Once taken up by these receptors, LDL-C will react further with lysosomes which contain cholesteryl esterase. The protein parts of LDL-C are degraded and the cholesterol esters are hydrolyzed to release free cholesterol (64,69). Normally, this free cholesterol may function as a component of cell membranes and as a regulator for reducing

cholesterol synthesis by suppressing the activity of 3-hydroxy-3-methyl glutaryl coenzyme A reductase (HMG CoA reductase). Also, the free cholesterol may activate cholesterol acyl transferase to form more cholesteryl esters for storage. Free cholesterol also suppresses the synthesis of the LDL-C receptor and thus prevents accumulation of too much cholesterol by the cell (64,69). However, the arterial cells may use the free cholesterol and phospholipids to synthesize the cholesteryl esters seen in the first period of fatty streaks, and thus break down the whole equilibrium. Acyl-CoA : cholesteryl transferase plays an important role in the process.

The results of both animal (70) and human (71) studies have confirmed that LDL-C degradation to produce free cholesterol or to transfer free cholesterol from lipoproteins to arterial cells plays a role in the accumulation of cholesterol in the early lesions (3). Using cell-culture experiments, Lundberg (71) found that cholesteryl esters which accumulated may be produced from just a small increase in the free blood cholesterol level. Hence, a high concentration of cholesterol-bearing lipoprotein may favor cholesteryl ester formation in the arterial wall (63). This may be the main reason for the initiation of atherosclerosis (25,64-65,72).

Low density lipoprotein cholesterol is taken up by the intima of fibroblast smooth muscle which synthesize and secrete collagen and elastin (73). Katz et al. (74) further verified this view in their studies, finding that about 20 percent of plaque lipids are elastin-associated. Cholesteryl ester of LDL lipids will bind to the abnormal elastic tissue of plaque (75). Moreover, LDL-C and LDL apoprotein B have been isolated from human plaque (75,63). Even though the large difference in lipid composition between LDL-C and human plaques may not give direct evidence to link these two, the presence of cell and cellular debris in atherosclerotic lesions may indicate that the cell membrane also plays a role in the development of plaques (75).

Since an exchange between blood and lesion cholesterol has been found, it has been suggested that HDL-C may play a digestive role at the myointimal cell level (3). Once the smooth muscle and connective tissue cells proliferate, a lipid-containing fibrous capsule may be formed. This capsule may interfere with oxygen uptake and the normal metabolic process of LDL-C carriers.

Hence, LDL-C has been regarded as the main cause of atherosclerosis (3). The most distinct stage in the development of atherosclerosis is the accumulation of lipid droplets and cholesterol monohydrate crystals in the arteries (63). This may exacerbate the consequences of smooth muscle cell proliferation and calcification of fibrous capsule. It also may effect elevated activity of lysosomes with further increased hydrolysis of cholesteryl esters to form reduced receptor sensitivity. Supersaturation of the lipid phases in the arterial lumen may result (76). At this point, the advanced plaques, containing a considerable excess of free cholesterol and many lipid-filled cells with necrotic cores, are thought to be sclerogenic which may hinder the regression of the plaque. The complicated lesions will interfere with the blood circulation and then cause systemic effects, such as ischemic heart disease (coronary vessel), myocardial infarction (coronary occlusion), cerebrovascular accident (brain vessels) (23), and renal disease (60). Two pathways which elucidate the development of atheromatus lesions, either from early fatty streaks through advanced fatty streaks or from fibro-fatty plaques to advanced plaques, have been proposed by Lundberg (63).

Role of Age and Sex in the Pathogenesis of CHD

Natural aging may explain part of the process of arteriosclerosis since aging effects changes in the lipid content, lipid type, and physical states of lipids within the intima (25). Several environmental factors as well as local factors in the vessel wall may affect the turnover of the lipid molecules in the artery (63). Fatty streaks in the aorta will be increased proportionally with advancing age (48,77); however, in any race or sex, it is not easy to find fatty streaks in the coronary arteries until about 15 years of age (654). Katz (25), who studied the normal human aortic intima from birth to old age, pointed out that there were no lipid deposits in the intima during early life and that the major lipids found subsequently were phospholipid (71.4 percent) and cholesterol (23.8 percent). As the age increased, the lipid content of the intima increased, doubling by the end of the first decade and increasing thirteenfold after 70 years of age, with cholesteryl esters predominating. Gordon et al. (24) also confirmed that advancing age is a natural risk relative to CHD and that

blood total cholesterol is no longer a predictor of risk in men beyond age 65. The mechanisms related to this lipid accumulation in the lesion-free intima with age may be due to hyperplasia and hypertrophy of the intimal cells. Secondly, it may be due to an increase in the uptake of cholesterol esters from LDL-C lipids by the intimal connective tissue, since Apo B, the most important apolipoprotein which exists in LDL-C, has been extracted from normal human intima and identified (25).

The specific roles of LDL-C and HDL-C in lipid metabolism and in the pathogenesis of CHD still need further elucidation (78). Studies of cord blood suggest that at birth, neither the level of TC nor the level of HDL-C is predictive of CHD prevalence in adult relatives (79-80). Moll et al. (78) concluded that, although the relationship between lipid and lipoprotein levels in childhood and later development of disease is unknown, they can infer from their study that children between the ages of 6 and 16 years with elevated LDL-C or low HDL-C are probably at increased risk for CHD in adulthood. That the prevention of cardiovascular disease should begin early in the life is not disputable (48).

Role of Genetics in the Pathogenesis of CHD

Several researches (67,81) have shown that genetics may be the most important factor in determining the level of blood cholesterol. Dietary cholesterol plays a significant role in most hyper-responsive patients. However, enough direct evidence to verify the connection between cholesterol intake and the level of cholesterol in the blood of normal, healthy people is yet to be found. Superko et al. (49) and Goldbourt et al. (82) discussed the possibility of a threshold effect among individuals. Especially, there appears to be a better correlation between diet and blood cholesterol among hyperlipidemic victims. Some individuals demonstrate a hypo-responsive effect to a high cholesterol diet; some familial hyperlipidemic patients show extremely hyper-responsive

results with low or little dietary cholesterol; and some respond sharply to dietary cholesterol level above the threshold level. This latter group should be targeted for dietary intervention (67).

Regression of Atherosclerosis

Regression of atherosclerosis has been achieved in animals by diet, drug therapy (83), exercise, and ileal bypass surgery (3). Latent, young lesions were more amenable to regression (3). The degree of regression in animals was roughly proportional to the blood cholesterol levels (83). The lower the cholesterol level, the higher the level of regression (3,84). Those who failed to regress had significantly higher cholesterol and TG levels (3). Regression varied depending on the artery involved, the site of the lesion, and the natural aging process, and on the duration of intervention since regression-inducing measures take variable times to be effective (3).

Animal studies (nonhuman primates and swine) have shown that advanced atherosclerotic plaques will regress when the blood total cholesterol is reduced (3). Cholestyramine, a bile-acid sequestering agent, has been used successfully in animals to lower blood cholesterol levels with regression of diet-induced lesions (83). An exchange of cholesterol between blood and atheromatous lesions, with net efflux of cholesterol from regressing lesions, has been observed (3). A decrease in accumulated atherosclerotic material (regression) would result in a net efflux greater than net influx (3). Increased levels of blood HDL-C lead to an increased efflux of cholesterol from the arterial wall pool to the blood pool, thereby contributing to regression (3). Generally, patients with clinically manifest CHD or atherosclerosis have a low linoleic acid content in blood cholesteryl esters and in adipose tissue (85). The regression of atherosclerosis usually is related to the elevated intake of linoleic acids (3,85).

Environmental Factors Related to Coronary Heart Disease

The relationships among development of atheromatus plaques in coronary artery intima and the prevalence of coronary artery disease and mortality from heart disease have been studied extensively. Most of the risk factors have been identified from long term epidemiological studies, clinical manifestations, and animal experiments. Aging, male gender, and genetics are usually regarded as unmodifiable risk factors (48,3). However, high blood pressure, high blood total cholesterol, low HDL-C and high LDL-C levels, cigarette smoking, physical inactivity, glucose intolerance and obesity are all regarded as modifiable factors (3,23,46,48-49). Hypertension, hypercholesterolemia, and cigarette smoking are the most potent factors leading to atherosclerosis (3,47). The risk increases significantly when other risk factors also are present (20-21). Many of the risk factors are interrelated and interdependent (3), giving rise to further difficulties in identifying their relative importance.

Lipid Hypothesis

The lipid hypothesis is based upon results from the Framingham study and other related studies (21,23,45-56). According to this hypothesis, people with higher blood cholesterol levels have more and earlier coronary heart disease than those with lower cholesterol levels (86). About 80 percent of endogenous cholesterol is synthesized by the liver, and only about 20 percent of cholesterol is obtained from daily dietary sources (exogenous). However, the blood cholesterol level is dependent on the sum of daily cholesterol intake, absorption (only 1/3 of the intake is absorbed), endogenous hepatic synthesis, and bile excretion (3, 64). High intakes of dietary cholesterol and/or saturated fat and low intakes of polyunsaturated fat are the major dietary components affecting the level of blood cholesterol and elevating the LDL-C to HDL-C ratio. Blood cholesterol was found to be one of the major risk factors for coronary heart disease in the Framingham study (3,21). A linear relationship, the higher the cholesterol values, the higher the CHD risk (20-23), was found

to exist between dietary cholesterol and blood cholesterol in several studies (3,66-67,87-88), although each individual showed a different response to dietary cholesterol. This relationship was not confined just to those with hypercholesterolemia (88).

In summary, populations with high intakes of fat (especially high saturated fat (4-8) and high cholesterol), and low intakes of polyunsaturated fats have higher blood cholesterol concentrations (4,22,89) and also have a higher risk of CHD (23) than those with a high P/S ratio in their diets (3-4,8-11,13). The major recommendation of some researchers is to reduce total fat intake and to increase the proportion of dietary fat from polyunsaturated fat. At present, evidence for a direct connection between dietary cholesterol and blood cholesterol levels is incomplete. The links between an atherogenic diet and CHD have been established by animal experiments, but since different species may react differently, the results from animal studies cannot be extrapolated to human beings. Moreover, different people may respond differently to dietary cholesterol (26).

Dietary Fatty Acids and Blood Lipid Levels

Saturated fatty acids can be hypercholesterolemic (17,23). Hopkins and Williams (46) proposed that high fat diets initiated atherogenesis by gradually destroying the integrity of the arterial endothelium and that a high degree of saturation in the fat increased the likelihood of thrombus formation. Variation of chain length or degree of saturation of different fatty acids may alter their hypercholesterolemic effects (14). Researchers have shown that three hypercholesterolemic fatty acids are lauric (C 12:0), myristic (C 14:0), and palmitic (C 16:0) acids. Among these, palmitic acid showed the least effect on blood cholesterol levels (14,23,90).

Saturated fatty acids were shown to be twice as potent in elevating blood cholesterol levels as polyunsaturates were in reducing them (17). The reason for the cholesterol-lowering effect of polyunsaturated fatty acids (PUFAs) is still not clear; however, several hypotheses have been advanced to explain the effect. First, animals fed corn oil produced VLDL with a larger diameter than

did animals fed coconut oil (13). Thus, it can be inferred that saturated fatty acids cause the formation of smaller VLDL particles that contain relatively more cholesterol (4,91). These may filter faster into the arterial wall than larger ones (4) and thus may be metabolized by extrahepatic tissues at a slower rate than are larger particles.

Secondly, polyunsaturated fatty acids are less efficiently incorporated into triglycerides of the lipoprotein particles (VLDL-C) than are saturated fatty acids (91), resulting in a decreased rate of synthesis and number of blood lipoprotein particles (16,92). When a diet rich in PUFAs was exchanged for one high in saturated fatty acids (fat providing 45 percent of energy in both diets) and given to 4 subjects, the synthetic rates of VLDL apo B and LDL apo B were decreased (8). This decreased synthesis of VLDL-C may then cause the concentration of LDL-C (12,16,91-91), of total blood cholesterol, and of triglycerides to decrease (16,22,92).

Third, a change in the composition of phospholipids in the surface coat of LDL-C may influence the catabolic rate of this lipoprotein. Polyunsaturated fatty acids will decrease the level of saturation in LDL triglycerides, cholesteryl esters, and phospholipids (8,12,93). Increase in the fractional catabolic rate of LDL-C (8,12,91,93) may alter the distribution of cholesterol from the blood into the tissues.

Fourth, modification of the composition of LDL-C may cause changes in the metabolism of cholesterol (4,12,94-96) and/or lipoproteins (11-12). Polyunsaturated fatty acids altered the configuration of the lipids, especially cholesteryl esters and phospholipid within LDL-C so that the capacity for cholesterol transport is reduced. This proposal was based on the observation of an increase in the cholesterol/protein ratio in LDL-C during administration of a highly saturated fat diet (97), and a faster decrease in LDL-C and phospholipids and a slower catabolism in LDL protein during diets rich in polyunsaturated fatty acids (4,95), compared to diets high in saturated fat. As a consequence, LDL-C had an altered composition containing less cholesterol (4,95,97) and phospholipids (4,95) and relatively more protein and triglycerides (TGs) (4,95,97) in PUFA diets.

This change in LDL-C structure and composition may alter the uptake and degradation of the lipoprotein.

Fifth, it has been proposed that cholesteryl esters in polyunsaturated fats are metabolized faster by the liver and other tissue than those in saturated fat (4,23,98). In turn, this enhanced the stimulation of cholesterol excretion into the intestine and the stimulation of the oxidation of cholesterol to bile acids (91,97). As a consequence, the cholesterol content of LDL-C might be reduced by decreased availability of secretion in lipoproteins (97). Polyunsaturated fats increased the linoleic acid (C 18:2) content of these lipids (4,95,97) and reduced that of the palmitate (C16:0) and oleate (C18:1) (95,97). In addition, cholesteryl linoleate is more easily mobilized and transported than is cholesteryl oleate (98).

During hydrogenation, unnatural trans isomers of fatty acids are formed (90). When the fats are hydrogenated, their original properties are changed, i.e., the effect of polyunsaturates are diminished, although the hydrogenated forms of PUFAs have not been found to increase blood cholesterol levels to the degree that saturated fats have. Because hydrogenated products tend to react more like saturated fat (17), they are seldom recommended in fat-controlled diets (90).

In summary, the P/S ratio is thought to be important in regulating blood cholesterol levels. The total amount of fat in the diet appears to be less important than the P/S ratio (23). However, a diet with increased PUFAs (above 15 percent) has been related to decreased blood HDL-C levels (11,92,105). Therefore, the polyunsaturated fat should replace only some of the total fat (99,104).

Monounsaturated fatty acids are synthesized by the body and are less susceptible to chemical oxidation than polyunsaturates (99). Monounsaturates previously were thought to have no effect on blood cholesterol levels (18-19,98-99). However, currently some researchers (11-12,93,96,99-100) proposed that monounsaturated fatty acids may lower the cholesterol and blood triglyceride levels (11-12,93,96,99) in LDL-C without affecting the HDL-C (99-100) in normotriglyceridemic patients but not in hypertriglycemic patients (99). Oleic acid, the principal

monounsaturate (C16:1), is as effective as linoleic acid (PUFA) in lowering LDL-C levels in normotriglyceridemic patients, and it seemingly reduces HDL-C levels less than does linoleic acid (99).

Since the predominant monounsaturated fatty acid in egg yolk is oleic acid, one study was set up that give different level of monounsaturated fatty acids from daily egg yolk intake (66). The energy percentages derived from saturated and polyunsaturated fatty acids remained constant. The results indicated that eating egg yolk significantly altered the composition of the LDL particles, specifically, the core become enriched in esterified cholesterol at the expense of TG and the ratio of core components to surface components increased by seven percent (12,66). This effect is similar to that of PUFAs.

Blood Cholesterol and Lipoproteins Levels

Suppression of total body cholesterol synthesis or increased fecal steroid excretion is usually the metabolic response to increasing dietary cholesterol (67,101-102). Increasing the cholesterol intake beyond 500 mg/day (87,103) or 800 mg/day (3) produced little additional rise in the blood cholesterol level. These results suggest a threshold effect (3,82). The majority of the rise in blood cholesterol was due to a rise in LDL-C (3). Intermediate density lipoprotein cholesterol (IDL-C), HDL2 (one sort of HDL-C), and apo B concentration also were increased (87). These findings are consistent with those of Packard et al. (68), who pointed out that dietary cholesterol increases the blood level in man by increasing the number of LDL-C (67-68), IDL-C (87) and HDL2 (87) particles rather than by altering the composition of LDL-C (68). Recently, Reckless (104) reported that a daily reduction of 100 mg of cholesterol may cause an average decrease of 0.2 mmol/liter in blood cholesterol. However, foods high in cholesterol usually are high in fat, which may be a confounding factor relative to increases in blood cholesterol levels.

Blood lipids play a principal role in the atherosclerotic process (3,20-23). HDL-C, LDL-C and VLDL-C are related to coronary incidence (20-21,78). Blood total and LDL cholesterol can be reduced by limiting the intake of total cholesterol (22,38,89). Dietary factors also may influence blood HDL-C concentration (9,22). HDL-C was associated negatively with saturated fat intake, and LDL-C was associated positively with the percentage of energy from fat, although most of the increase in blood cholesterol resides in the LDL fraction (20-21,66-68). Several authors pointed out that the cholesterol in the HDL-C fraction also responded consistently with increasing blood cholesterol level after cholesterol feeding (9,66,102,106) and decreased in response to reduced fat intake (4,9,16,22,107). Therefore, HDL-C must be assessed in relation to LDL-C, i.e., the ratio of LDL-C/HDL-C may give a better notion as a risk indicator (9,108), especially if LDL-C is also low. Knuiman and West (108) stressed that HDL-C can serve as a risk predictor on high-fat diets because it reflects the ability to handle dietary fats, while HDL-C will lose its predictive effect on a low-fat diet since the challenge of fat loads is lacking. The protective effect of HDL-C is no less than the atherogenic effect of LDL-C (20). It is postulated that there is about a 50 percent change in coronary risk for every 10 mg change in HDL-C (20). Both the LDL-C and HDL-C show powerful but independent predictive effect (20,22).

An inverse correlation between blood HDL-C concentration and LDL-C receptor activity was reported (109). These results suggest that hyper-responders should have higher blood HDL-C levels than hypo-responders. The decrease in HDL-C observed during a low-fat, high P/S diet may be transient (4) and could be due to a fall in the HDL2 cholesterol (4). It is less clear whether the excess cholesterol that appears in the blood after cholesterol feeding is atherogenic (66).

Several researchers reported that in healthy normolipidemic men, a high PUFA diet (P/S = 2.82, P/S = 4.0, respectively) with a low cholesterol intake (112 mg/day, or 198 mg/day, respectively) decreased the blood levels of total cholesterol. On the other hand, a low PUFA diet (P/S = 0.23, P/S = 0.4, respectively) with a high cholesterol intake (667 mg/day or 1021 mg/day, respectively) failed to produce lower cholesterol levels. The decrease in total blood cholesterol rel-

ative to the first diet was due primarily to a decrease in LDL-C, VLDL-C, and HDL-C (11,22,107,110).

Blood Triglyceride Level and CHD

Total blood triglyceride is made up of the triglyceride components of LDL-C, HDL-C and VLDL-C (3). Triglycerides are transported predominantly in VLDL-C (3). People with high TG values have an increased risk for CHD (21), since it is usually accompanied by an increase in blood total cholesterol and LDL-C (53). Cabin et al. (116) pointed out that severe narrowing of the left main coronary artery was found in subjects with high triglyceride levels, but little relationship with blood total cholesterol values was observed. It is uncommon to find an association between high blood triglyceride (TG) levels and the prevalence of CHD (53,111). Epidemiologic studies (3,21,112-113) do not support a causal relationship between increased blood triglycerides and CHD. The blood TGs themselves seemingly do not deposit in atherosclerotic lesions (115). Rather, any such correlation is probably due to increased blood cholesterol or other factors such as obesity and diabetes mellitus (3,21,114). Moreover, blood triglyceride is usually inversely correlated with HDL-C (28-29,35-36,53,115) which suggests that the effect of the removal of cholesterol from peripheral tissue may be defective (53). Clinical hypertriglyceridemia for adults is usually defined at the value above the 95th percentile. The value from the Lipid Research Clinics Population Studies (LRCPS) (50) for the 95th percentile of middle-aged American men was between 250 and 300 mg/dl (See appendix A).

In hypertriglyceridemias, smaller VLDLs and larger chylomicrons may alter the catabolism of chylomicron and VLDL-C and indirectly increase the atherosclerotic effect (53). Therefore, VLDL-C and the TG associated with it may play an indirect role with an independent contribution to CHD risk (20,112,114-115). Elevation of blood free fatty acids also will lead to increased VLDL-C secretion by the liver, involving extra triacylglycerol and cholesterol output into the circulation. Factors leading to higher or fluctuating levels of free fatty acids include emotional stress,

nicotine from cigarette smoking, coffee drinking, and intake of few, but large meals rather than more, small continuous feeding (61).

Blood Pressure and CHD

Hypertension is the most frequent cardiovascular risk factor in obese patients (47,117). Evidence from epidemiologic studies and animal experiments confirm that blood pressure is associated with age (47,117-118). Recently, Smith-Barbaro et al. (119) suggested that BP is controlled by several factors including the rate of prostaglandin synthesis, blood renin levels, and the amounts of fat and salt ingested.

Different amounts of fat (120-121) and types of fat (5,7,122-123) may mediate blood pressure at different levels. On a low fat diet, both systolic and diastolic blood pressure decreased (121). The decrease was greater among hypertensives than among normotensive persons (121). The blood pressure increased when the diet return to normal (121). When fatty acid chain length is similar, the degree of unsaturation is a key factor in controlling blood pressure. Diets high in saturated fat alone, despite the dietary salt level, demonstrated a hypertensive effect (7,119-120,124), whereas recent research suggests that blood pressure can be lowered, during high or basal salt feeding, by supplementing the diet with polyunsaturated fatty acids (119-120,124) such as linoleic acids.

Rats fed diets rich in essential fatty acids showed lower blood pressure than rats fed diets without enough essential fatty acids (6,122). When the latter group of rats were supplied with sufficient essential fatty acids, the blood pressure then returned to the level similar to the former group (122). Researchers using rats who were fed low salt and three levels of linoleic acid indicated that reduced systolic blood pressure was seen with higher (9.4 percent) and moderate (4.2 percent) levels of linoleic acid intake but not with lower levels (0.41 percent) of linoleic acid intake. The mechanism involved may be that the low linoleic acid diet may impair the synthesis of renal

prostaglandins involved in blood pressure regulation (5,7,122-123) since linoleic acid is a precursor of prostaglandins (123).

An inverse relationship between P/S ratio and blood pressure was seen (120). Increasing the P/S ratio by increasing the polyunsaturated fat had a reducing effect on blood pressure in rats (120). A significant drop in blood pressure also was seen in human studies (123,125). For example, when the P/S ratio was increased from 0.22 to 0.95, a significant drop in blood pressure was reported (125). The use of PUFA rich oils as supplements also may help reduce the level of medication used in the treatment of hypertension (123). Therefore, PUFA rich vegetable oils may be useful for patients with mild-to-moderate hypertension (123).

In surveying the available literature, it appeared that hypertensives usually were more obese (117). There might be a genetic reason for this higher body weight seen in the hypertensive patients (117). Siervogel et al. (126) reported positive partial correlations between blood pressure and percent body fat, total body fat mass, or fat cell number. Both systolic and diastolic blood pressure were associated with body mass index (117): the highest blood pressure was found in the highest body mass index categories (117). Weight reduction improves cardiac function as well as arterial pressure levels in patients with hypertension (39,127) by reducing levels of blood renin activity (128), blood aldosterone levels (128), catecholamine levels, and blood insulin levels (117). Weight reduction also may reverse the increased blood volume, cardiac output, stroke volume, and ventricular and diastolic pressure (127). In addition, weight loss is associated with reduced resting circulating levels of blood norepinephrine (127) and thus might also decrease renal tubular reabsorption of sodium (117). Slower heart rate, reduced venous return and cardiopulmonary volume, and increased cardiac output enhance a redistribution of intravascular volume away from the cardiopulmonary area (127). Patients who did not lose weight showed no changes in any of these hemodynamic measurements (127).

Cigarette Smoking and CHD

Cigarette smoking has been cited as one of the major risk factors in ischemic heart disease (129), but it has only a weak association with stroke (130). Although the mechanisms involved are still unclear, they are thought to increase fibrinolysis (131,132), platelet stickiness (133), and endothelial permeability (134). A significant thickening of the walls of the myocardial and renal arterioles of smokers are thought to be due to an increase in collagen and smooth muscle in the adventitia of small to medium-sized intramyocardial coronary arteries (60). These changes in the arteriolar are thought to give strong supportive evidence that cigarette smoking is closely related to arteriolar thickening. In addition, platelet-derived growth factor, released in the vessel wall, may promote proliferation of connective tissues (60) and smooth muscle (135), which may, in turn, cause thickening of the vessels (60). Surveys of human smokers have shown no change in platelet counts, but have shown differences in platelet aggregation and platelet turnover (136). Furthermore, cigarette smokers dying of conditions other than CHD have been found at autopsy to have intimal thickening of the coronary arteries and an excess of atheroma. This finding suggests that a duration effect of smoking in relation to myocardial infarction (MI) might be anticipated (137).

Rogers et al. (138) have suggested that cigarette smoking and nicotine may change normal behavioral, sensory, and metabolic processes with respect to energy regulation and body weight. Smokers have an increased metabolic rate compared with nonsmokers (139-140). Other physiological possibilities also exist. Smokers may have less efficient absorption from the gut as gastric emptying is increased in smokers during smoking (141) and the speed of material through the alimentary tract is increased in smokers (142). Decreased caloric storage is supported in animal studies in which nicotine infusion resulted in weight loss without decreased food consumption (143). Moreover, smoking may favor catabolism rather than anabolism as a consequence of increased catecholamine level (144), circulatory cortisol, and growth hormone (145) in the blood with concomitant decreased insulin secretion (146). Therefore, it is not uncommon to find that nonsmokers were higher in weight and fatness than smokers even though smokers consumed more

calories per day on the average (33,129). A dose-response relationship between the number of cigarettes smoked before cessation of smoking and weight gain has been found (47,129,147).

Cigarette smoking has been reported to increase the risk of MI. The relative risks rose steadily as the amount of cigarette smoking increased. The relation was most evident at the youngest ages and became progressively weaker with increasing age (148). Doll and Peto (148) have observed from their 20 year study that heavy cigarette smokers had 15 times more ischemic heart disease than did non-smokers under 45 years of age; while, at age 65 years and over, cigarette smoking was more closely related to myocardial degeneration than ischemic heart disease (148). The mortality rates between quitters and persistent smokers relative to CHD were compared by Friedman et al. (149). After adjustment for major base-line differences in risk characteristics, the persistent smokers showed 2.2 times more CHD than did quitters. Smoking cessation was associated with a decreased incidence of major manifestations of CHD, particularly mortality (150).

Hjermann et al. (151) carried out a 5-year trial on 1232 normotensive (systolic blood pressures below 150 mmHg) and hypercholesterolemic (290-380 mg/dl) men to determine if the lowering of high levels of blood lipids by dietary changes and the cessation of smoking among healthy middle-aged men could reduce the incidence of CHD. The results showed that changes in blood cholesterol appeared to correlate more closely with incidence of CHD than did the change in cigarette smoking. The changes in blood lipid levels accounted for about 60 percent of the reduction in the incidence of CHD; whereas, the changes in smoking status accounted for only 25 percent (151).

Whether smoking will affect total blood cholesterol levels is still a controversial issue. The mechanisms by which cigarette smoking depressed HDL-C (152-153) is uncertain but might be due to nicotine (154) and might be operative through other risk factors or predictors of atherosclerosis (155). No significant differences in total blood cholesterol, VLDL-C + LDL-C, HDL-C, or triglyceride concentrations have been found in baboons (156). However, recent surveys indicated that human smokers have had lower HDL-C concentration (152-153,157) and higher LDL-C levels (157) and that a minimal number of cigarettes must be consumed per day in order to observe an

effect (152-153). Nonsmokers and exsmokers had a significant increase in HDL-C (39), body weight, and skinfold thickness, but no change in LDL-C, TG, or blood pressure. Moreover, the usual inverse relationship between body weight and HDL-C does not exist with cessation of cigarette smoking (154).

Physical Activity and CHD

The results of epidemiologic studies have shown that increased physical activity is associated with a low incidence of CHD whereas a sedentary life-style is regarded as a risk factor (41). Physiologically, physical exercise can improve cardiovascular fitness by improving muscle strength, especially stroke volume and organ maximal oxygen uptake (35,41). Physical exercise also may change body weight (38,40-41,158), decrease systolic blood pressure (35,38,159), pulse rate (159), body fat (41,161-162), skinfold thickness (41), and increase lean body mass (41,160). Physical exercise also may play a role in improving glucose tolerance (161-163), and further changing blood lipids composition and distribution (35,38,161). Most of the researchers suggested that exercise training produces lower concentrations of TG (28-31,35-36,43,159,164-166) and higher concentrations of HDL-C (28-42) both in normal and hypercholesterolemic or hypertriglyceridemic subjects, whereas only the prolonged effort of a marathon run reduced total cholesterol (36,164) and LDL-C (31-32,165). Stamford et al. (33), involved in animal studies, also confirmed these results.

The physiological mechanisms responsible for these findings may be related to the elevated activity of lipoprotein lipase (30-31,35-37,166) and the suppressed activity of hepatic triglyceride lipase (36) as well as the elevated level of lipolysis. Lipoprotein lipase (LPL), an enzyme located mainly at the capillary endothelium of muscle and adipose tissue (30-31,36,43,166), is a key enzyme in the hydrolysis of triglyceride-rich lipoproteins, mainly intestinal chylomicron and hepatic-secreted VLDL-C. When the muscle undergoes extended periods of exercise, the main energy source is from its own TG and from blood free fatty acids. If under prolonged acute exercise, the muscle will depleted its own TG and the energy expenditure derived from fat is increased (167), i.e.,

the lower blood free fatty acids level will trigger lipolysis in the adipose tissue (30,37,167,167-169) to balance the blood free fatty acids level (165,167). Under these circumstance, both lipoprotein lipase and lipolysis are elevated and the former show a higher pronounced effect than the latter. The absolute decrease in TG is positively related to the duration of exercise (32) and the greatest decrease is usually found in pronounced pre-exercise hypertriglyceridemic and hypercholesterolemic subjects (24,30,159,170). Several researchers have found strong supportive evidence that exercise may elevate the activity of lipoprotein lipase in muscle and adipose tissue (30-31). In addition, since runners usually showed higher basal levels of both adipose and muscle LPL (30) and less prolonged exercise did not cause any acute changes in TC, LDL-C or HDL-C (32,165,171-172), Some researchers think that the lipoprotein differences between sedentary and active individuals are due to metabolic adaptations to exercise training rather than to an acute exercise effect (30-32,41). Herbert et al. (173) proposed that the diminished HDL-C clearance by the liver may be another reason for higher HDL-C levels shown in runners.

Some researchers did not agree that the changes in the components and the distributions of the lipoproteins were due to physical exercise itself, but thought that some confounding factors might contribute to these results (42,158,178). First of all, body weight may contribute to the different rates of production of VLDL-C by the liver. The results from animal studies confirmed that obese rats may have higher VLDL-C production (164). Weight loss itself has been thought to be responsible for the improved lipoprotein lipid profiles in patients with hypercholesterolemia (174). Davis et al. (174) pointed out that weight loss via medical manipulation (through intake of colestipol) increased blood HDL-C and decreased TC, LDL-C and total TG concentrations and was responsible for the lipoprotein changes associated with endurance training (40-42,158,171,175-177). Epidemiologic evidence suggests that obesity is associated with an increased risk of CHD (178), which might be mediated in part by the association between obesity and blood lipid levels. Results of several studies indicate that HDL-C level are inversely correlated with body weight and LDL-C levels are positively correlated with body weight. Furthermore, other researchers (158,178) have pointed out that weight loss can produce an increase in HDL-C levels and

a decrease in LDL-C levels, i.e., weight loss can reduce an obese person's risk of CHD by decreasing the LDL-C/HDL-C ratio (40,41) or the TC/HDL-C ratio (41). However, physical training combined with weight loss may yield a synergistic effect on lowering total blood cholesterol (42,158), LDL-C (42,158), triglyceride levels (179), and on elevating HDL-C levels (158,179) and the LDL-C/HDL-C ratio (39,158). Another possible confounding factor could be the higher oxygen uptake by physically active individuals. As a result of this higher uptake, the TC/HDL-C ratio was decreased proportionally and an inverse relationship to triglycerides, TC, and LDL-C+VLDL-C was noted (163,180). Higher caloric intake (181), especially a higher percentage of fat consumption, was found in a training group (159). This may be another factor which stimulated the hepatic system to produce more HDL apoproteins---mainly the HDL2 subclass (173)--- or to elevate the LPL activity to digest more HDL-C precursors (173). Blood catecholamine (epinephrine and norepinephrine) (37,168), somatotropin, cyclic adenosine monophosphate (cAMP), glycerol, and lactate concentrations will increase after vigorous exercise, and the degree of increase will be closely related to the intensity of exercise (168). These hormones and metabolites will affect the entire metabolic cycle of lipoproteins. Finally, a strong positive correlation has been found between aerobic capacity and HDL-C (38) and the TC/HDL-C ratio (36,38) on males with varying degrees of habitual physical activity.

On the other hand, several studies have failed to show that exercise training increased HDL-C levels (38,165,171-172) or lowered levels of TG and LDL-C (165). Some researchers stressed that there may be a critical exercise threshold needed to achieve an elevated HDL-C (34,41); however, the intensity required has not been defined. In general, prospective studies employing less intensive exercise regimens have shown little or no change in HDL-C concentration (31,171-172).

Physical training has been accompanied by a decrease of insulin secretion and a lowering of blood insulin levels without any change in glucose tolerance (36,182). Training seems to increase the insulin sensitivity of tissues (30). It is not known whether this effect involves an increase in the number of insulin receptors in muscle and adipose tissue (30). In man, the functional LPL in both adipose tissue and skeletal muscle is sensitive to insulin (30).

In summary, physical training may be associated with an adaptive increase of LPL activity both in skeletal muscle and in adipose tissue and thus result in a positive correlation with blood HDL-C levels and a negative relationship with blood TG levels. However, the level of intensity and duration of physical training which may bring about a distinctive benefit for the heart and circulatory system is still a subject of controversy and the mechanism by which physical exercise influences coronary risk factors is an area which needs more study. For example, it is disputed whether mild-to-moderate physical exercise may be a useful adjunct to regress or prevent of CHD (35). Furthermore, Stamford et al. (33) hypothesized that smoking may diminish the effects of exercise to elevate HDL-C since high activity smokers who were physically active did not differ from nonsmokers who were relatively inactive with respect to HDL-C (33).

Estimation of Risk of CHD

The major risk factors stated above usually predict cardiovascular events to different degrees. Systolic and diastolic blood pressures were found to be higher in hypertriglyceridemics than in hypotriglyceridemics and were lower in hyperalpha-lipoproteinemics than hypoalpha-lipoproteinemics (115). Cerebrovascular insults, especially stroke, are highly related to hypertension and inversely related to total blood cholesterol levels (20). Hypertension, cigarette smoking, and high levels of cholesterol in the bloodstream are the greatest risk factors for heart attacks, MI, and peripheral arteriosclerosis (183). However, total blood cholesterol is no longer as valuable a predictor of risk for CHD in men beyond age 65 (20,24).

Despite strong evidence from animal studies plus some from human studies, there is no clearly defined cut-off point regarding the level at which blood cholesterol is considered to be elevated, since it is age-dependent in the U.S. (26,184) and the results from different authoritative groups are inconsistent. The average blood cholesterol level among American adult males is about 210 mg/dl

(26). However, most researchers (184) using the data from The Lipid Research Clinics Population Studies (LRCPS) agreed that persons in the top ten percent of the average blood cholesterol level for their age/sex group were hypercholesterolemic (appendix A), and that those around the seventy-fifth percentile were thought to be at moderate-risk. For many years, total blood cholesterol levels were used to identify the potential high risk targets for CHD (21). The optimal values of cholesterol for prevention of coronary disease and atherosclerosis are probably between 140 to 180 mg/dl (20). Total cholesterol values greater than 200 mg/dl generally appear to increase the risk for cardiovascular events proportionately (3,20). The 95th percentile of blood cholesterol among the American population is still above this critical value; therefore, the whole American population should be targeted for intervention (3).

LDL-C has a strong inverse relationship to the incidence of stroke, but is strongly associated with CHD (20,27). LDL-C is thought to be atherogenic, since approximately 1/2 to 2/3 of the cholesterol in the blood is carried in this form (21). Gordon et al. (24) have thought that LDL-C is a powerful predictor of CHD in persons older than 50 years of age.

On the contrary, HDL-C carries about one quarter of the blood cholesterol, which is transported away from the artery wall to the liver by mechanisms involving lecithin cholesterol acyl transferase (LCAT) (64). In addition, HDL-C may compete with LDL-C for receptors binding in tissues, thus interfering with the cellular uptake of LDL-C. Both mechanisms make HDL-C a protective factor for CHD (20-21,26-27) and the most powerful predictive indicator for CHD currently available, especially in ages older than 50 (21). The normal range of HDL-C is between 30 and 65 mg/dl, and is age dependent. Although increased HDL-C is associated with lower risk of CHD, the significance of the protective effect of HDL-C is much less important than low blood cholesterol levels (3). A TC/HDL-C ratio of 5.0 is associated with an average risk of CHD. An optimal ratio around 3.5, corresponds to half the standard risk (20).

Very low-density lipoprotein cholesterol (VLDL-C) does not seem to exert much of an influence of any sort on any of the other atherosclerotic events (20,27), and adding TG to the HDL-C

and LDL-C lipid profile appears to do nothing to improve the efficiency of the estimation of risk (20). Hypertriglyceridemia was thought to be an important risk factor for MI (185-186) but not for angina pectoris (186), especially in the presence of other major risk factors such as blood cholesterol, smoking, and hypertension (186). Hulley et al. (113) showed that elevated total triglyceride levels in association with total cholesterol levels below the seventy-fifth percentile did not indicate increased risk for mortality from CHD. More recently, the partition of the blood into the various lipoprotein fractions and the measurement of the ratio of LDL-C/HDL-C (21,26,41,158), or the ratio of TC/HDL-C (41,90-91,111) is used for diagnosis of risk for CHD. The higher the ratio, the higher the risk of CHD (Appendix B).

Recommendations

Researchers have shown that high blood cholesterol levels are associated with a high risk of CHD. Reduction of high blood cholesterol levels reduces atherosclerosis and CHD in some individuals. Using data from the Division of Vital Statistics at National Center for Health Statistics (187), Goor et al. (188) investigated the secular trends of these diseases and suggested that the reasons for the recent decline in the mortality and incidence of ischemic heart disease may be due to the improvements in diet, mainly the decreased intakes of cholesterol and saturated fat and an increased P/S ratio with concomitant declines in blood cholesterol concentration, decreased cigarette smoking, improved hypertension control, and possibly increased leisure-time physical exercise. Up to now, no article has indicated any harm caused from this reduction of fat and cholesterol intake. Therefore, it is reasonable to follow the dietary guidelines proposed by the American Heart Association (AHA) (53,56). These guidelines are as follows: total fat should comprise 30 percent of the total kilocalories; carbohydrate, 55 percent, and protein, 15 percent. The fat should contain approximately equal amounts of saturated, monounsaturated, and polyunsaturated fatty acids (i.e., each should contribute about 10 percent of the total kilocalories). Complex carbohydrates should constitute the major source of the total carbohydrates. Total kilocalories should be sufficient to

maintain the individual's best weight. Cholesterol intake should be less than 300 mg per day for the general public and below 200-250 mg per day and even 100-150 mg per day for moderate and high risk populations, respectively.

METHODOLOGY

Subjects

Upon approval by the University Review Board Involving Human Subjects at Virginia Tech, sixty-seven white males who were participants in the Cardiac Therapy and Intervention Program conducted at Virginia Tech between June, 1982, and July, 1983, were recruited as subjects. These subjects were identified as belonging to either the cardiac or the intervention group.

By definition, the cardiac group consisted of subjects 1) who had experienced at least one cardiovascular event, (e.g., coronary heart disease, myocardial infarction or heart attack, angina pectoris, or ischemic heart disease), 2) who exhibited a combination of cardiac risk factors (i.e., hypertension, obesity, hypercholesterolemia, diabetes), or 3) who demonstrated abnormal electrocardiograph (EKG), blood pressure, or angina and dyspnea responses under conditions of graded exercise testing. The intervention group was defined as those subjects having no cardiovascular events nor electrocardiograph abnormalities, asymptomatic ischemic heart disease (IHD), or significant patterns of IHD risk, but no known disease and negative exercise test results.

Three time periods were chosen for this study: 1) a 1982-83 baseline period, 2) a 1983-84 short-term follow-up period, and 3) a 1986 long-term follow-up period. Subjects were further identified as being either active or inactive. An active participant was defined as one who continued involvement in the program since the 1982-83 baseline period. An inactive participant was one who ceased active involvement six to nine months after the 1982-83 baseline period.

These males were targeted as subjects because they had a complete set of data (i.e., body weights and fasting blood lipid profiles as well as dietary records in the baseline and the short-term follow-up periods). The protocol excluded patients either less than 28 years old (11 in the intervention group) or more than 68 years old (1 in the cardiac group). In addition, subjects who had not continued involvement in the program for at least 6 to 9 months (7 in the cardiac group and

15 in the intervention group) as well as those who stayed in this program but who did not have complete dietary or blood records for either the short-term or long-term follow-up periods (9 in the cardiac group and 25 in the intervention group) also were not included in this study. In total 67 of 138 males enrolled in the program qualified for the study. All subjects were identified as belonging to one of four subgroups (CA-cardiac active, CI-cardiac inactive, IA-intervention active, II-intervention inactive) based on the definitions given above (Table 1).

Procedures

Recruitment of Subjects

A letter with a returnable postcard enclosed was sent to each of the potential subjects to provide information about the purpose of this study (Appendix C). Follow-up assessments constitute a routine practice of the Cardiac Therapy and Intervention Program. Specifically, for currently active participants, blood chemistry data (including blood lipid profiles) are obtained every three months for the cardiac group and every six months for the intervention group during the first year. Thereafter, this information was obtained every six months for the cardiac group and once a year for the intervention group. If the subject was currently active and recently participated in such a routine follow-up assessment, that data was used for the long-term follow-up period. An appointment was made to have blood drawn for all other subjects. All currently and formerly active participants who constituted the sample signed a consent form (Appendix D)

Table 1. Sample sizes for the cardiac and intervention groups at each time period

Time period	Cardiac		Intervention	
	Active	Inactive	Active	Inactive
1982-83 baseline	16	19	19	13
1983-84 short-term follow-up	16	19	19	13
1986 long-term follow-up	16	15	19	12

Collection of Data for the Baseline and Short-term Follow-up Periods

The blood lipid profiles, heights, weights, and the systolic and diastolic blood pressure values for the baseline period and the short-term follow-up period were obtained from the medical records of each participant. Dietary records on file also were reviewed so that the following dietary intake information could be obtained: total kilocalories, total fat, saturated fat, linoleic acid, oleic acid, dietary cholesterol, carbohydrate, and protein. Throughout this study, linoleic acid was used as a measure of the PUFA content of the diet and oleic acid was used as a measure of the monounsaturated fatty acid content of the diet. All of these data were obtained when the participants joined the program. Between the time they joined the program and the short-term follow-up period, all participants received individualized dietary recommendations based on guidelines developed by the AHA. Further reinforcement was provided through nutrition education by the program staff.

Collection of Data for the Long-term Follow-up Period

Each subject in the long-term follow-up group kept a 3-day dietary intake record (one non-working day and two working days). These were compared with the 3-day dietary records from the baseline and the short-term follow-up periods. A sample copy of the food record and the guidelines for recording food consumed and estimating food portion sizes is given in Appendix E. Each of the records from the long-term follow-up period was reviewed with the subject to insure completeness and accuracy. Throughout the study, all subjects provided their own meals. All dietary data were coded and processed using the computer program developed by Wentworth and Choquette (189) and currently employed in the Cardiac Therapy and Intervention Program at Virginia Tech. The data base was from Home and Garden Bulletin No. 72 (190), and additional foods added from commercial sources and USDA Agriculture Handbook No. 8 (191). Dietary cholesterol values were calculated by using values from Leveille et al (192). Also, the percentages

of kilocalories consumed as total fat, saturated fat, linoleic acid, oleic acid, carbohydrate, and protein and the P/S ratio were determined. The protein intake also was compared to the Recommended Dietary Allowance (RDA).

Other parameters which were compared for each subgroup included the following: blood lipid profile (total cholesterol, LDL-C, HDL-C, and TG), weight change, and changes in body mass index (BMI) and blood pressure (BP). Blood samples were drawn from an antecubital vein by a registered nurse after an overnight fast of at least 12 hours and were used to determine serum total cholesterol (TC), HDL-C, LDL-C and TG concentrations. LDL-C was calculated according to the following formula: $LDL = TC - HDL - 1/5TG$ (21). The blood samples were sent to Roche Biomedical Laboratories for analysis since this procedure was consistent with that routinely followed by the Cardiac Therapy and Intervention Program.

Heights were measured using a Holtain stadiometer and weights (in light clothing) were measured on a beam balance scale. Quetelet's index: $(\text{weight in KG})/(\text{height in M})^2$ was used to estimate the body mass index (BMI) (193). Systolic and diastolic blood pressures (BP) were measured on the right arm with a mercury manometer after 10 minutes rest. Information regarding general medical history and smoking habits was obtained by using a questionnaire (Appendix F). Twenty-five subjects returned these questionnaires. For those who did not return their questionnaires, medical records were reviewed to determine if the information was available. Subjects were identified as either smokers, nonsmokers, or exsmokers. The number of blood lipid profiles and dietary records obtained from individuals in each subgroup for each period of time are shown in Tables 2 and 3, respectively.

Table 2. Numbers of blood lipid profiles obtained at each time period

Time period	Cardiac		Intervention	
	Active	Inactive	Active	Inactive
1982-83 baseline	16	19	19	13
1983-84 short-term follow-up	16	18	19	13
1986 long-term follow-up	16	15	19	11

Table 3. Numbers of dietary records obtained at each time period

Time period	Cardiac		Intervention	
	Active	Inactive	Active	Inactive
1982-83 baseline	16	19	18	13
1983-84 short-term follow-up	14	16	11	9
1986 long-term follow-up	11	8	10	2

Data Analysis

All data were analyzed within categories---dietary, plasma lipids, anthropometric measures, and blood pressure. The analysis was comprised of three major parts: 1) profiles of each group at each time period, 2) univariate comparisons of the four subgroups across all time periods, and 3) a discriminant analysis.

The first examination was done to determine if there were any differences between groups (cardiac or intervention) and status (active and inactive) across periods of times (baseline, short-term follow-up, and long-term follow-up). Differences in means of blood lipid components, blood pressure values, dietary components, and anthropometric data were evaluated. All variables were analyzed separately by the general linear model (GLM) procedure of the Statistical Analysis System (SAS) (194). The model contained the main effects of group, status, and time, and all interactions. Significant differences among any main effect were further analyzed using the LSD t test, Duncan's multiple range test, and the Scheffe' test. These tests were chosen to indicate a range of differences among any significantly different main effects. The LSD t test and Duncan's multiple range test give a liberal interpretation of these differences while the results of the Scheffe' test give a conservative interpretation. Note that for two treatments (group---cardiac or intervention and status---active and inactive), there were only two levels. Hence, multiple comparisons were not needed. The results of the above analysis gave the overall profile of each subgroup at each time period. Secondly, the Least Square Means (LSMeans) method was used to compare means using a model based approach to investigate interactions among group, status, and time. Finally, a discriminant analysis was employed to determine which combination of risk factors was most influential in distinguishing the cardiac group from the intervention group. Stepwise discriminant analysis and canonical discriminant analysis were used to select the variables which would produce a good discrimination model. This model could be used as a criterion for classifying program clients in the future.

The dietary intake parameters were assessed to determine how well the subjects complied with the dietary guidelines of the AHA. The following dietary guidelines recommended by the AHA were used in these comparisons:

1. Total kilocalories should be sufficient to maintain the individual's best body weight.
2. The kilocalorie containing nutrients---fat, protein, and carbohydrate---should contribute approximately 30, 15, and 55 percent of the kilocalories, respectively.
3. The fat should contain approximately equal amounts of saturated, monounsaturated, and polyunsaturated fatty acid, (i.e., each should contribute about 10 percent of the total kilocalories).
4. Cholesterol intake should be below 300 mg per day for the general public and below 200-250mg per day and even 100-150 mg per day for moderate and high risk populations, respectively.

The blood lipid profiles were compared with the reference values from the LRCPS data (50) to indicate how the values of these subjects stood in reference to those of the general American population.

RESULTS AND DISCUSSION

Description of Subjects

The means and standard deviations for age and anthropometric data (i.e., height, weight, and BMI) for each subgroup at the baseline period are given in Table 4. The BMI was used as an index of obesity since it reflects the degree of adiposity better than the absolute weight. It has been used in such clinical trials as the Framingham Study (178) and the Lipid Research Clinics Coronary Primary Prevention Trials (50). Frame sizes were not estimated in this study. However, a general idea regarding the relative degree of adiposity of these subjects can be obtained by comparing their BMI values with the following minimum body mass indices indicating obesity (194):

Frame size	BMI (Males)
Small	25.4
Medium	27.5
Large	29.9

The intervention inactive (II) group was younger, heavier, and taller than the other three groups. Compared to the value given for males of medium frame size, only the II group had a higher mean value than the reference value, yet the degree of difference was minimal (27.8 ± 2.66 vs. 27.5) (Table 4). However, when the standard deviations were taken into consideration, both intervention subgroups tended to exceed the reference value indicating obesity assuming a large frame size; therefore, there is a need for continuing monitoring of those subjects who exceed their desirable weight ranges. The number of subjects exhibiting selected risk factors for CHD is shown in Table 5. This provides a profile of the four subgroups at the baseline period.

Table 4. Means and standard deviations for age and anthropometric data (i.e., height, weight, and BMI) for each subgroup at the baseline period

Parameter	Cardiac		Intervention	
	Active	Inactive	Active	Inactive
Age (Years)	53.4± 5.8	53.5± 7.4	45.1± 11.4	40.6± 7.0
Weight (lbs)	192± 24.8	182± 31.6	188.3± 29.1	200.3± 22.5
Height (Inches)	70.7± 2.6	70.3± 2.5	70.4± 2.2	71.2± 3.2
BMI	26.9± 2.75	25.8± 3.52	26.6± 3.37	27.8± 2.66

Table 5. Numbers of subjects in each subgroup with selected risk factors for CHD at the baseline period

Factor	Cardiac		Intervention	
	Active	Inactive	Active	Inactive
Family history early heart disease	4	2	7	4
Myocardial infarction (Heart attack)	11	16	3	0
Angina pectoris	2	5	0	0
Angina type chest pain	3	6	1	0
Ischemic heart disease (CHD)	1	1	0	0
Other type CHD	1	1	0	0
Abnormal EKG	8	15	1	0
Hypertension	12	17	11	4
Hypercholesterolemia	9	10	9	7
Hypertriglyceridemia	7	11	12	8
Diabetes mellitus	2	5	1	1
Coronary bypass surgery	3	3	0	0
Smoking	4	9	6	5

Profile of Each Subgroup Regarding Anthropometric Measurements, Dietary Intake, Blood Lipid Profile, and Blood Pressure at Three Time Periods

The analysis of variance using the general linear model (GLM) identified the effects of group (cardiac or intervention), status (active or inactive), and time (baseline, short-term follow-up, and long-term follow-up); furthermore, the interaction between group and status at each time period was used to test each subgroup change. From these results, a general profile of each subgroup regarding anthropometric measurements, dietary intakes, blood lipid profile, and blood pressure at the three time periods was derived. These are shown in Tables 6-9.

Cardiac Active Group (CA)

The body weight and BMI remained virtually unchanged across all time periods. In this group, most of the dietary components had the lowest mean values in the short-term follow-up period except for the mean values of linoleic acid, P/S ratio, and protein. These had the lowest mean value at the long-term follow-up period (Table 6).

Compared to the specific dietary guidelines of the AHA (53,56), the CA group had higher percentages of kilocalories as fat (30 percent) and lower percentages of kilocalories provided by linoleic acid (10 percent) across all time periods. The lowest percentage of kilocalories provided by linoleic acid was found in the long-term follow-up period. The percentage of kilocalories as protein was also higher than the recommended value (15 percent) but a trend toward a gradual decrease was found. The percentages of kilocalories provided by carbohydrates showed a gradually increasing trend over time. The dietary cholesterol level was higher than the recommended value (200-250 mg/day) for this group.

Table 6. Sample means and standard deviations of selected parameters of the Cardiac Active Group at three time periods

Variable	Baseline period	Short-term follow-up	Long-term follow-up
Body weight (lb)	192 ± 24.8	190 ± 23.4	192 ± 23.0
Body mass index (W/H ²)	26.9 ± 2.75	26.7 ± 2.50	26.9 ± 2.62
Total kilocalories intake (Kcal)	1839 ± 561	1657 ± 390	1740 ± 475
Total fat intake (gm)	72 ± 30.4	62 ± 15.4	65 ± 25.9
Percent of kilocalories as fat	34.4	34.1	32.9
Saturated fat intake (gm)	22 ± 9.1	18 ± 4.4	20 ± 9.0
Percent of fat as SFA	30.6	29.3	32.3
Percent of kilocalories as SFA	10.7	10.1	10.4
Linoleic acid intake (gm)	14 ± 8.7	12 ± 4.4	11 ± 5.4
Percent of fat as linoleic acid	19.2	19.7	17.9
Percent of kilocalories as linoleic acid	6.6	6.7	5.8
Oleic acid intake (gm)	22 ± 9.6	19 ± 6.8	21 ± 8.9
Percent of fat as oleic acid	31.5	30.4	32.0
Percent of kilocalories as oleic acid	10.8	10.4	10.9
Dietary P/S ratio	.68 ± .10	.74 ± .10	.68 ± .10
Dietary cholesterol intake (mg)	314 ± 144.9	263 ± 125.2	265 ± 181.7
Carbohydrate intake (gm)	216 ± 77.6	203 ± 54.0	228 ± 83.2
Percent of kilocalories as carbohydrate	47.0	49.1	52.7
Protein intake (gm)	86 ± 28.6	75 ± 14.5	71 ± 20.6
Percent of kilocalories as protein	19.0	18.6	16.5
Percent RDA protein intake	153 ± 51.2	134 ± 26.0	152 ± 85.9
Systolic blood pressure (mmHg)	133 ± 23	127 ± 15	125 ± 19
Diastolic blood pressure (mmHg)	84 ± 15	80 ± 10	79 ± 13
Blood cholesterol (mg/dl)	204 ± 37.2	212 ± 39.3	225 ± 45.6
Blood HDL-C (mg/dl)	35 ± 7.2	35 ± 9.3	37 ± 6.1
Blood LDL-C (mg/dl)	140 ± 37.1	145 ± 33.2	159 ± 48.1
Blood triglyceride (mg/dl)	147 ± 54.2	163 ± 62.1	163 ± 84.3
TC/HDL-C	6.0 ± 1.37	6.7 ± 2.68	6.2 ± 1.42
LDL-C/HDL-C	4.1 ± 1.24	4.6 ± 2.26	4.3 ± 1.00

The mean values of systolic and diastolic blood pressure decreased across time periods; while the mean level of blood cholesterol, LDL-C and TG, showed an increasing trend from the baseline period. Moreover, the mean values of TC/HDL-C and LDL-C/HDL-C ratio were highest in the short-term follow-up period.

The mean values of total cholesterol were around the 50th percentile relative to the data from the LRCPS data (appendix A). The mean values of HDL-C were around the 25th percentiles. The mean values of LDL-C were between the 50th and the 75th percentiles. The mean values of TG were between the 50th and the 75th percentiles. Both the ratios of TC/HDL-C and LDL-C/HDL-C were between the average risk and two times the average risk.

Cardiac Inactive Group (CI)

The means and standard deviations of the selected dietary components, the blood lipid parameters, and the blood pressure values for the CI group at the three time periods are shown in Table 7. The mean values for body weight and BMI for this group were slightly increased in the long-term follow-up period. The mean values of the kilocalories, fat, linoleic acid, carbohydrate, and protein were highest in the long-term follow-up period, while the highest mean values of saturated fat and cholesterol intake were found in the short-term follow-up period. Compared to the guidelines of the AHA, the percentage of kilocalories provided by total fat was above the recommended value (30 percent) across all time periods; the percentages of kilocalories as saturated fat and as oleic acid were greater than 10 percent in the short-term follow-up period but at or below 10 percent in the other two time periods, and all the percentages of kilocalories provided by linoleic acid were much less than the recommended value (10 percent) across all time periods. The percentage of kilocalories provided by protein was higher than 15 percent in all three time periods, and the percentage of kilocalories provided by carbohydrate was less than 55 percent in the baseline and the short-term follow-up periods, but improved by the long-term follow-up period.

Table 7. Sample means and standard deviations of selected parameters of the Cardiac Inactive Group at three time periods

Variable	Baseline period	Short-term follow-up	Long-term follow-up
Body weight (lb)	182 ± 31.6	182 ± 30.3	186 ± 38.5
Body mass index (W/H ²)	25.8 ± 3.52	25.8 ± 3.41	26.7 ± 4.38
Total kilocalories intake (Kcal)	1713 ± 433.1	1723 ± 482.4	2084 ± 331.2
Total fat intake (gm)	64 ± 22.8	69 ± 30.8	73 ± 23.1
Percentage of kilocalories as fat	33.6	35.1	31.0
Saturated fat intake (gm)	19 ± 8.4	22 ± 11.3	20 ± 7.4
Percentage of fat as SFA	31.9	27.5	32.7
Percentage of kilocalories as SFA	9.9	11.5	8.6
Linoleic acid intake (gm)	12 ± 6.0	13 ± 8.3	14 ± 5.1
Percentage of fat as linoleic acid	18.6	18.8	20.1
Percent of kilocalories as linoleic acid	6.2	6.5	6.0
Oleic acid intake (gm)	19 ± 8.1	22 ± 10.7	22 ± 7.6
Percentage of fat as oleic acid	29.7	32.0	29.2
Percentage of kilocalories as oleic acid	10.2	11.4	9.2
Dietary P/S ratio	.80 ± .09	.64 ± .09	.66 ± .09
Dietary cholesterol intake (mg)	368 ± 198.8	370 ± 183.9	334 ± 187.5
Carbohydrate intake (gm)	206 ± 70.3	204 ± 86.8	282 ± 58.9
Percentage of kilocalories as carbohydrate	47.9	46.9	54.3
Protein intake (gm)	74 ± 18.1	79 ± 17.6	89 ± 17.0
Percentage of kilocalories as protein	17.7	19.4	17.3
Percentage RDA protein intake	131 ± 32.5	141 ± 31.8	159 ± 30.3
Systolic blood pressure (mmHg)	125 ± 20	129 ± 19	124 ± 16
Diastolic blood pressure (mmHg)	78 ± 8	78 ± 5	75 ± 8
Blood cholesterol (mg/dl)	206 ± 34.5	216 ± 35.5	228 ± 50.6
Blood HDL-C (mg/dl)	36 ± 7.7	39 ± 10.3	34 ± 8.4
Blood LDL-C (mg/dl)	135 ± 31.6	137 ± 34.9	159 ± 41.9
Blood triglyceride (mg/dl)	166 ± 54.1	180 ± 63.6	171 ± 74.5
TC/HDL-C	5.9 ± 1.22	5.8 ± 1.53	6.8 ± 1.75
LDL-C/HDL-C	3.8 ± 0.97	3.7 ± 3.68	4.7 ± 1.31

Finally, the dietary cholesterol level was higher than the recommended value (200-250 mg/day) across the three time periods.

Systolic and diastolic blood pressures differed minimally across the three time periods and were below the upper limit critical values (140 mmHG). The highest mean levels of blood cholesterol, LDL-C, and the ratios of TC/HDL-C and LDL-C/HDL-C were found in the long-term follow-up period. The highest mean value of TG was in the short-term follow-up period, whereas the baseline period had the lowest one. The expected result of the elevated mean level of HDL-C was found in the short-term follow-up period and the lowest values in the long-term follow-up period.

The mean values of total cholesterol were around the 50th percentile of the LRCPS data (Appendix A). HDL-C levels were around the 25th percentile, LDL-C levels were around the 50th to the 75th percentiles, and TG levels were around the 75th percentile. Both the TC/HDL-C and the LDL-C/HDL-C ratios were between average risk and two times the average risk.

Intervention Active Group (IA)

The means and standard deviations of the selected dietary components, blood lipid parameters, and blood pressure values in the IA group at the three time periods are listed in Table 8. The mean values of body weight and BMI were lowest in the short-term follow-up period and highest in the baseline period. For most of the dietary components except cholesterol intake, the highest mean values were found in the long-term follow-up period, whereas the short-term follow-up period had the lowest one. The highest P/S ratio and the lowest dietary cholesterol intakes were found in the long-term follow-up period. All the P/S ratios were lower than the recommended value, 1.0, in any time period. The dietary cholesterol level was gradually decreased across the time periods. The percentage of kilocalories provided by total fat, saturated fat, oleic acid, and protein were above the values recommended by the AHA (53,56) and showed minimum alterations across time. The

Table 8. Sample means and standard deviations of selected parameters of the Intervention Active Group at three time periods

Variable	Baseline period	Short-term follow-up	Long-term follow-up
Body weight (lb)	188 ± 29.1	183 ± 24.6	187 ± 27.0
Body mass index (W/H ²)	26.6 ± 3.37	25.9 ± 2.72	26.4 ± 3.01
Total kilocalories intake (Kcal)	1877 ± 411	1659 ± 447	2076 ± 533
Total fat intake (gm)	80 ± 17.2	66 ± 20.6	88 ± 31.3
Percentage of kilocalories as fat	38.9	36.5	37.8
Saturated fat intake (gm)	26 ± 6.0	20 ± 5.6	27 ± 11.2
Percentage of fat as SFA	32.7	30.1	30.5
Percentage of kilocalories as SFA	12.8	10.8	11.6
Linoleic acid intake (gm)	14 ± 3.9	12 ± 3.8	17 ± 7.2
Percentage of fat as linoleic acid	17.9	18.6	19.3
Percentage of kilocalories as linoleic acid	7.0	6.8	7.3
Oleic acid intake (gm)	26 ± 5.8	21 ± 7.0	28 ± 12.5
Percentage of fat as oleic acid	32.0	30.9	31.0
Percentage of kilocalories as oleic acid	12.5	11.3	11.7
Dietary P/S ratio	.58 ± .10	.63 ± .10	.67 ± .10
Dietary cholesterol intake (mg)	393 ± 266.1	319 ± 147.9	269 ± 95.5
Carbohydrate intake (gm)	199 ± 52.7	180 ± 49.7	221 ± 65.3
Percentage of kilocalories as carbohydrate	42.6	44.3	43.1
Protein intake (gm)	84 ± 27.0	72 ± 17.2	87 ± 26.7
Percentage of kilocalories as protein	17.9	17.8	17.3
Percentage RDA protein intake	144 ± 58.1	130 ± 31.0	156 ± 47.7
Systolic blood pressure (mmHg)	139 ± 16.0	132 ± 17.3	129 ± 21.3
Diastolic blood pressure (mmHg)	88 ± 10.7	84 ± 10.7	86 ± 10.2
Blood cholesterol (mg/dl)	213 ± 37.8	217 ± 28.9	219 ± 33.1
Blood HDL-C (mg/dl)	41 ± 10.7	40 ± 10.5	39 ± 9.3
Blood LDL-C (mg/dl)	135 ± 28.4	142 ± 26.7	150 ± 36.0
Blood triglyceride (mg/dl)	182 ± 97.3	170 ± 75.7	146 ± 73.8
TC/HDL-C	5.5 ± 1.73	5.9 ± 2.58	5.9 ± 1.70
LDL-C/HDL-C	3.5 ± 1.20	3.9 ± 1.93	4.1 ± 1.44

percentage of kilocalories provided by linoleic acid and carbohydrate were much less than the recommended values across all time periods. The highest dietary cholesterol intake was found in the baseline period and the lowest one was seen in the long-term follow-up period.

The systolic and diastolic blood pressures of the IA group remained similar across all time periods. The mean levels of blood cholesterol, LDL-C, and the ratios of TC/HDL-C and LDL-C/HDL-C gradually increased across time. The opposite results were found for the mean levels of HDL-C and TG, but all of the ratios were higher than the recommended values. The mean values of total cholesterol were around the 50th percentile. HDL-C levels were around the 25th to the 50th percentiles of the reference LRCPS data (Appendix A). LDL-C levels were around the 50th percentile, and TG levels were around the 75th percentile. Both the TC/HDL-C and LDL-C/HDL-C ratios were between the average risk and two times the average risk.

Intervention Inactive Group (II)

The means and standard deviations of the selected dietary components, blood lipid parameters, and blood pressure values in the II group at the three time periods are given in Table 9. The body weight and the BMI gradually decreased in the II group. The lowest mean values of dietary components (except the P/S ratio) were found in the short-term follow-up period, while the highest mean values were in the baseline period, except for linoleic acid, dietary cholesterol, and protein intake which were highest in the long-term follow-up period. The percentages of kilocalories provided by total fat, saturated fat, oleic acid, and protein were higher than the values recommended by the AHA. The percentages of kilocalories provided by linoleic acid and carbohydrate were lower than each separately recommended values. Although the intake of linoleic acid showed a gradually increasing trend across time but the recommended level (10 percent) was never achieved. The dietary cholesterol level was lowest in the short-term follow-up period and highest in the long-term follow-up period; however, in each time period, the mean values of cholesterol were higher than the suggested value of 300 mg/day. The P/S ratio gradually increased across the time periods.

Table 9. Sample means and standard deviations of selected parameters of the Intervention Inactive Group at three time periods

Variable	Baseline period	Short-term follow-up	Long-term follow-up
Body weight (lb)	200 ± 22.5	195 ± 20.8	192 ± 21.9
Body mass index (W/H ²)	27.8 ± 2.66	27.1 ± 2.86	26.9 ± 3.08
Total kilocalories intake (Kcal)	2013plm.360	1630 ± 383	1978 ± 128
Total fat intake (gm)	91 ± 20.2	73 ± 24.9	90 ± 4.5
Percentage of kilocalories as fat	40.8	38.2	41.1
Saturated fat intake (gm)	29 ± 8.8	23 ± 6.2	29 ± 0.5
Percentage of fat as SFA	31.3	31.7	31.7
Percentage of kilocalories as SFA	12.9	12.1	13.1
Linoleic acid intake (gm)	15 ± 5.5	12.9 ± 6.7	20 ± 10.5
Percentage of fat as linoleic acid	16.6	19.9	22.4
Percentage of kilocalories as linoleic acid	6.7	7.1	9.0
Oleic acid intake (gm)	28 ± 7.6	24 ± 7.1	28 ± 2.3
Percentage of fat as oleic acid	30.4	33.7	30.5
Percentage of kilocalories as oleic acid	12.5	12.8	12.6
Dietary P/S ratio	.57 ± .11	.71 ± .11	.73 ± .11
Dietary cholesterol intake (mg)	395 ± 155.1	349 ± 157.9	401 ± 135
Carbohydrate intake (gm)	211 ± 62.3	172 ± 44.0	208 ± 21.2
Percentage of kilocalories as carbohydrate	41.8	44.0	42.2
Protein intake (gm)	78 ± 16.0	71 ± 14.9	92 ± 5.7
Percentage of kilocalories as protein	15.7	17.4	18.4
Percentage RDA protein intake	140 ± 28.7	126 ± 26.7	164 ± 11.7
Systolic blood pressure (mmHg)	135 ± 15	132 ± 18	129 ± 14
Diastolic blood pressure (mmHg)	86 ± 8	87 ± 12	86 ± 13
Blood cholesterol (mg/dl)	215 ± 71.1	205 ± 64.0	219 ± 53.2
Blood HDL-C (mg/dl)	33 ± 9.3	40 ± 9.3	38 ± 15.0
Blood LDL-C (mg/dl)	137 ± 50.4	121 ± 21.1	138 ± 37.9
Blood triglyceride (mg/dl)	210 ± 112	181 ± 134	213 ± 174
TC/HDL-C	7.1 ± 3.3	5.3 ± 1.6	6.6 ± 3.3
LDL-C/HDL-C	4.5 ± 2.1	3.2 ± 0.9	4.1 ± 2.0

The highest mean value of systolic blood pressure was in the baseline period and the lowest in the long-term follow-up period. The diastolic blood pressure showed minimum change across the time periods. The mean values of blood cholesterol, LDL-C, and TG were highest in the long-term follow-up period, and the lowest in the short-term follow-up period; whereas, the highest mean levels of HDL-C and the ratios of TC/HDL-C and LDL-C/HDL-C occurred in the short-term follow-up period, and the lowest levels and ratios occurred in the baseline period. Compared with the LRCPS data (Appendix A), the mean values of total cholesterol were around the 50th to the 75th percentiles. HDL-C levels were around the 25th percentile, LDL-C levels were around the 25th to the 50th percentiles, and TG levels were around the 75th to the 90th percentiles. The TC/HDL-C ratio was between the average risk and two times the average risk and the LDL-C/HDL-C ratio was around the average risk.

Summary of the Four Subgroups

The lowest mean values of the dietary components, blood pressure, and blood lipid values were expected to be found in either the short-term follow-up period for all groups or the long-term follow-up periods for the active groups since the subjects received regular nutrition education intervention and exercise three times per week from the program. Among the four subgroups, the II group had the highest values of BMI across time, but the values gradually decreased. On the other hand, the BMI values gradually increased in the CI group. There were minimal alterations in the CA and the IA groups across the three time periods, but both had the lowest values in the short-term follow-up period. Therefore, the active groups behaved in accordance with the previous expectation. The following is a summary of findings relative to dietary parameters:

- The average percentages of kilocalories provided by protein were above the values recommended by the AHA (53,58) for all subgroups across all time periods, and also were higher than the average American daily intake value of around 15 percent (56).
- None of the groups had fewer than 30 percent of total kilocalories provided by fat at any time period examined. Both of the intervention groups had higher values than both of the cardiac groups. The former groups had a total fat intake near 40 percent of the total kilocalories, which was similar to the average intake of Americans (56).
- The cardiac groups were compliant with the recommendation that saturated fat intake should account for 10 percent of the total kilocalories.
- None of the groups met the recommendation that PUFAs constitute 10 percent of the total kilocalories. The least desirable condition (5.8 percent) was found in the CA group in the long-term follow-up period, while the most desirable condition (9.0 percent) was seen in the

II group in the long-term follow-up period. The current average dietary intake of PUFA among the general American public is about 5-7 percent of the total kilocalories (56).

- The percentage of calories provided by oleic acid was slightly higher than the recommended values (10 percent) for all four subgroups in each of the three time periods except that the CI group had a low oleic acid intake in the long-term follow-up period.
- None of the groups in any of the time periods ever had a P/S ratio close to 1. The highest P/S ratio was found in the short-term follow-up period for the CA group, at the baseline period for the CI group, and in the long-term follow-up period for the IA and the II groups. The lower P/S ratio (below 1) might partially explain the higher levels of blood parameters and is a very important point which should be stressed in improving the dietary habits of the program participants.
- In the three time periods, few groups ever exhibited higher carbohydrate intake than the recommended value of 55 percent. Both of the cardiac groups had diets containing higher kilocalories from carbohydrates than did either of the intervention groups across time. This difference was most clearly observable in the long-term follow-up period. In the rest of the time periods, the four subgroups had a carbohydrate intake similar to the average intake of the general U.S. public (40-45 percent of calories) (56).
- The suggestion that cholesterol intake should be below 300 mg for the general public and 200-250 mg or even 100-150 mg for hypercholesterolemics has been recommended by the AHA (53). The usual American diet contains 500 to 750 mg of cholesterol per day (3). All four subgroups had higher mean intakes of cholesterol than the suggested values. Generally, the CI group had a higher mean intake of cholesterol than the CA group, and the II group had a higher mean intake of cholesterol than the IA group. Thus, both inactive groups had higher cholesterol intakes than both active groups. The two cardiac subgroups should reduce the

cholesterol intake below 200-250 mg/dl (53) and the two intervention subgroups should reduce the cholesterol intake below 300 mg/dl (53).

- The caloric intake of all of the subgroups across the three time periods was around 2,000 kilocalories which was an energy expenditure value associated with a lesser risk of experiencing a myocardial infarction (195).

The observations relative to blood lipid parameters are given below. The blood cholesterol values and its fractions related to CHD risk are usually adjusted by age; therefore, the mean age of each subgroup (Table 4) should be taken into consideration in interpreting these observations. Values of these blood parameters around the 75th percentile were thought to indicate moderate-risk; around the 90th percentile, high risk.

- The lowest mean values of TC, LDL-C, and TG were found in the baseline period for the CA, CI, and IA groups (but not TG), and in the short-term follow-up period for the II group. The highest mean values of HDL-C were found in the long-term follow-up period for the CA group, in the short-term follow-up period for the CI and the II groups, and in the baseline period for the IA group.
- The lowest mean values of the ratios of TC/HDL-C and LDL-C/HDL-C were found in the baseline period for the CA and the IA groups, and in the short-term follow-up period for the CA and the II groups. The II group had the lowest mean values of both TC/HDL-C and of LDL-C/HDL-C. Both of these were found in the short-term follow-up period.
- Normal adult values for blood cholesterol levels in the U.S. are 120-250 mg/dl (20). The average blood cholesterol level among American adult males is about 210 mg/dl (26). Levels below 200 mg/dl are associated with significantly less coronary risk than levels above 200 mg/dl (20). The mean values of blood cholesterol were between 50th and the 75 percentile for both of the intervention groups and below the 50th percentile for both of the cardiac groups.

- The levels of blood HDL-C were 35-37 mg/dl in the CA group and 34--39 mg/dl in the CI group among the three time periods. However, compared to the figures from the LRCPS Data (50), both the CA and the CI groups had HDL-C levels around the 10th to the 25th percentiles. The levels of blood HDL-C ranged from 39--41 mg/dl for the IA group and from 33--40 mg/dl for the II group among the three time periods. Yet, compared to the age-adjusted figures from the LRCPS data, the IA group had HDL-C levels between the 20th and the 50th percentiles, and the II group had values between the 10th and the 50th percentiles. All four subgroups had HDL-C values above the critical value (29 mg/dl) for coronary risk suggested by Hoeg et al. (201), but had lower values than the general American public (see appendix A).
- The normal value of LDL-C is 104-109 mg/dl by Kannel's study (20). In the baseline and the short-term follow-up periods, all four subgroups had a value below or close to the 50th percentile of the LRCPS data; while all had values above the 50th percentile in the long-term follow-up period.
- The average risk (refers to normal value or standard value such that people at this value have a lower chance of having CHD) of CHD for males regarding the TC/HDL-C and the LDL-C/HDL-C ratios were 4.97 and 3.55, respectively; the ratios associated with two times the average risk were 9.55 and 6.25, respectively (90). Consistent results were seen in the four groups in the three time periods regarding the ratios of TC/HDL-C. The mean values of the TC/HDL-C ratio were higher than 4.97 and lower than 9.55, i.e., each subgroup had a CHD risk between average and two times the average. Also, the mean values of the LDL-C/HDL-C ratio were higher than 3.55 (average risk) for each group in all three time periods except that the II group had a lower mean value in the short-term follow-up period. Detailed information regarding these risk values is in Appendix B. Risk values provided by Kannel et al. (196) are given in Appendix B.

- The CA group had all TG values below the 75th percentile as reported in the LRCPS data across all time periods. The CI group had a value below the 75th percentile only in the baseline period. The IA group decreased their blood TG level from 182 mg/dl in the baseline period to 146 mg/dl in the long-term follow-up period. The latter value was below the 75th percentile of the LRCPS data. The opposite condition was found in the II group which had all mean values of TG above the 75th percentile across all time periods.

Comparison Among Four Subgroups Across All Time Periods

Since there were virtually no significant differences due to the interaction of subgroups and time periods, comparisons were made among the four subgroups without considering a time effect. The means of total kilocalories, total fat, saturated fatty acid, oleic acid, linoleic acid, P/S ratio, dietary cholesterol, carbohydrate, and protein intakes did not differ significantly among the subgroups. Detailed information for each dietary and blood lipid variable and for blood pressure values is shown in Table 10. Note that the variances in this and the three following tables (Tables 10-13) are derived from the statistical model used (least square means).

The lowest intake of the overall dietary components was seen in the CA group. All the groups had higher percentages of kilocalories from fat, saturated fat, oleic acid, and protein than the values recommended by the AHA, although the values for the cardiac groups were closer to the recommended levels. There were no statistically significant differences among the four subgroups. The percentages of kilocalories provided by linoleic acid and carbohydrate were lower than each of the separately recommended values, and there were no significant differences among the four subgroups.

Table 10. Sample means and estimated standard errors of the means of selected parameters for the CA, CI, IA, and II subgroups across all time periods

Variable	Cardiac active	Cardiac inactive	Intervention active	Intervention inactive
§ Body weight (lb) (P = .03)	191 ± 3.9	184 ± 3.8	186 ± 3.6	196 ± 4.4
Body mass index (W/H ²)	26.8 ± 0.45	26.1 ± 0.44	26.3 ± 0.4	27.2 ± 0.51
Total kilocalories intake (Kcal)	1745 ± 70	1840 ± 73	1871 ± 75	1874 ± 123
Total fat intake (gm)	66 ± 3.8	68 ± 3.9	78 ± 4.1	85 ± 6.7
Percentage of kilocalories as fat	33.8	33.2	37.7	40.0
Saturated fat intake (gm)	20 ± 1.3	20 ± 1.3	24 ± 1.4	27 ± 2.3
Percentage of total fat as SFA	30.7	29.4	31.1	31.6
Percentage of kilocalories as SFA	10.4	10.0	11.7	12.7
Linoleic acid intake (gm)	13 ± 1.0	13 ± 1.0	14 ± 1.1	16 ± 1.7
Percentage of total fat as linoleic acid	18.9	19.2	18.6	19.6
Percentage of kilocalories as linoleic acid	6.3	6.2	7.0	7.6
Oleic acid intake (gm)	21 ± 1.3	21 ± 1.4	25 ± 1.4	27 ± 2.3
Percentage of total fat as oleic acid	31.3	30.3	31.3	31.5
Percentage of kilocalories as oleic acid	10.7	10.2	11.8	12.6
Dietary P/S ratio	.70 ± .06	.70 ± .05	.63 ± .06	.67 ± .07
Dietary cholesterol intake (mg)	281 ± 28.9	357 ± 29.3	327 ± 30.4	382 ± 49.6
Carbohydrate intake (gm)	216 ± 10.5	231 ± 10.9	200 ± 11.2	197 ± 18.4
Percentage of kilocalories as carbohydrate	49.6	49.7	43.3	42.7
Protein intake (gm)	77 ± 3.3	81 ± 3.4	81 ± 3.5	80 ± 5.7
Percentage of kilocalories as protein	18.0	18.2	17.6	17.2
Percentage RDA protein intake	146 ± 7	144 ± 7	143 ± 8	143 ± 12
Systolic blood pressure (mmHg)	128 ± 3	126 ± 3	133 ± 2	132 ± 3
Diastolic blood pressure (mmHg)	81 ± 2	77 ± 1	86 ± 1	86 ± 2
Blood cholesterol (mg/dl)	214 ± 6.3	217 ± 6.1	216 ± 5.8	213 ± 7.4
Blood HDL-C (mg/dl)	36 ± 1.4	37 ± 1.3	40 ± 1.3	37 ± 1.6
Blood LDL-C (mg/dl)	148 ± 5.2	144 ± 5.0	143 ± 4.8	132 ± 6.1
Blood triglyceride (mg/dl)	157 ± 12.7	172 ± 12.3	166 ± 11.7	201 ± 15.0
§ TC/HDL-C (P = .02)	6.0 ± 0.3	6.0 ± 0.3	5.5 ± 0.2	6.6 ± 0.3
§ LDL-C/HDL-C (P = .01)	4.1 ± 0.2	3.8 ± 0.2	3.5 ± 0.2	4.2 ± 0.2

(§ indicates significant differences)

In general, the II group consumed more total kilocalories, total fat, saturated fatty acid, linoleic acid, oleic acid, and dietary cholesterol than the other groups did, although no statistically significant difference was found. Moreover, group II exhibited the highest mean body weight and BMI values among the four subgroups. The body weight of the II group was significantly different from that of the CI group ($P = .03$). There were no significant differences relative to body weight among any of the other subgroups.

The lowest mean levels of blood cholesterol and LDL-C and the highest mean value of TG were found in the II group, although the differences from the other groups were not statistically significant. The highest mean values of the TC/HDL-C and the LDL-C/HDL-C ratios also were found in the II group. These differed significantly from the other groups ($P = .02$, and $P = .01$, respectively). The lowest mean values of the TC/HDL-C and the LDL-C/HDL-C ratios were found in the IA group. The II and the IA groups had higher mean values of systolic and diastolic blood pressures than the CA and the CI groups did, although no significant differences among them was found.

Comparison Among Three Time Periods

The differences among the three time periods for all four subgroups were compared. According to the results of the GLM procedure, there were no significant changes in daily protein, fat, saturated fatty acid, linoleic acid, oleic acid, and dietary cholesterol intakes across the three time periods. The P/S ratio also remained constant across the three time periods. However, despite the lack of statistically significant differences, there was a trend exhibited that, for most of the dietary components, the lowest consumption occurred in the short-term follow-up period. This may be explained by the effective results of dietary education by the program staff. Detailed information regarding these comparisons is listed in Table 11. There was a significant decrease ($P = .02$) in the

Table 11. Sample means and estimated standard errors of the means of selected parameters for the three time periods across all four subgroups

Variable	Baseline period	Short-term follow-up	Long-term follow-up
Body weight (lb)	191 ± 3.4	188 ± 3.4	189 ± 3.5
Body mass index (W/H ²)	26.8 ± 0.39	26.4 ± 0.39	26.7 ± 0.41
§ Total kilocalories intake (Kcal) (P = .02)	1860 ± 55	1667 ± 64	1969 ± 101
Total fat intake (gm)	77 ± 3.0	68 ± 3.5	79 ± 5.5
Percentage of kilocalories as total fat	37	36	36
Saturated fat intake (gm)	24 ± 1.0	21 ± 1.2	24 ± 1.9
Percentage of total fat as SFA	30.8	30.8	30.5
Percentage of kilocalories as SFA	11.6	11.1	10.9
Linoleic acid intake (gm)	14 ± 0.8	13 ± 0.9	16 ± 1.4
Percentage of total fat as linoleic acid	18.1	19.2	19.9
Percentage of kilocalories as linoleic acid	6.6	6.8	7.0
Oleic acid intake (gm)	24 ± 1.0	22 ± 1.2	25 ± 1.9
Percentage of total fat as oleic acid	30.9	31.7	30.7
Percentage of kilocalories as oleic acid	11.5	11.5	11.1
Dietary P/S ratio	.66 ± .05	.68 ± .05	.68 ± .05
Dietary cholesterol intake (mg)	368 ± 22.3	325 ± 26.0	317 ± 41.0
§ Carbohydrate intake (gm) (P = .04)	208 ± 8.3	190 ± 9.6	235 ± 15.1
Percentage of kilocalories as carbohydrate	44.8	46.1	48.1
Protein intake (gm)	80 ± 2.6	74 ± 3.0	85 ± 4.8
Percentage of kilocalories as protein	17.6	18.3	17.4
Percentage RDA protein intake (gm)	142 ± 6	133 ± 6	158 ± 10
Systolic blood pressure (mmHg)	133 ± 2	130 ± 2	127 ± 2
Diastolic blood pressure (mmHg)	84 ± 1	82 ± 1	82 ± 1
Blood cholesterol (mg/dl)	209 ± 5.4	212 ± 5.5	223 ± 5.8
Blood HDL-C (mg/dl)	36 ± 1.2	38 ± 1.2	37 ± 1.3
§ Blood LDL-C (mg/dl) (P = .03)	137 ± 4.5	136 ± 4.5	152 ± 4.8
Blood triglyceride (mg/dl)	176 ± 10.9	173 ± 11.1	173 ± 11.7
TC/HDL-C	6.1 ± 0.2	6.0 ± 0.2	5.9 ± 0.2
LDL-C/HDL-C	4.0 ± 0.2	4.0 ± 0.2	3.8 ± 0.2

(§ indicates significant differences)

total kilocalorie intake in the short-term follow-up period and a slight but nonsignificant increase in the kilocalorie intake in the long-term follow-up period.

There were no significant differences regarding the percentages of kilocalories from total fat, saturated fatty acid, oleic acid, or protein among the three time periods, although each of them was above the recommended values suggested by the AHA (53,58). The percentages of kilocalories as linoleic acid and carbohydrate were not significantly different among the three time periods, and each of them were below the values recommended by the AHA (53,58).

Since the percentage of kilocalories provided by linoleic acid was so low, the recommendation of approximately equal amounts of saturated fatty acid, oleic acid, and linoleic acid was not achieved by the participants. There also was a decreased consumption of dietary cholesterol across the time periods. Conversely, the mean levels of blood cholesterol and LDL-C increased. This was in contrast to the results of most other researchers who proposed that high levels of blood cholesterol and LDL-C would be a response of higher intakes of dietary cholesterol. The lack of relationship between the dietary intake and blood cholesterol response might be due to the fact that this was not a strictly controlled study; and, as such, blood lipid values were not obtained at the same time that dietary intake data was. The lack of change in the P/S ratio across time might be a point which should be stressed in further dietary education.

The mean level of LDL-C in the long-term follow-up period was found to be statistically significantly different ($P = .03$) from the mean levels in the other two periods when the LSD *t* and Duncan's multiple range tests were used. On the other hand, the comparative results from the Scheffe' test grouped the baseline and the short-term follow-up periods together in their mean values. Moreover, there were also no significant differences in LDL-C values between the short-term and the long-term follow-up periods.

Comparison Between the Cardiac and the Intervention Groups Across All Time Periods

Detailed values for each CHD related risk factors studied are presented in Table 12. The intervention group had slightly higher mean values for body weight and BMI than did the cardiac group. Although no statistical significance was found, the mean values of the total kilocalories, dietary cholesterol, and protein intakes also were higher in the intervention group. The intervention group also consumed more total fat, saturated fatty acid, linoleic acid, and oleic acid than did the cardiac group. For these latter dietary components, the differences between the two groups were significantly different ($P = .004$, $P = .001$, $P = .04$, and $P = .01$, respectively).

There were no significant differences among the two groups regarding the percentages of kilocalories as saturated fatty acid, linoleic acid, oleic acid, carbohydrate, and protein. However, the intervention group exhibited higher percent intakes of kilocalories as saturated fatty acid, linoleic acid, and oleic acid and lower percent intakes of kilocalories as carbohydrate and protein than did the cardiac group. Both groups had higher percent intakes of kilocalories as fat than the recommended value and were significantly different from one another ($P = .0002$). The percent intakes of kilocalories as linoleic acid were lower than the recommended values in both groups.

Table 12. Sample means and estimated standard errors of the means of selected parameters for the cardiac and the intervention groups across all time periods

Variable	Cardiac group	Intervention group
Body weight (lb)	187 ± 2.7	191 ± 2.8
Body mass index (W/H ²)	26.5 ± 0.31	26.8 ± 0.33
Total kilocalories intake (Kcal)	1793 ± 51	1872 ± 72
§ Total fat intake (gm) (P = .004)	67 ± 2.7	81 ± 3.9
§ Percentage of kilocalories as fat (P = .0002)	33.5	38.9
§ Saturated fat intake (gm) (P = .001)	20 ± 0.9	26 ± 1.3
Percentage of total fat as SFA	30.1	31.4
Percentage of kilocalories as SFA	10.2	12.2
§ Linoleic acid intake (gm) (P = .04)	13 ± 0.7	15 ± 1.0
Percentage of total fat as linoleic acid	18.8	19.4
Percentage of kilocalories as linoleic acid	6.3	7.3
§ Oleic acid intake (gm) (P = .01)	21 ± 1.0	26 ± 1.4
Percentage of total fat as oleic acid	30.8	31.4
Percentage of kilocalories as oleic acid	10.5	12.2
Dietary P/S ratio	.70 ± .04	.65 ± .04
Dietary cholesterol intake (mg)	319 ± 20.6	354 ± 29.1
Carbohydrate intake (gm)	223 ± 7.6	198 ± 10.8
Percentage of kilocalories as carbohydrate	49.7	43.0
Protein intake (gm)	79 ± 2.4	81 ± 3.4
Percentage of kilocalories as protein	18.1	17.4
Percentage RDA protein intake (gm)	145 ± 5	143 ± 7
§ Systolic blood pressure (mmHg) (P = .04)	127 ± 2	132 ± 2
§ Diastolic blood pressure (mmHg) (P = .0001)	79 ± 1	86 ± 1
Blood cholesterol (mg/dl)	215 ± 4.4	215 ± 4.7
Blood HDL-C (mg/dl)	36 ± 0.9	38 ± 1.0
Blood LDL-C (mg/dl)	146 ± 3.6	137 ± 3.9
Blood triglyceride (mg/dl)	165 ± 8.8	184 ± 9.5
TC/HDL-C	6.0 ± 0.2	6.1 ± 0.2
LDL-C/HDL-C	4.0 ± 0.1	3.9 ± 0.1

(§ indicates significant differences)

Nevertheless, the cardiac group appeared to have a greater level of compliance with the AHA dietary guidelines relative to percent intakes of kilocalories as total fat, oleic acid, and carbohydrate intake than did the intervention group. The fatty acid composition, as reflected by the P/S ratio, was higher in the cardiac group than in the intervention group, but the difference was not statistically significant. Both systolic and diastolic blood pressure values were significantly different between the two groups ($P = .04$ and $P = .0001$, respectively) with the higher values in the cardiac active group.

Comparison Between the Active and Inactive Statuses Across All Time Periods

The only statistically significant difference between the active and inactive status was related to the TC/HDL-C ratio. The active participants had a lower mean value than did the inactive participants ($P = .05$). Other detailed information relative to the effects of status are given in Table 13. Both active and inactive participants had higher percentages of kilocalories as total fat, saturated fatty acid, oleic acid, and protein, and lower percentages of kilocalories as linoleic acid and carbohydrate than the respective values suggested by the AHA. The active participants had a slightly lower intake of total kilocalories, total fat, saturated fatty acid, oleic acid, dietary cholesterol, carbohydrate, and protein than did the inactive participants. Compared to the inactive participants, the active participants had similar mean levels of TC and HDL-C, higher mean levels of LDL-C, and lower mean levels of TG. None of these differences were statistically significant.

Table 13. Sample means and estimated standard errors of the means of selected parameters for the active and inactive participants across all time periods

Variable	Active group	Inactive group
Body weight (lb)	189 ± 2.7	190 ± 2.9
Body mass index (W/H ²)	26.6 ± 0.31	26.7 ± 0.33
Total kilocalories intake (Kcal)	1808 ± 51	1857 ± 71
Total fat intake (gm)	72 ± 2.8	77 ± 3.9
Percentage of kilocalories as fat	35.8	36.6
Saturated fat intake (gm)	22 ± 1.0	23 ± 1.3
Percentage of total fat as SFA	30.9	30.5
Percentage of kilocalories as SFA	11.1	11.4
Linoleic acid intake (gm)	14 ± 0.7	14 ± 1.0
Percentage of total fat as linoleic acid	19.1	19.1
Percentage of kilocalories as linoleic acid	6.7	7.0
Oleic acid intake (gm)	23 ± 1.0	24 ± 1.3
Percentage of total fat as oleic acid	31.3	30.9
Percentage of kilocalories as oleic acid	11.3	11.4
Dietary P/S ratio	.66 ± .04	.68 ± .04
Dietary cholesterol intake (mg)	304 ± 21.0	370 ± 28.8
Carbohydrate intake (gm)	208 ± 7.7	214 ± 10.7
Percentage of kilocalories as carbohydrate	46.5	46.2
Protein intake (gm)	79 ± 2.4	80 ± 3.3
Percentage of kilocalories as protein	17.8	17.7
Percentage RDA protein intake (gm)	145 ± 5	143 ± 7
Systolic blood pressure (mmHg)	131 ± 2	129 ± 2
Diastolic blood pressure (mmHg)	84 ± 1	82 ± 1
Blood cholesterol (mg/dl)	215 ± 4.3	215 ± 4.8
Blood HDL-C (mg/dl)	38 ± 0.9	37 ± 1.0
Blood LDL-C (mg/dl)	145 ± 3.5	138 ± 4.0
Blood triglyceride (mg/dl)	162 ± 8.6	187 ± 9.7
§ TC/HDL-C (P = .05)	5.7 ± 0.2	6.3 ± 0.2
LDL-C/HDL-C	3.8 ± 0.1	4.0 ± 0.1

(§ indicates significant differences)

Multivariate Separation of Subjects

The Cardiac Therapy and Intervention Program has established criteria for classifying clients when they first join the program. Namely, participants in the cardiac group either have had at least one cardiovascular event, have exhibited a combination of cardiac risk factors, or have demonstrated abnormal EKG, blood pressure, angina, and dyspnea responses to graded exercise testing. The intervention group was described as those who were without any of the above conditions and who had no known disease and negative graded exercise test results.

The accuracy of this classification system was explored through the use of discriminant analysis. Separation addresses the question of differences between two or more group means, while classification is referred to as using information to assign a new client to one of the groups. These two terms are usually used interchangeably. The information regarding each individual relative to CHD related risk factors or any cardiovascular event was reviewed from each individual's medical record in the baseline period (Table 5). The variables considered in this study were as follows: 1) age at the baseline period; 2) body weight at the baseline period; 3) the existence of any angina pectoris (AP), myocardial infarction (MI), coronary bypass surgery (CBS), and the major kinds of coronary heart disease (CHD); 4) the experience of any of the following events which might increase the chance of any kind of cardiovascular events in later life: family history of early heart disease (EARLY), abnormal EKG (EKG), angina type of chest pain (PAIN), hypertension (HBP), hypercholesterolemia (HCHOL), hypertriglyceridemia (HTG), and diabetes mellitus (DM); 5) taking cholesterol-lowering (DRUG) or anti-hypertensive (ANTI) medications; and 6) smoking status (SMOKE). Canonical variates were created using these factors. A canonical variate is a linear combination of the variables, chosen to maximize separation. Figure 1 represents the canonical scatter plot of the four subgroups using the first two canonical variates as the axes. In this plot, all variables listed above were used.

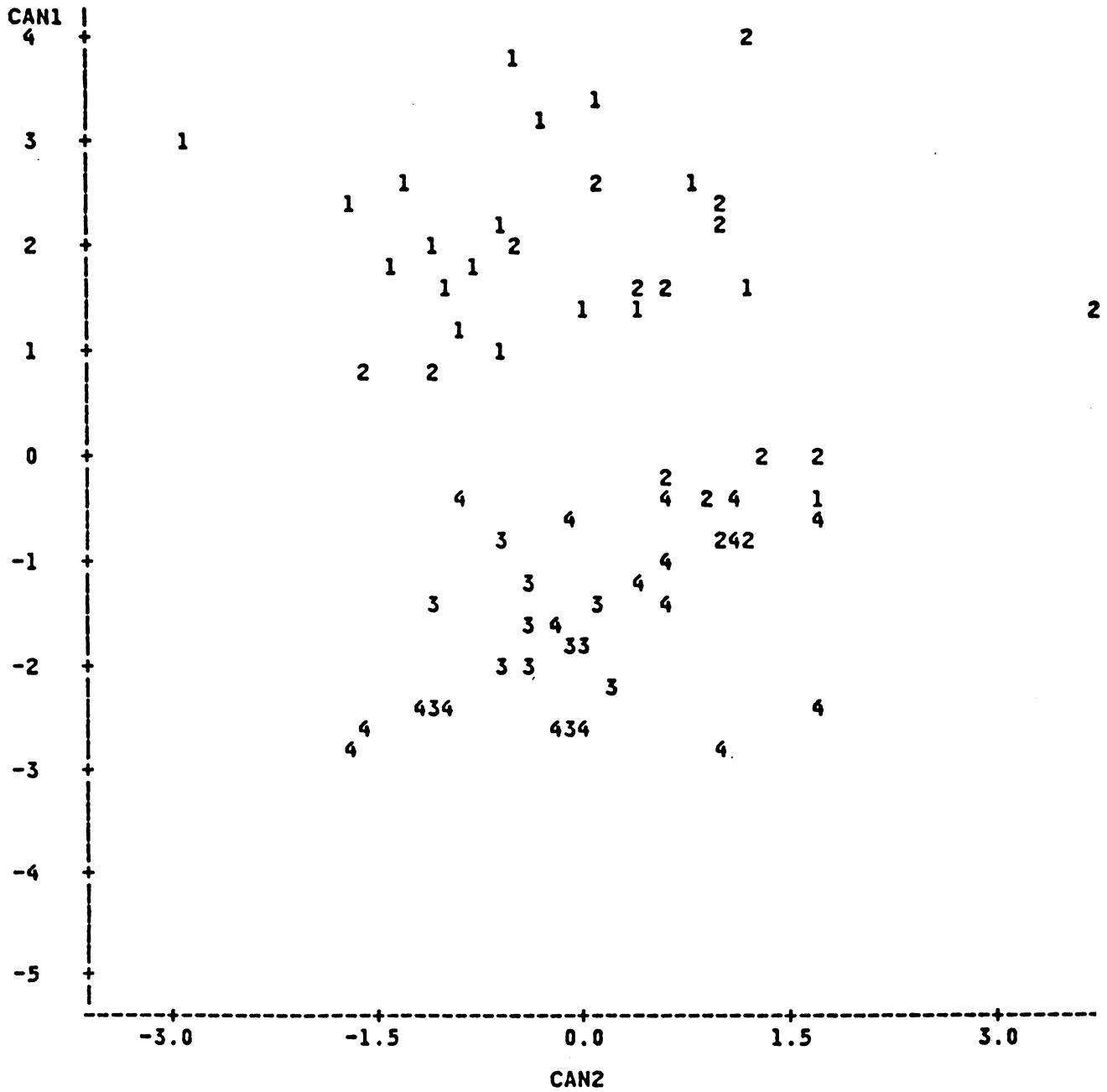


Figure 1. The scatter plot of four subgroups, using the first two canonical variates; 1 = CI group, 2 = CA group, 3 = II group, and 4 = IA group (all variables are used)

On the basis of the information listed in Table 5 relative to selected risk factors for CHD in the baseline period, the expected outcome was that the cardiac inactive group (represented by number 1) with the highest number of risk factors for CHD should have gathered on the top left corner. The 2s and 3s, which represented the CA group and the II group, respectively, should have gathered at middle section of the plot. The IA group (4s) was the group with fewer cardiovascular risk factors and should have had the least probability of experiencing any type of cardiovascular event. Thus, the 4s were expected to be found on the bottom right corner. Figure 1 shows that the groups overlapped. It is not easy to draw straight lines to separate the four groups. Stepwise discriminant analysis was used to test if all these variables listed above were equally influential for classifying patients into either the cardiac group or the intervention group.

Figure 2 represents the canonical scatter plot of four subgroups using the reduced subset of variables that could contain almost as much information as the original collection which contributed to the cardiovascular events. The variables were selected at a significance level of .05. The following variables were found to be useful relative to separation of the two groups: age, body weight, the existence of angina pectoris, coronary bypass surgery, abnormal EKG, hypertension, and the use of antihypertension or cholesterol lowering medications. Among this reduced subset of variables, EKG and AGE were the most influential variables for canonical 1; while AGE and DRUG were the most influential variables for canonical 2. The higher values of canonical 1 represent people who were older, had abnormal EKG responses, and were taking anti-hypertensive medication; while the higher the values of canonical 2 represent people were older without abnormal EKG responses. Consequently, people who are older and have abnormal EKG responses tended to be assigned to the cardiac group and people who are older but without any abnormal EKG responses tended to be assigned to the intervention group.

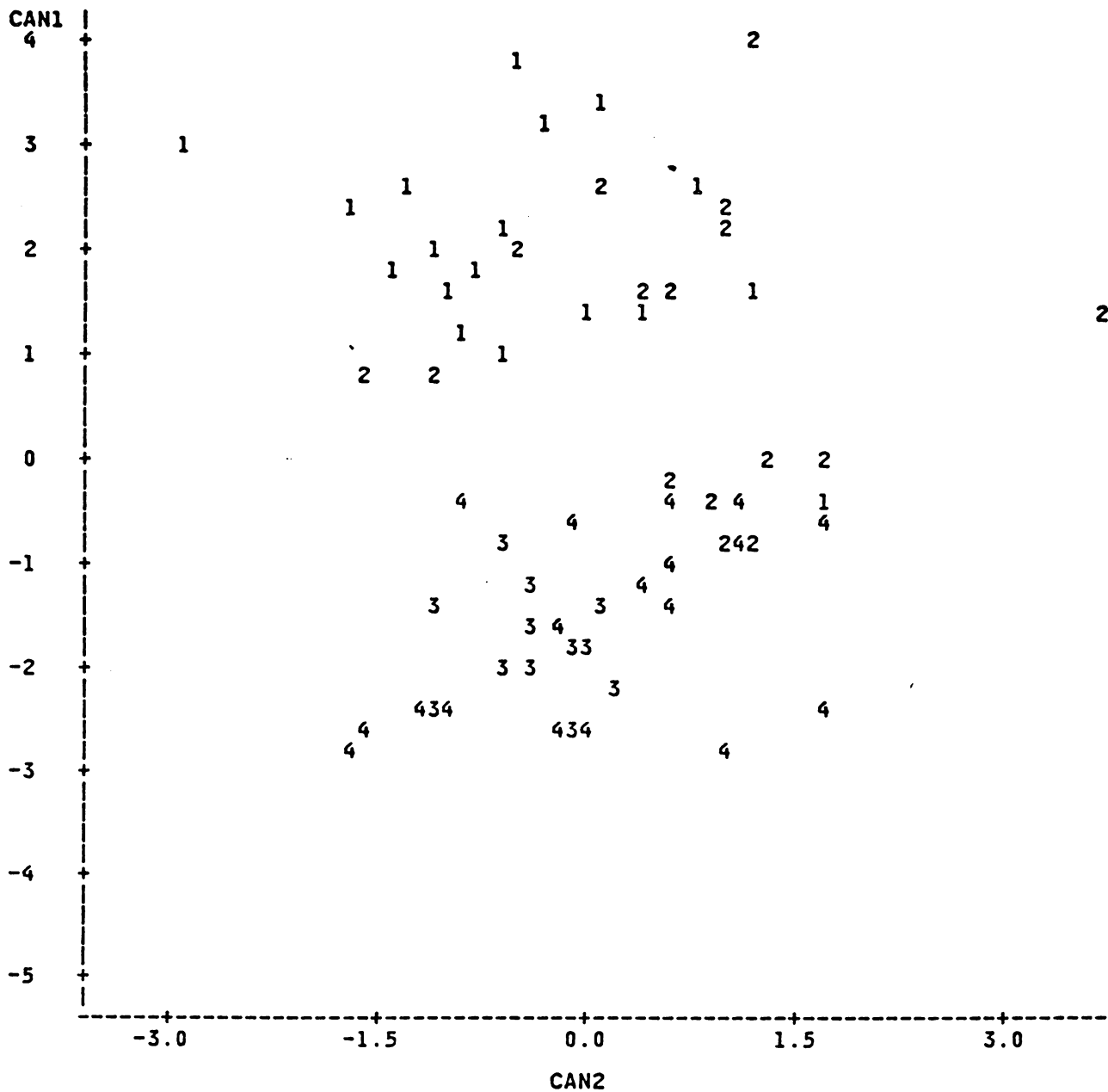


Figure 2. The scatter plot of four subgroups using variables from improved model; 1 = CI group, 2 = CA group, 3 = II group, and 4 = IA group.

Conclusion

The focus of this study was twofold: 1) to investigate the effect of a cardiac rehabilitation and intervention program relative to risk reduction in the participants and 2) to compare those participants who continued to be active in the program with those who discontinued participation relative to status on selected risk factors. The expected results were that the lowest mean values of body weight, BMI, overall dietary components, dietary components, blood pressures, and blood lipid profiles would be found in either the short-term follow-up period or the long-term follow-up period for those who continued to be active in the program since they received regular nutritional education intervention and exercise three times a week from the program. But, in some cases, the results were different from those that were expected. However, it is important to note that individuals who were in the program across the three time periods may not necessarily have had a physically active life style, since there was no control for activity levels of either active or inactive participants. This might influence the finding regarding active or inactive status. However, despite this possible confounding factor, there are several common points regarding dietary patterns, blood pressure, and blood lipid profile among four subgroups.

The lowest mean intakes of most of the dietary components and the blood lipid levels were seen in the short-term follow-up period, and then showed a slight increase in the long-term follow-up period. These results suggest that effective lowering of cardiac risk factors in CHD might take at least several months and that even short-term participation in this program can provide some benefit to those at high risk for CHD.

The results of the comparisons among the four subgroups across all three time periods showed that none of the groups had ever achieved the following dietary guidelines recommended by the AHA:

- 30 percent of the total kilocalories as fat--- Among the four subgroups, the IA and the II groups had a higher percent of fat intake constituting the total kilocalories than did the CA and the CI groups. Among all groups, the percentage of total kilocalories as fat remained virtually unchanged across the three time periods. The intervention group had a significantly higher total fat intake and percentage of kilocalories as fat than did the cardiac group. The inactive participants had a higher but nonsignificantly different total fat intake and percentage of kilocalories as fat than did the active participants.
- 55 percent of kilocalories as carbohydrate---The CA and the CI groups had a higher carbohydrate intake and percentage of kilocalories provided by carbohydrate than the IA and the II groups. (216 gm and 231 gm vs. 200 gm and 197 gm and 49.6 percent and 49.7 percent vs. 43.3 percent and 42.7 percent, respectively). Across all groups, the lowest amount of carbohydrate consumed was found in the short-term follow-up period, and the highest one in the long-term follow-up period, yet this was still below the recommended value. The actual carbohydrate intake was higher for inactive participants (208 gm for the active participants and 214 gm for the inactive participants), while the percentages of kilocalories as carbohydrate were the same between the two statuses of participants (46.5 percent vs. 46.2 percent).
- 15 percent of kilocalories as protein---The actual protein intakes among the four subgroups were similar, while the CA and the CI groups had a slightly higher percentage of kilocalories provided by protein than did the IA and the II groups (18.0 percent and 18.2 percent vs. 17.6 percent and 17.2 percent, respectively). The protein intake was lowest in the short-term follow-up period; however, the percentage of kilocalories as protein was highest in this period. The main reason for this was that participants dramatically reduced their total kilocalorie intakes in this period. Since the intervention group had both a higher total kilocalorie intake and a higher protein intake, the percentage of kilocalories as protein was lower than in the cardiac group (18.1 percent for the cardiac group and 17.4 percent for the intervention group). There were minimal differences between active and inactive participants regarding protein intake and percentage of kilocalories as protein (17.8 percent vs 17.7 percent).

- The fat should be divided approximately equally among saturated, monounsaturated, and polyunsaturated fatty acids, i.e., each should contribute about 10 percent of the total kilocalories---since the intakes of linoleic acid were so low (at any combination of comparisons) compared to the intakes of saturated and oleic acids, it was impossible to achieve the goal that each should contribute equally to the total fat consumption. Consequently, the P/S ratio also was below the recommended value of 1:1.
- Cholesterol intake should be below 300 mg per day for the general public, below 200-250 mg per day and even 100-150 mg per day for moderate and high risk populations, respectively--- The higher than recommended value of dietary cholesterol intakes for each combination of comparisons may be another point which should be improved in the daily dietary pattern of the participants.

Results of this study also suggest the need for a total integrated evaluation regarding modifiable environmental risk factors in the program participants. The periodic evaluation regarding the dietary intake, body weight, exercise intensity, and blood lipid response is necessary. More nutritional education is needed regarding the choosing of low cholesterol containing foods and using foods higher in polyunsaturated fatty acids rather than using food containing high saturated fatty acid or hydrogenated fatty acid to achieve the P/S ratio of 1:1. At the same time the protein intake should be decreased and the carbohydrate intake, particularly the complex carbohydrates, should be increased.

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Appendix A. Distributions of Blood Lipid Levels of American White Male from Lipid Research Clinic Population Studies Data Book

*Plasma Total Cholesterol (mg/dl) (population distribution)**

Age (years)	White males						
	Percentiles						
	5	10	25	50	75	90	95
0-4
5-9	125	131	141	153	166	183	199
10-14	124	131	144	160	173	188	202
15-19	119	123	136	152	168	183	191
20-24	118	126	142	159	179	197	212
25-29	120	137	154	176	199	223	234
30-34	142	152	171	190	213	237	258
35-39	147	157	176	195	222	248	267
40-44	150	160	179	204	229	251	260
45-49	163	171	188	210	235	258	273
50-54	157	168	189	211	237	263	274
55-59	161	172	188	214	236	260	280
60-74	163	170	191	215	237	262	287
65-69	166	174	192	213	250	275	288
70+	144	160	185	214	236	233	265

*Plasma HDL-Cholesterol (mg/dl) (population distribution)**

Age (years)	White males						
	Percentiles						
	5	10	25	50	75	90	95
0-4
5-9	38	42	49	54	63	70	74
10-14	37	40	46	55	61	71	74
15-19	30	34	39	46	52	59	63
20-24	30	32	38	45	51	57	63
25-29	31	32	37	44	50	58	63
30-34	28	32	38	45	52	59	63
35-39	29	31	36	43	49	58	62
40-44	27	31	36	43	51	60	67
45-49	30	33	38	45	52	60	64
50-54	28	31	36	44	51	58	63
55-59	28	31	38	46	55	64	71
60-64	30	34	41	49	61	69	74
65-69	30	33	39	49	62	74	78
70+	31	33	40	48	56	70	75

(Continued)

*Plasma LDL-Cholesterol (mg/dl)(population distribution)**

Age (years)	White males						
	Percentiles						
	5	10	25	50	75	90	95
0-4	-	-	-	-	-	-	-
5-9	63	69	80	90	103	117	129
10-14	64	72	81	94	109	122	132
15-19	62	68	80	93	109	123	130
20-24	66	73	85	101	118	138	147
25-29	70	75	96	116	138	157	165
30-34	78	88	107	124	144	166	185
35-39	81	92	110	131	154	176	189
40-44	87	98	115	135	157	173	186
45-49	98	106	120	141	163	186	202
50-54	89	102	118	143	162	185	197
55-59	88	103	123	145	168	191	203
60-64	83	106	121	143	165	188	210
65-69	98	104	125	146	170	199	210
70+	88	100	119	142	164	182	186

Plasma Triglycerides (mg/dl)

Age (years)	White males						
	Percentiles						
	5	10	25	50	75	90	95
0-4	-	-	-	-	-	-	-
5-9	28	34	39	48	58	70	85
10-14	33	37	46	58	74	94	111
15-19	38	43	53	68	88	125	143
20-24	44	50	61	78	107	146	165
25-29	45	51	67	88	120	171	204
30-34	46	57	76	102	142	214	253
35-39	52	58	80	109	167	250	316
40-44	56	69	89	123	174	252	318
45-49	56	65	89	123	174	252	318
50-59	63	75	94	128	178	244	313
55-59	60	70	85	117	167	210	261
60-64	56	65	84	111	150	193	240
65-69	54	61	78	108	164	227	256
70+	63	71	87	115	152	202	239

Source :

Lipid Research Clinics Coronary Primary Prevention Trial Results. I. Reduction in incidence of coronary heart disease. II. The relationship of reduction in incidence of coronary heart disease to cholesterol lowering. J. Am. Med. Assoc. 251: 351, 1984.

Appendix B. Risk Values Related to Blood Cholesterol for American Males

<u>Level of Risk</u>	<u>Total Cholesterol to HDL-C Ratio</u>
1/2 Average	3.43
Average	4.97
2 X Average	9.55
3 X Average	23.39

<u>Level of Risk</u>	<u>LDL-C to HDL-C Ratio</u>
1/2 Average	1.00
Average	3.55
2 X Average	6.25
3 X Average	7.99

Source:

Kannel W.B., Castelli W.P., and Gordon T. Cholesterol in the prediction of atherosclerotic disease. *Annals of Int. Med.* 90: 85, 1979.

Appendix C. Letter

September 12, 1986

I am a graduate student in nutrition at Virginia Tech. For my thesis project, I am comparing currently active and formerly active participants in the Cardiac Therapy and Intervention Program at Virginia Tech with respect to changes in risk factors related to cardiovascular disease. Specifically, I would like to collect information about changes in blood lipid levels, body weight, exercise patterns, smoking status, blood pressure and use of cholesterol lowering or antihypertensive medications.

I would appreciate your help with this study. This would involve keeping a 3-day food record, completing a short questionnaire, and having 15 mls (about 1/2 ounce) of blood drawn by a registered nurse. Also, height, weight and a blood pressure reading will be obtained. It will take approximately thirty minutes to collect this information. Appointments will be arranged during the next six weeks. An overnight fast of 12 hours will be necessary prior to the blood draw. There is no cost for participating in this study. The results of your blood lipid profile will be sent to you for your own use. All information will be kept strictly confidential.

I sincerely hope that you can participate in this study. Please return the enclosed postcard as soon as possible. The results will be helpful to the Cardiac Center staff regarding the exercise and nutritional counseling aspects of the programs. If you agree to participate, I will be contacting you with more detailed information. If you have any questions, please feel free to call me at 961-3343, or Mrs. Ellen Coale at 961-7277.

Thank you for your help and cooperation. I appreciate that so much.

Sincerely,

Ren-Chian Chu
Graduate Student-Human Nutrition and Foods
Dr. Mary Ann Novascone
Academic Advisor (703) 961-5778

Encl: Post card

Appendix D. Consent Form

Consent for Participation in Cardiovascular Risk Reduction Study

I have received a written explanation of this study and I understand the following:

Records presently on file with the Cardiac and Intervention Program will be reviewed for the following laboratory values: total cholesterol, low density lipoprotein cholesterol, high density lipoprotein cholesterol, and triglycerides. Other data which will be reviewed include height, weight, and blood pressure values and past food records. Approximately 15 mls (1/2 ounce) of blood will be drawn by a registered nurse. This procedure requires an overnight fast of 12 hours. Also, current height, weight, and blood pressure values will be obtained. Participants in the study will complete one 3-day food record (one non-working day and two working days) and a short questionnaire addressing smoking habits and exercise patterns.

The identity of the participants will be held confidential in all reports of this research. Participants are at essentially no risk by participating in the project. No compensation is available if injury is suffered as a result of this research. However, information about their health status can be gained by the participants. There will be no monetary cost for participation in the study and neither will there be any monetary reward. Consent for participation may be withdrawn at any time. Questions regarding this project will be answered by the investigator.

I understand the above, and agree to participate in this study.

Date

Signature

Investigators:

Ren-Chian Chu (961- 3343)---Graduate student/HNF

Dr. Mary Ann Novascone (961- 5778)---Academic advisor/HNF

Ellen Coale, M.S., R.D. (961-7277)---Cardiac Therapy and Intervention Center

Appendix E. Guidelines for Recording Food

Consumed

Food consumed in your daily diet is an important factor in this study. Please complete the enclosed 3-day dietary record (one non-working day and two working days).

1. Read through the attached sample menu first to become familiar with the methods you will follow.
2. Record as accurate information as possible regarding the type and amount of food items you have eaten. Write down what you have eaten immediately after it has been consumed so as not to forget what was eaten.
3. Be sure to record the hidden or extra foods which accompany the main dish, such as sugar, tartar sauce, gravy, butter, oil, or topping on desserts.
4. Do not change your food intake habits when you are recording. It is important that you consistently follow the usual food pattern you are used to.
5. Be sure to give the methods of food preparation (e.g., broiled, fried, baked) and types of food purchased (e.g., low fat or whole milk).
6. Please follow these guidelines when recording amounts eaten:
 - Milk, juice, and other liquids: record in terms of measuring cup or ozs (1 measuring cup = 8 ounces).
 - Butter: record as level teaspoon (1 average pat = 1 level teaspoon)

- Sugar: record as level teaspoon (1 packet = 1 teaspoon)
- Egg: please note whether whole egg, white or yolk only, and egg substitute. Also note size of egg and preparation method.
- Cereal and vegetables: record in terms of measuring cup or tablespoons (1/2 measuring cup = 8 level tablespoons). Use level tablespoons when measuring food.
- Fruit: if whole, give size (small, medium, large). If sliced or canned, express in terms of measuring cup or tablespoons as above.
- Meat: record as ounces, if possible. Otherwise, estimate size as accurately as possible (see attached diagrams).
- Other foods: record as accurately as possible in terms of ordinary household measurements.

ONE DAY FOOD RECORD (SAMPLE)

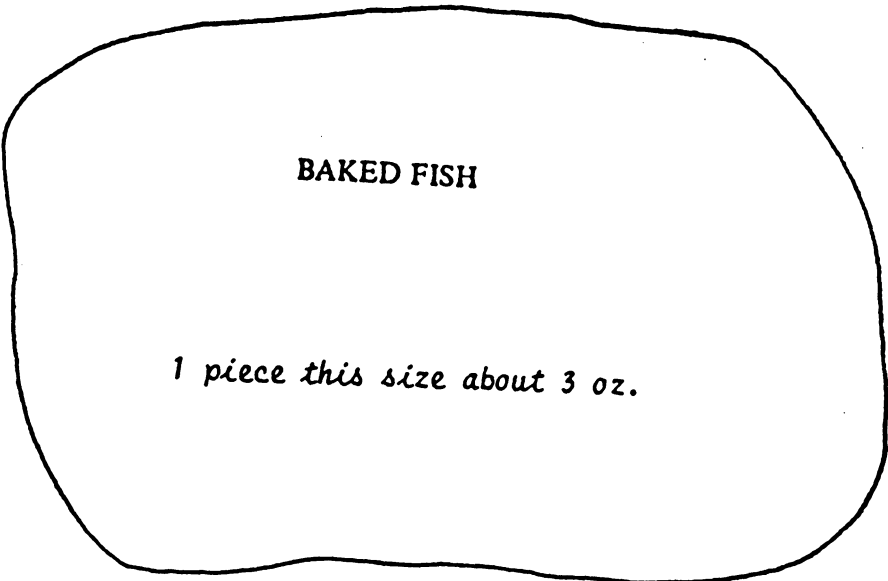
Name _____ Date _____ Code no. _____

	Amount of food eaten (cup,tbsp,oz)	Description of food consumed	Description of food purchased or prepared
Breakfast	1/4 medium 1 large 1 cup 1 tsp 1/2 cup	Canteloupe Egg Oatmeal with cinnamon Margarine Milk-1 percent low fat	Soft cooked Soft
Lunch	1 1 oz 1 oz 2 slices 2 tsps 1 1/4 cup 1 tbsp 1/2 cup	Ham and cheese sandwich contains: lean ham natural Swiss cheese rye bread mayonnaise-type salad dressing Tossed salad--green onion lettuce,tomato,carrots, Italian dressing Sliced peaches	 Canned in syrup
Dinner	4 oz 1 medium 2 tsps 1/2 cup 1 small 6 oz 5	Roast loin of pork Potato Sour cream Green peas(not buttered) Roll Vanilla yogurt mixed with strawberries	Roast, lean only Baked Frozen Whole wheat Low fat Fresh,unsweetened
Snack	1 whole 1 tbsp	English muffin Marmalade	

ONE DAY FOOD RECORD

Name _____ Date _____ Code no. _____

	Amount of food eaten (cup,tbsp,oz)	Description of food consumed	Description of food purchased or prepared
Breakfast			
Lunch			
Dinner			
Snack			

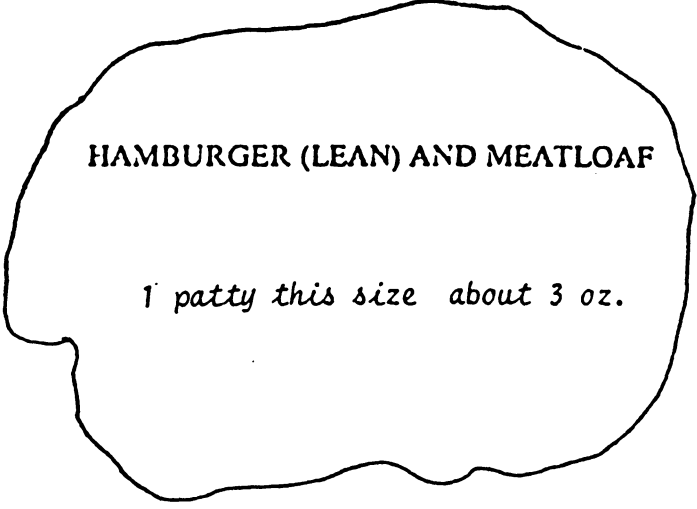


BAKED FISH

1 piece this size about 3 oz.



**This
Thick**



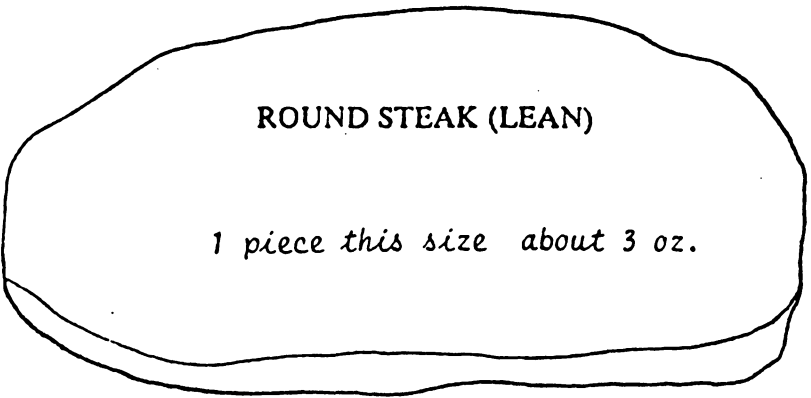
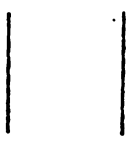
HAMBURGER (LEAN) AND MEATLOAF

1 patty this size about 3 oz.

**This
Thick**

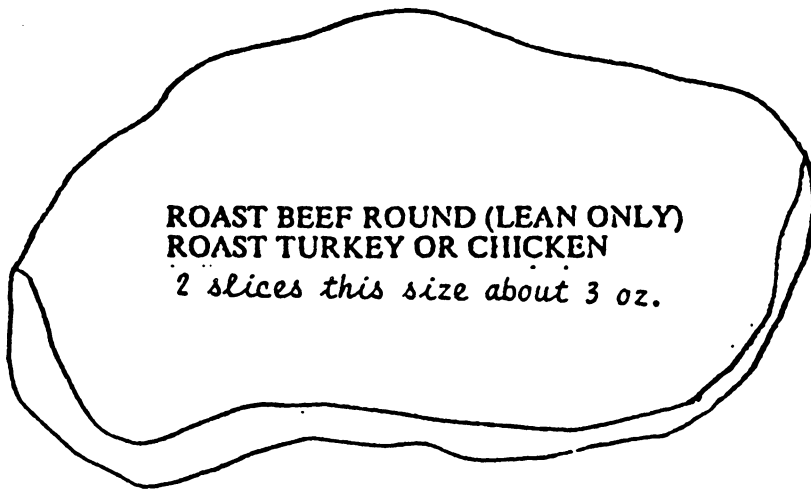


This Thick



ROUND STEAK (LEAN)

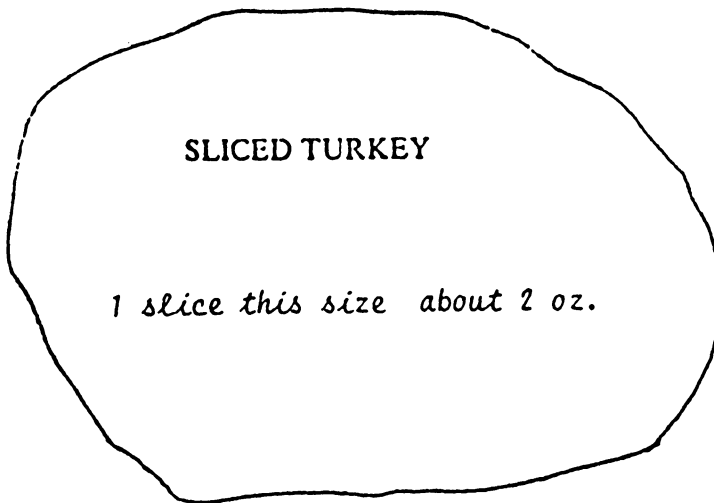
1 piece this size about 3 oz.



**ROAST BEEF ROUND (LEAN ONLY)
ROAST TURKEY OR CHICKEN**

2 slices this size about 3 oz.

**This
Thick**

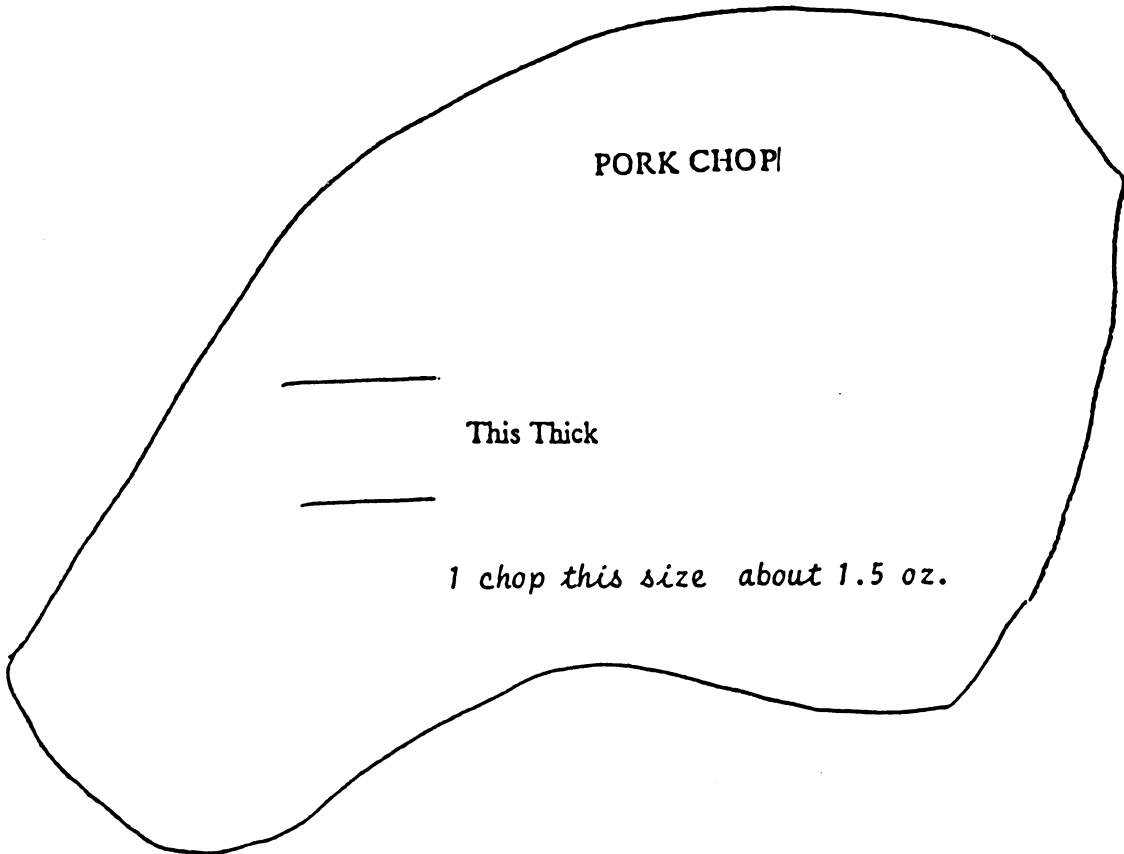


SLICED TURKEY

1 slice this size about 2 oz.

This Thick

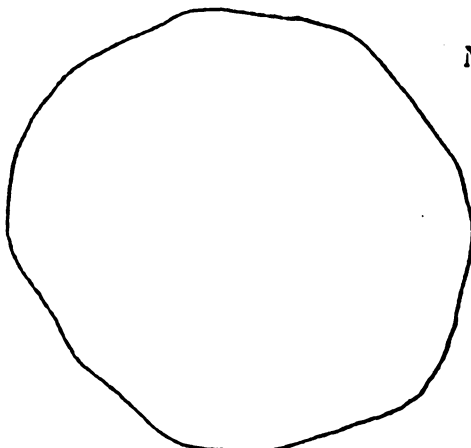




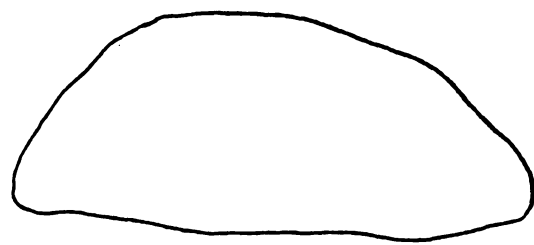
PORK CHOP

This Thick

1 chop this size about 1.5 oz.



MEAT BALLS



This Thick

2 meat balls this size about 3 oz.

Appendix F. Questionnaire of Medical History

Name _____ Age _____ Code _____

1. Have you ever had any of the following cardiovascular events? (Check *all* that apply)

Angina pectoris _____

Myocardial infarction _____

Ischemic heart disease _____

Coronary bypass surgery _____

Abnormal EKG _____

Other heart related problems (Please specify) _____

2. Have you ever been diagnosed as having

high blood levels of cholesterol (above 220 mg/dl)? Yes _____ No _____ Not sure _____

or of triglyceride (above 160 mg/dl)? Yes _____ No _____ Not sure _____

3. Are you taking any cholesterol-lowering medication? Yes _____ No _____

If yes, what kind of medication do you currently use? _____

4. Are you taking any antihypertensive medication? Yes _____ No _____

If yes, what kind of medication do you currently use? _____

5. What are your smoking habits? Smoker _____ Ex-smoker _____ Non-smoker _____

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