THE EFFECTS OF DIET AND EXERCISE ON SERUM LIPOPROTEINS OF ADULT WOMEN

by

Cheryl Lynn McClure

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ABSTRACT

While aerobic exercise has been associated with decreased triglycerides and increased HDL-C in studies performed in men, less consistent results have been found in the few studies performed in women. This experiment was designed to investigate the effects of aerobic exercise and dietary modification on serum lipid and lipoprotein levels in adult sedentary women. The eight experimental subjects (aged 27 to 42 years) were counselled to consume a low-fat, low cholesterol diet and attended 3-45 minute aerobic exercise classes per week for 24 weeks. The ten control subjects (aged 39 to 47 years) were encouraged to maintain their regular diet and exercise habits. All subjects underwent blood lipid analysis and fitness and dietary assessments. An analysis of test results revealed no significant differences between the exercise and control groups. Within the exercise group, levels of serum triglycerides, total cholesterol, and LDL-C did not change significantly from baseline after 24 weeks. However, transient decreases in these variables were detected at 6 weeks, and were found to be significant for total cholesterol and LDL-C only ($p<0.05$). A transitory increase in HDL-C occurred at 6 weeks ($p<0.05$), followed by a

significant drop to below baseline at 24 weeks ($p<0.01$). A significant decrease in HDL-C was also observed in the control group, suggesting that seasonal variation might be responsible for this change. Aerobic fitness did not improve in the experimental group and may relate to the intensity and frequency of the exercise program, as well as the initial fitness level of the subjects. Body composition also remained stable for this group. No significant change in diet composition of the exercise subjects was observed after 24 weeks. The results of this study suggest that 24 weeks of dietary modification and aerobic exercise does not produce positive changes in the lipoprotein profiles of adult women.

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REVIEW OF LITERATURE

1.1 INTRODUCTION

Coronary heart disease (CHD) is the leading cause of death in North America today. The disease is characterized by atherosclerosis, of which the principal feature is the deposition of cholesterol esters in the inner walls of the arteries (Miller, 1978). Evidence from both epidemiological and experimental studies demonstrates that a relationship exists between serum lipid and lipoprotein levels and the development of CHD (Stamler et al, 1986). Specifically, increased serum triglyceride (TG) and low density lipoprotein (LDL) levels are associated with an increased risk of CHD (Witztum and Schonfeld, 1979; Thompson and Bortz, 1978). Elevated levels of high density lipoprotein (HDL) are correlated with a reduced CHD risk and may protect against development of the disease (The Lipid Research Clinics Study, 1984; Miller, 1978; Wallentin and Sundin, 1985). Thus, LDL and HDL appear to act antagonistically in relation to coronary heart disease.

There are a variety of factors, both environmental and genetic, which influence the levels of lipoproteins in man. Cigarette smoking has been associated with depressed levels

of HDL-C (Criqui et al, 1980; Stamford et al, 1984a), whereas moderate alcohol consumption (Stamford et al, 1984b; Crouse and Grundy, 1984) and chronic physical exercise (Huttunen et al, 1979; Haskell, 1984) have been associated with elevations in HDL-C. Physical exercise has also been correlated with decreased VLDL-C, LDL-C and serum TG concentrations (Lopez et al, 1974; Huttunen et al, 1979; Nye et al, 1981). The observed alterations in the lipoprotein levels of exercising individuals have been deemed favorable, as increases in HDL-C and decreases in VLDL-C, LDL-C, and TG have previously been associated with a reduced risk of CHD development (Miller, 1978; Gordon et al, 1977; The Lipid Research Clinics Study, 1984). However, the mechanism by which exercise effects these lipoprotein changes is currently unknown. Several dietary factors have also been linked to changes in serum lipoprotein concentrations. Research has shown that the ingestion of saturated fats leads to an elevation of serum cholesterol whereas polyunsaturated fats produce the opposite effect (Shepherd et al, 1978). The effect of both dietary cholesterol and dietary carbohydrate on serum lipoprotein levels has also provided a focus for numerous investigations, which have yielded conflicting results (Tan et al, 1980; Coulston et al, 1983).

It is the purpose of this research to investigate the effects of dietary modification and aerobic exercise on serum lipoprotein levels of adult women. Favorable changes could reduce the risk of development of coronary heart disease.

1.2 LIPOPROTEIN METABOLISM

The plasma lipoproteins are the transport vehicles for both endogenously synthesized and exogenous (dietary) lipids (Haskell, 1984). All lipoproteins contain cholesterol, triglyceride, phospholipid, and protein. They are classified, on the basis of their size and density, into four categories: chylomicrons, very low density lipoproteins (VLDL), low density lipoproteins (LDL), and high density lipoproteins (HDL).

Chylomicrons originate in the intestine, and are the carriers of dietary TG from the gastrointestinal tract (Stanbury et al, 1983). Triglycerides constitute more than 85% of the chylomicron mass (Haskell, 1984), and are hydrolyzed to glycerol and free fatty acids by the lipoprotein lipase (LPL) enzyme. The main receptors sites for the hydrolysis products are located in adipose tissue and skeletal muscle. The chylomicron remnants are then released into the circulation and taken up by the liver for further metabolism (Haskell, 1984).

VLDL is responsible for the transport of endogenous TG, which is formed in the liver from dietary carbohydrate and fat. VLDL originates in the liver, and to a lesser extent in the intestine (Haskell, 1984). The removal of VLDL is similar to that of chylomicrons. The very low density particles are catabolized to LDL via an intermediate density lipoprotein (Stanbury et al, 1983). Since most TG in the plasma is carried by VLDL, total plasma TG levels closely parallel those of VLDL-TG (Haskell, 1984).

LDL, the metabolic end product of VLDL, functions in the delivery of cholesterol to the peripheral cells (Stanbury et al, 1983). Because LDL transports the majority of cholesterol in the plasma, its concentration correlates closely with that of total plasma cholesterol (Haskell, 1984).

The physiologic role of HDL is primarily that of cholesterol transport from the peripheral tissues to the liver, for catabolism and excretion (Witztum and Schonfeld, 1979; Haskell, 1984). It has also been suggested that HDL interferes with the cellular uptake of LDL, and in this way impedes the atherogenic process (Carew et al, 1976; Lewis, 1983).

There are three main sources of HDL (Haskell, 1984). The liver and intestine secrete HDL in the form of disc-shaped particles, or "nascent" HDL. The TG-rich

lipoproteins (i.e. chylomicrons and VLDL) also act as a source of HDL (Durrington, 1980). These "nascent" discs acquire free cholesterol readily from cell membranes (Witztum and Schonfeld, 1979; Haskell, 1984). The enzyme lecithin cholesterol acyl transferase (LCAT) then esterifies the free cholesterol to cholesterol esters, which enter the hydrophobic core of the "nascent" HDL, converting the disc-like structure to a sphere (Witztum and Schonfeld, 1979; Haskell, 1984). The major site of HDL degradation is believed to be the liver (Haskell, 1984), via the hepatic lipase enzyme.

Two subfractions of HDL have been identified, HDL_3, and the less dense HDL_2 (Wallentin and Sundin, 1985). Evidence suggests that the protection from CHD, conferred by HDL, resides primarily in the HDL_2 subfraction (Shepherd et al, 1980).

1.3 DIET AND SERUM LIPOPROTEINS

The relationship of diet to serum lipoprotein metabolism continues to attract a good deal of investigative attention and stimulates an even greater amount of controversy (Wood et al, 1985; Fisher et al, 1983). Accumulated data now document that dietary factors do indeed affect levels of lipoproteins and their metabolism (Goldberg and Schonfeld, 1985; Pietinen and Huttunen, 1987; Pyorala,

1987). The dietary factors of most interest to researchers include cholesterol, fat and carbohydrate.

1.3.1 Dietary Cholesterol

Because of the strong positive correlation between serum cholesterol levels and the risk of CHD (The Lipid Research Clinics Study, 1984; Kuusi et al, 1985), numerous metabolic studies have investigated the possible effects of dietary cholesterol on serum cholesterol levels in humans (Tan et al, 1980; Flaim et al, 1981; Beynen and Katan, 1985). Results of several studies suggest that the cholesterol levels of the individual lipoproteins (HDL, LDL and VLDL) are better risk predictors of CHD than is the level of total serum cholesterol alone (Gordon et al, 1977; Mahley et al, 1978). Increased LDL levels are related to an increased risk of CHD (Castelli et al, 1977), while elevated HDL may protect against development of the disease (Gordon et al, 1977). Thus, LDL and HDL appear to act antagonistically with respect to the progression of CHD.

Tan and co-workers (1980) studied the effects of cholesterol feeding on serum lipoprotein levels in humans. Six normolipidemic subjects consumed a diet of mixed, natural foods over a 4-week period. During the first week, the diet provided 100 mg cholesterol/day, with a P/S ratio of 1.6. For the next 3 weeks, the cholesterol intake was

increased to 1028 mg/day, accompanied by a reduced P/S ratio of 0.14. When compared with the low cholesterol diet, the high cholesterol diet resulted in a 23% increase in serum cholesterol by raising the cholesterol concentration in VLDL to 59%, LDL to 15%, and HDL to 30%. A problem exists in this study in that the increase in dietary cholesterol was accompanied by an increase in saturated fat intake. Thus, the independent effect of dietary cholesterol on serum cholesterol could not be determined.

More recently, Beynen and Katan (1985) investigated the effects of increased dietary cholesterol on serum lipoproteins in 6 healthy adults. The subjects consumed a low cholesterol diet (200 mg/day) for 10 days, and then, added 6 egg yolks to their diet daily to increase cholesterol to 1800 mg/day for another 10 days. Unlike Tan's study, the levels of saturated and polyunsaturated fatty acids in this diet remained constant (P/S = 0.5). With cholesterol feeding, the mean serum total cholesterol level increased by 13%. A 21% rise in LDL-C accounted for the bulk of this increase. This rise in total cholesterol and LDL-C was ameliorated by a concomitant 36% rise in HDL-C.

In contrast to the results of both Tan and co-workers (1980), and, Beynen and Katan (1985), Flaim and associates (1981) failed to demonstrate any dietary cholesterol effect

on plasma cholesterol levels. Twenty-three healthy males were recruited for the 4-week study. The subjects were divided into 2 groups. The first group consumed a "common American diet" containing 400 mg cholesterol/day from a non-egg source. The second group consumed a similar diet with an added 1000 mg cholesterol/day as 4 whole eggs. Differences in total protein, carbohydrate, fat, and the P/S ratio (0.5) between the 2 groups were kept to a minimum. Data from this study showed no significant differences in plasma total cholesterol, HDL-C, LDL-C, and VLDL-C levels between the groups. Within the second group, however, responses to the high cholesterol diet varied. This observation of interindividual variation in plasma cholesterol response to changes in dietary cholesterol intake has been reported by other researchers (Flynn et al, 1979; Chenoweth et al, 1981).

Epidemiological studies have established that elevated plasma total and LDL cholesterol play a major role in the development of CHD (Gordon et al, 1977). Furthermore, the reduction of total cholesterol via the lowering of LDL-C can reduce the incidence of CHD morbidity and mortality (The Lipid Research Clinics Study, 1984; Pyorala, 1987). As well, a consistent negative correlation has been found between the concentration of HDL-C and the risk of CHD in studies within populations (Pietinen and Huttunen, 1987). Despite extensive research, the role of dietary cholesterol

in the alteration of these lipoprotein cholesterol levels, and thus in the development of CHD, remains a highly controversial issue, and a general consensus has yet to be reached (Conner and Conner, 1986).

1.3.2 Dietary Fat

The influence of the type and amount of dietary fat on serum cholesterol levels has been a focus of research in recent years. A number of human studies have shown that ingestion of saturated fat leads to an elevation of plasma cholesterol, whereas polyunsaturated fat produces the opposite effect (Shepherd et al, 1980; Schaefer et al, 1981; Ehnholm et al, 1984). Although the mechanism responsible for these changes remains obscure, the key seems to lie in the alteration of the ratio of polyunsaturates to saturates (P/S ratio).

In 1978, Shepherd and co-workers investigated the effects of dietary polyunsaturated and saturated fat on serum total cholesterol and lipoprotein cholesterol levels in men. Four healthy subjects were studied over 2 five-week periods, using diets containing 20% protein, 40% carbohydrate, 40% fat and 400 mg cholesterol per day. The P/S ratio was 0.25 for the high-saturated fat diet and 4.0 for the high-polyunsaturated fat diet. On the high-polyunsaturated fat diet, total plasma cholesterol decreased

by 24%. Most of this reduction was due to a 60% decrease in LDL-C, although decreases in VLDL-C (8%) and HDL-C (32%) also occurred.

A similar study was carried out by Schwandt and co-workers (1982), using more moderate P/S ratios. In a cross-over design, two groups of 15 normolipidemic males consumed either a control diet (P/S ratio 0.3, cholesterol content 370 mg per day) or a modified fat diet (P/S ratio 1.0, cholesterol content 250 mg per day) for 3 months each. Both diets were identical with regard to quantities of protein (15%), carbohydrate (48%), and fat (37%), and were provided from mixed natural foods. With a P/S ratio of 1.0, researchers observed decreases of 15% and 9% in total serum cholesterol and 19% and 13% in LDL-C for each group, respectively. However, unlike Shepherd's results, HDL-C levels remained unaffected by the dietary change. These findings are in accordance with those of Thompson et al (1980), who found a 21% decrease in LDL-C and no change in HDL-C in a group of eleven males fed a diet containing a P/S ratio of 1.5 and less than 300 mg cholesterol per day.

Based on these studies, it appears that an increase in the P/S ratio has no consistent effect on serum HDL-C concentrations. Results from more current research have failed to settle the issue, with some reports suggesting that HDL-C decreases with diets containing high P/S ratios

(Schaefer et al, 1981; Jackson et al, 1984; Mattson and Grundy, 1985), and others showing no change in this lipoprotein fraction (Weisweiler et al, 1983). Further research into the effects of dietary fat alteration on HDL is warranted, due to the protective role which HDL appears to play in the development of CHD.

It is generally agreed that the substitution of polyunsaturated fat (PUFA) for saturated fat leads to a lowering of both total and LDL cholesterol levels (Goldberg and Schonfeld, 1985). Since the risk of developing CHD is proportional to these serum cholesterol levels, diets high in PUFA have been advocated for the prevention of CHD in the general population. Specifically, the Committee on Diet and Cardiovascular Disease (Health and Welfare Canada, 1976) has recommended the replacement of saturated fat with polyunsaturated fat, to attain a P/S ratio of 1.0-1.5:1.0. This recommendation has led to the need for further research concerning the safety of consumption of a high-PUFA diet over the long term (Conner and Conner, 1986).

1.3.3 Dietary Carbohydrate

In an effort to reduce the incidence of CHD, it has been recommended that total fat intake be reduced to 30-35% of total calories, with a compensatory increase in carbohydrate consumption of up to 50-55% of total calories

(Health and Welfare Canada, 1976; American Heart Association, 1978). These dietary recommendations are based on the assumption that increased carbohydrate consumption will not increase the risk of developing CHD (Coulston et al, 1983). Several studies have been carried out to test the validity of this assumption.

Schonfeld and co-workers (1976) examined the plasma lipid levels of 16 normolipidemic adults before and after five days of high-carbohydrate feeding. Subjects were fed a formula diet containing 80% of the energy as carbohydrate, 20% as protein and less than 1% as fat. This high-carbohydrate diet resulted in an increased hepatic secretion of VLDL-TG. The LDL-C decreased from a mean of 102 to 76 mg/dl, while LDL-TG remained unchanged. The HDL-C decreased from 43 to 32 mg/dl, while mean HDL-TG levels rose. Thus, the high-carbohydrate feeding resulted in the TG-enrichment of all 3 lipoprotein fractions. This is considered to be a negative effect, as elevated serum TG levels have been positively correlated with CHD development (Carlson et al, 1979; Hallfrisch et al, 1985).

Dietary manipulations by Gonen and co-workers (1981) yielded results comparable to those of Schonfeld et al (1976). A fat-free formula diet (85% carbohydrate, 15% protein) was fed to 11 healthy subjects for 7 days, resulting in a mean increase in serum VLDL-TG and VLDL-C,

along with significant decreases in serum levels of both LDL-C and HDL-C. Of the HDL subfractions, the most marked and uniform decrease occurred in HDL_2. This reduction is considered to be unfavorable as it is generally believed that HDL_2 is the subfraction with "anti-atherogenic" properties (Gonen et al, 1981).

From the data cited, no conclusions can be drawn regarding the mechanism responsible for the reduction in HDL. However, Blum et al (1977) have suggested that carbohydrate-rich diets result in decreased HDL synthesis. Gonen et al (1981) have theorized that below a certain level of fat intake (as on a low-fat, high-carbohydrate diet) production of HDL apoprotein A_1 is low, and results in a reduced concentration of HDL in the plasma. Further, Gonen et al (1981) speculate that the observed decrease in LDL-C following high-carbohydrate feeding is in some way related to the diet-induced impaired HDL production.

Coulston and associates (1983) criticized the work of both Schonfeld et al (1976) and Gonen et al (1981), stating that the diets used were extreme, with excessively high levels of carbohydrate and negligible amounts of fat. As well, formula diets were used, rather than those composed of mixed natural foods. Coulston et al (1983) proceeded to investigate the effects of carbohydrate-rich diets on serum TG and lipoprotein cholesterol levels, using moderate

amounts of carbohydrate and conventional foods. Eleven healthy subjects consumed a control diet of 40% carbohydrate and 41% fat and a high-carbohydrate diet which contained 60% carbohydrate and 21% fat for 10 days each, in a cross-over design. Sucrose accounted for 22-25% of the total carbohydrate in each diet. The high-carbohydrate diet resulted in a significant increase in plasma TG and a significant reduction in plasma HDL-C.

The results of Coulston et al (1983) confirm those of previous studies, and indicate that the short-term consumption of a low-fat, high-carbohydrate diet can lead to metabolic changes which have been associated with an increased incidence of CHD. It has been suggested that the deleterious impact of this diet may be transitory, and that beneficial effects may occur with chronic use (Coulston et al, 1983). As well, the type of carbohydrate consumed is of importance, as the effects of simple vs complex carbohydrates on blood lipid levels have been shown to vary (Reiser et al, 1978; Anderson et al, 1980), with sucrose having the least desirable effect. Until these issues have been clarified, it seems reasonable to question the advocacy of high-carbohydrate diets for the prevention of CHD.

In conclusion, current dietary recommendations include suggestions for the public to decrease the intake of total fat (including saturated fat), cholesterol, salt and sugars,

and to increase the intake of polyunsaturated fat and complex carbohydrates, including fibre (American Heart Association, 1978; Food and Nutrition Board, 1980). In a recent investigation by Hallfrisch and co-workers (1985), minimal modifications in the typical American diet were made to incorporate these dietary recommendations. Effects of these changes on blood lipid levels were determined. Over 13 weeks, 53 healthy men and women consumed a diet which supplied 35% of the energy from fat, 50% from carbohydrate (35% complex, 15% simple), 15% from protein and a P/S ratio of 0.7. As a result, plasma total cholesterol, LDL-C and VLDL-C were significantly reduced, while plasma TG and HDL-C levels remained unchanged. Thus, a diet which conforms to current dietary guidelines appears to produce beneficial changes in the blood lipids of both men and women. Unfortunately, the independent effects of the various dietary factors were not discernible and have yet to be determined. Nevertheless, the results of the research of Hallfrisch et al (1985) shed a more positive light on the dietary recommendations currently being held in practice.

1.4 EXERCISE AND SERUM LIPOPROTEINS

While studies related to the effects of diet on serum lipids have been carried out for greater than 30 years, it is only recently that investigative attention has been directed toward exercise and its relation to lipoprotein

metabolism. Epidemiological data suggest that high levels of physical activity are associated with a decreased incidence of CHD (Paffenbarger et al, 1978; Moore et al, 1983). Exercise is reported to exert a preventive effect on the progression of CHD through the alteration of serum lipid and lipoprotein levels (Rifai et al, 1987). Elevated TG levels have been related to an increased CHD risk (Witztum and Schonfeld, 1979), as have reduced levels of HDL-C (Miller and Miller, 1975). The specific effects of exercise on these lipid fractions have been investigated through both cross-sectional and longitudinal studies.

1.4.1 Cross-Sectional Studies

Results of cross-sectional studies indicate that persons engaged in routine vigorous physical activity tend to have higher HDL-C levels (Wood et al, 1983) and lower TG levels (Haskell et al, 1980) than their sedentary counterparts. The observed increase in HDL-C is of particular interest due to its proposed "anti-atherogenic" properties.

During the Lipid Research Clinics Program Prevalence Study (Haskell et al, 1980), plasma lipoprotein determinations and treadmill exercise testing were performed on 2319 men and 2067 women, age 20 years or older. Level of physical activity was determined via subject self-report.

An analysis of the data failed to reveal a correlation between treadmill exercise test performance and HDL-C concentration for men or women. However, individuals who reported regular strenuous physical activity generally had higher HDL-C levels than those who reported being physically inactive. When HDL-C level was adjusted for age, body mass index, alcohol use, cigarette smoking, and interclinic variation, more active men and women had higher HDL-C levels than their sedentary counterparts. Thus, a positive association between activity level and HDL-C concentration was deemed evident.

Smith and co-workers (1982) compared the lipoprotein cholesterol levels of 7 female competitive swimmers (12 hours swimming/week), 11 synchronized swimmers (6 hours swimming/week) and 6 sedentary controls. The 3 groups were similar with respect to age (mean=20 years), body weight, diet composition, alcohol consumption and smoking habits. Total plasma cholesterol and LDL-C were not significantly different among the groups. HDL-C concentrations were significantly increased in the competitive swimmers compared with the synchronized swimmers and the control group. The researchers concluded that plasma HDL-C is significantly elevated in those women participating in an intensive, but not a moderate, exercise program.

Hartung and associates (1980) examined the lipoprotein concentrations and dietary intakes of 59 marathon runners, 85 joggers, and 74 inactive controls. All subjects were healthy males between the ages of 35 and 66 years. While dietary intake was observed to be similar, the 3 groups differed significantly with respect to body weight and number of cigarettes smoked per day. After statistical adjustment for these variables, a significant difference in HDL-C concentration among the 3 groups was revealed. Long distance runners had significantly higher HDL-C levels than joggers, who, in turn, had significantly higher levels than sedentary controls. Results of a study by Moore and associates (1983), employing female subjects yielded results similar to those of Hartung et al. HDL-C was found to be significantly higher in long distance runners compared to joggers or inactive controls.

Results of these and other cross-sectional studies (Enger et al, 1977; Vodak et al, 1980) support the hypothesis that regular physical activity may, at least in part, be responsible for increases in HDL-C concentration in humans.

1.4.2 Longitudinal Studies

With longitudinal studies, the possibility of self-selection is reduced, as each subject acts as his or her own

control. One such study, conducted by Huttunen and co-workers (1979), investigated the effects of moderate physical exercise on serum lipoproteins in middle-aged males. One hundred physically inactive men (age 40-50 years) were randomly assigned to either an exercise or a control group. Subjects in the control group were advised to maintain their previous exercise habits. The exercise group participated in a 4-month program consisting of 3-4 weekly exercise sessions. Each session involved 30 minutes of walking, jogging, cycling or swimming. This study was not controlled for food intake, however all subjects were advised to consume a constant diet. Following training, blood lipid analysis revealed a significant increase in HDL-C in the exercise group, while serum TG levels decreased. Slight but significant decreases in serum total cholesterol and LDL-C were observed in both the exercise and control groups, and were attributed to seasonal trends typical for the population during the spring months (Aromma et al, 1975).

Huttunen's findings confirm those of an earlier study by Lopez et al (1974), in which a significant decrease in serum TG and a significant increase in HDL-C were noted in 13 males following a 7-week exercise program. Both studies support the thesis that moderate endurance exercise results in elevations in HDL-C concentration in man.

Nye and co-workers (1981) examined serum lipoprotein changes in 17 sedentary men (age 30-45 years) who participated in a moderate-intensity calisthenics program twice weekly for 10 weeks. Dietary intake was not regulated. While LDL-C concentrations decreased significantly, total HDL-C concentrations remained unchanged. However, a significant shift did occur in the proportions of the HDL subfractions. The concentration of HDL_2 increased, with a corresponding decrease in the HDL_3 component. This finding may be of significance, as it has been suggested that HDL_2 is the more "anti-atherogenic" of the two subfractions (Shepherd et al, 1980).

A study by Lipson and associates (1980) confounds the issue. Five men and five women (age 19-22 years) performed 30 minutes of treadmill exercise daily, for 6 weeks. Exercise intensity was adjusted to produce an oxygen consumption which was 70% of the subjects' maximal oxygen uptake (VO_2 max). The diet of all subjects was monitored throughout the experiment and total caloric intake was adjusted daily to maintain constant body weight. Lipson reported a significant decrease in total plasma cholesterol, but no change in TG or lipoprotein cholesterol levels. The authors concluded that exercise conditioning did not alter HDL-C levels, and that the elevation in HDL-C shown in previous studies may well be due to changes in body weight (Avogaro et al, 1978). It should be noted that there was a

small but significant weight loss during the experiment and that the intensity and duration of the regimen may have been insufficient to produce changes in the HDL-C level. Problems in experimental design were intensified by the use of a small heterogeneous sample.

The relationship between exercise, weight loss and HDL-C is not clearly understood. The data from both cross-sectional (Haskell et al, 1980; Hartung et al, 1980; Moore et al, 1983) and longitudinal studies (Huttunen et al, 1979; Lopez et al, 1974; Nye et al, 1981) strongly suggest that endurance activities increase the plasma concentration of HDL-C. Moreover, population studies indicate that lean individuals have higher HDL-C levels than those who are more obese (Huttunen et al, 1979). Because an association exists between weight loss and exercise training, it is of importance to determine to what extent the higher HDL-C concentrations, characteristic of physically active people, can be attributed to their relative leanness.

Williams et al (1983) examined the interaction between weight loss and exercise-induced increases in HDL-C. In this one-year study, eighty-one healthy males (age 30-55 years) were assigned to either a moderate running program or to a sedentary control group. Weight loss was strongly associated with increases in HDL-C in the exercise group, however weight changes in the absence of exercise training

produced no change in HDL-C levels. Thus it appears that the metabolic consequence of the weight loss differed between the two groups.

Williams' findings are supported by Huttunen's (1979) study, which, as previously cited, investigated the effects of an aerobic exercise program on serum lipoproteins in men. A small but significant decrease in weight was observed in both the exercise and the control groups, however only in the exercise group was an increase in HDL-C evident. To further investigate the relationship between weight loss and changes in lipoprotein concentration, the exercise subjects from Huttunen's (1979) study were subdivided into two groups, those who maintained their weight, and those whose weight decreased by greater than 1 kg. A significant elevation in HDL-C was seen in both subgroups, suggesting that factors other than weight loss may be responsible for the exercise-induced increases in this lipoprotein fraction.

While a number of studies have demonstrated an association between exercise and serum lipoproteins in men, less consistent results have been found in the few reported studies performed with female subjects.

The effects of acute, moderate intensity exercise are reportedly similar for men and women (Lennon et al, 1983). It would be expected, then, that exercise over a longer duration would reduce serum TG and increase HDL-C in women,

as reported for men. Lewis et al (1976) reported the effects of a moderate exercise program and caloric restriction on weight loss in obese middle-aged women. Twenty-two women participated in the program, which consisted of a 20-minute walk-jogging session twice a week for 17 weeks. Caloric restriction was self-determined and was generally moderate. Lewis' data revealed a significant training effect and a loss of body fat among the subjects. While only minor changes occurred in the various lipoprotein levels, the HDL-C:LDL-C ratio did increase significantly.

Moll and co-workers (1979) found that vigorous exercise did not alter the HDL-C levels in females. Fourteen non-obese women, age 22-26 years, participated in a 6-week exercise program, which involved 45 minutes of jogging, 5 days per week. There was a marked improvement in aerobic performance, with no change in body weight. While a significant fall in total cholesterol occurred after training, there was no significant alteration in HDL-C. This is in contrast to the increased HDL-C concentration noted by both Altekruse and Wilmore (1973) and Lopez et al (1974), whose studies in men employed a training protocol similar to that of Moll. This disparity suggests that the duration of training needed to alter lipoprotein levels may differ in males and females.

Neither Wynne et al (1980) nor Frey et al (1982) were able to demonstrate a beneficial increase in the HDL-C levels of women as a result of exercise training. The two studies employed a similar protocol. Female subjects participated in a bicycle ergometer training program consisting of 3-30 minute sessions per week for 10 weeks. The training program resulted in an increase in maximum oxygen uptake and a decrease in per cent body fat for both study groups. However, post-training values of HDL-C and TG did not differ from pre-training values.

Why exercise conditioning elevates HDL-C in men, but not in women, is currently unknown. It is known, however, that pre-training levels of HDL-C are higher in females than in males at all ages beyond puberty (Hazzard et al, 1984; Zonderland et al, 1986). Although the mechanism of action is not clear, this sex difference is thought to be related, at least in part, to the effects of sex steroids on lipoprotein metabolism (Goldberg et al, 1985; Sherwin et al, 1987).

Repeated research has shown that exogenously administered estrogens (Cauley et al, 1982; Schaefer et al, 1983; Zonderland et al, 1986; Sherwin et al, 1987), as well as endogenous estrogens (Frey et al, 1983), are positively associated with HDL-C levels in women. Studies involving the administration of androgens to men (Solyom, 1972; Hurley

et al, 1984; Webb et al, 1984; Zonderland et al, 1986) and women (Taggart et al, 1982) have demonstrated a reduction in the concentration of HDL-C, particularly the HDL_2 subfraction. On the other hand, analysis of the relationship between endogenous testosterone levels and HDL-C concentration has demonstrated a positive correlation in some reports (Nordoy et al, 1979; Mendoza et al, 1981; Frey et al, 1983) and a negative association in others (Semmens et al, 1983; Goldberg et al, 1985).

Thus, a sensitivity to sex steroids has been exhibited by HDL-C (Tikkanen et al, 1982). Based on this observation, researchers have speculated that exercise-induced changes in HDL-C may be mediated by changes in sex hormone concentrations, and that these hormone responses may be gender-specific (Frey et al, 1983; Hazzard et al, 1984). Further, females may require a greater exercise stimulus than males in order to elicit a sex hormone response leading to elevations in HDL-C concentration (Wynne et al, 1980).

While increases in HDL-C have been well-documented in exercising males, the limited reports in physically active females have shown no such effect. In addition to differential sex hormone responses, this observed lack of change in women may be due to the confounding influence of diet, alcohol intake, smoking and oral contraceptive use on HDL-C levels (Wynne et al, 1980; Frey et al, 1982).

The proposed research is designed to investigate the effects of both aerobic exercise and diet on serum lipoprotein levels in adult sedentary women, while controlling for the confounding influence of alcohol intake, smoking, and oral contraceptive use.

Chapter II

MATERIALS AND METHODS

2.1 SUBJECTS

The 24-week study involved 2 groups of sedentary females, all employees or students at the University of Manitoba. The 8 experimental subjects were chosen from among new members of the University of Manitoba's Target Fitness Centre, and ranged in age from 27 to 42 years (mean age 36 years). This group was counselled to consume a low fat, low cholesterol diet, and attended 3-45 minute aerobic exercise classes per week, for the duration of the study. The 10 control subjects were chosen from among respondents to University newspaper advertisements and notices posted on campus, and ranged in age from 39 to 47 years (mean age 42 years). These subjects were encouraged to maintain their regular diet and exercise habits.

Initial screening was done via telephone interview. Subject selection was based on body weight, physical fitness level, serum lipid levels, smoking habits, alcohol consumption and oral contraceptive use. A detailed explanation of the requirements can be found in Appendix A. Subjects next attended an orientation session designed to

familiarize them with the experimental protocol. Informed consent was then obtained from each subject (see Appendix B).

Testing procedures included dietary and fitness assessments, and blood lipid analysis. Experimental subjects underwent dietary assessment and blood lipid analysis at 0, 6, 12, 18, and 24 weeks. Fitness levels of this group were assessed at 0, 12 and 24 weeks. Control subjects underwent complete testing at 0, 12 and 24 weeks.

2.2 BLOOD ANALYSIS

Venous blood samples were taken by a medical technologist following a 14-hour fast. Approximately 10 ml of blood was drawn from each subject into vacutainer tubes. This blood was allowed to clot for 30 minutes at room temperature, and then centrifuged (Model CS) (IEC International Centrifuge, International Equipment Co., Boston, Massachusetts) for approximately 5 minutes at 900xg to precipitate the clot. Sera were pipetted into 5 ml screw-top vials and stored at $4^{o}C$ until required for analysis.

2.2.1 Separation of Lipoproteins

The serum lipoproteins were fractionated by ultracentrifugation (Beckman L5-50B ultracentrifuge) (Beckman Instruments, Palo Alto, California 94304), using

rotor type 40.3 at 18°C and 34,000 rpm (calculated 104,000xg) for 18 hours (Appendix C).

2.2.2 Cholesterol Determination

A cholesterol colorimetric assay kit was used for cholesterol determination (Fisher Diagnostics, Nepean, Ontario), based on the method of Allain et al (1974) (Appendix D).

2.2.3 Triglyceride Determination

Serum triglyceride concentrations were quantitated using a triglyceride colorimetric assay kit (Fisher Diagnostics, Ottawa, Ontario) and based on the methods of Fossati and Lorenzo (1982) and McGowan et al (1983) (Appendix E).

2.3 FITNESS ASSESSMENT

The subjects were instructed to refrain from eating large meals or consuming beverages containing caffeine for 2 hours prior to their exercise assessment. They were also asked not to engage in any strenuous exercise during the previous 12 hours. Upon reporting to the Human Performance Laboratory, it was ensured that all subjects had signed an informed consent form explaining the nature of the exercise tests to be conducted, and any risks or discomfort that might be encountered. The Physical Activity Readiness

Questionnaire (PAR-Q) (Appendix F) was administered verbally in order to screen out subjects for whom exercise testing was contraindicated.

2.3.1 Body Composition

Height and weight were measured according to the Standardized Test of Fitness protocol (1986). Body mass index (BMI) was calculated as weight(kg)/height^2 (m).

Body fat was estimated from body density using the equation of Brozek et al (1963). Body density was determined by hydrostatic weighing. The mean of 4 valid measurements was used to calculate body density.

Recent studies have determined that density of the lean component of the body may be highly variable among individuals. In young and older populations, especially, variations in bone mineral and total body water may lead to overestimation of relative body fat (Wilmore, 1983). In order to monitor this source of error, and to collect additional useful information regarding the relationship between per cent body fat and skinfold measurements in females, the sum of 5 skinfold measurements (SOS) was determined. The biceps, triceps, suprailiac, medial calf and subscapular skinfolds were measured using Harpenden calipers, according to the protocol outlined in the Standardized Test of Fitness Operations Manual (1986).

2.3.2 Maximum Oxygen Uptake

Maximum oxygen uptake (VO_2 max) was predicted from the heart rate response to a submaximal, graded cycle ergometer test. Subjects were instructed to pedal for 3 continuous four-minute stages. Steady state target heart rates ranging from 130 to 170 bpm were established for Stage 1 and 2, respectively. A metronome and verbal encouragement were used to maintain each subject's pedalling cadence at 50 rpm.

Heart rate was monitored for the last 15 seconds of each minute using an ECG (Cambridge) and a CM_5 lead. The Astrand-Ryhming nomogram (1954) as modified for age (Astrand, 1960) was used to predict VO_2 max from steady state heart rate.

The fitness assessments were conducted and analyzed by qualified fitness appraisers at the University of Manitoba's Target Fitness Centre.

2.4 EXERCISE PROGRAM

Subjects in the exercise group took part in a community-based aerobic exercise program, and attended 3 aerobic fitness classes per week for 24 weeks. Each 45 minute exercise session included a 5 minute warm-up, 20 minutes of aerobic conditioning, and a 5 minute cool-down. The remainder of each workout was devoted to flexibility and strength exercises.

Exercise subjects were taught to monitor their heart rate and were encouraged to exercise at an intensity between 70 and 85% of their maximum heart rate during the aerobic phase of each fitness session. Twice during each session, participants were asked to determine their heart rate, and to record them at the conclusion of the class. This record served as an index of physical activity and attendance.

The fitness classes were conducted on campus at the Target Fitness Centre, Faculty of Physical Education and Recreation Studies. Classes were conducted in a standardized fashion by trained fitness instructors.

The fitness instructors monitored the exercise program for compliance, and provided encouragement to prevent participants from losing interest in the program. The instructors also monitored the level of physical activity in the control group by questionnaire (see Appendix G). The control subjects completed these questionnaires at Weeks 0, 12 and 24 of the study.

2.5 DIETARY ASSESSMENT

All subjects were instructed in the completion of a three-day diet record. A copy of the instructions for completion of the diet record is included in Appendix H.

The computer nutrient data bank (Canadian Nutrient File, Health and Welfare Canada, 1986) at the University of

Manitoba was used to analyze the diet records for the per cent contribution to total energy of protein, carbohydrate, total fat, polyunsaturated fatty acids, monounsaturated fatty acids and saturated fatty acids, as well as total kilocalories.

At the start of the study, the exercise subjects were familiarized with the Dietary Recommendations for Canadians (Health and Welfare Canada, 1976) (Appendix I), and instructed to consume a low fat, low cholesterol diet based on these recommendations. Each exercise subject was given a copy of the "American Heart Association Cook Book" and "Dietary Fats and Your Heart", along with a list of dietary guidelines (Appendix J), and a sample menu (Appendix K). The initial orientation to the diet was followed by diet counselling at 3-week intervals to reinforce new dietary habits and to provide encouragement to the subjects.

The control subjects were advised to maintain their regular dietary habits throughout the study period.

2.6 STATISTICAL ANALYSIS

Data was analyzed using the Statistical Analysis System (SAS), 1984 and 1986 versions. Student's t-tests were used for the comparisons between the exercise and control groups. Student's t-tests for paired data were employed to determine the statistical significance of within-group differences.

Chapter III

RESULTS

3.1 BETWEEN-GROUP COMPARISONS

Comparisons were made between the experimental and control groups for all variables at 0 and at 24 weeks. Student's t-tests revealed that protein intake was the only variable which differed significantly among the groups, being higher in the experimental group at 24 weeks ($p \leq 0.0128$).

3.2 WITHIN-GROUP COMPARISONS

Comparisons were made within groups using Student's t-tests for paired data.

3.2.1 Serum Lipid and Lipoprotein Variables

Serum lipid and lipoprotein values are presented in Table 1.

Serum TG levels did not change significantly in the experimental or the control group over the 24-week study period.

TABLE 1

Serum Lipid and Lipoprotein Variables for the Exercise and Control Groups over 24 Weeks

Variable	Week 0	Week 6	Week 12	Week 18	Week 24
Exercise group:					
Triglyceride (mg/dl)	63.9±15.5	54.9±14.3	59.6±20.9	62.4±27.6	63.3±17.4
Total Cholesterol (mg/dl)	194.3±21.7[a]	168.1±13.5[b]	177.7±21.6[c]	186.4±25.7[ac]	185.9±21.6[ac]
HDL-C (mg/dl)	48.0±12.6[a]	52.0±9.8[b]	43.8±7.0[ac]	40.0±10.9[c]	34.4±14.0[c]
LDL-C (mg/dl)	129.0±24.9[a]	103.9±16.2[b]	119.9±26.8[a]	128.8±25.8[a]	129.6±22.2[a]
VLDL-C (mg/dl)	17.3±11.4[ab]	15.4±5.0[ab]	14.0±9.5[ac]	17.8±10.4[ab]	21.9±8.4[b]
HDL-C:Total Cholesterol	0.25±0.06[a]	0.31±0.06[b]	0.25±0.05[a]	0.22±0.07[ac]	0.18±0.06[c]
Control group:					
Triglyceride (mg/dl)	83.1±35.7	-	73.0±26.9	-	69.6±22.9
Total Cholesterol (mg/dl)	207.7±23.4	-	191.2±28.3	-	201.4±26.4
HDL-C (mg/dl)	48.4±6.5[a]	-	41.5±5.8[b]	-	39.2±5.1[b]
LDL-C (mg/dl)	147.3±26.0[a]	-	128.6±19.1[b]	-	137.1±24.9[ab]
VLDL-C (mg/dl)	14.3±7.8[a]	-	21.3±12.8[ab]	-	25.0±11.4[b]
HDL-C:Total Cholesterol	0.24±0.05[a]	-	0.22±0.04[a]	-	0.19±0.02[b]

Means with the same letter are not distinguished by the Student's t-test for paired differences at the 0.05 significance level.

Values are mean ± SD.

A significant decrease in total cholesterol ($p \leq 0.0013$) and LDL-C ($p \leq 0.0013$) was observed at 6 weeks in the exercise group, followed by a return to baseline range by 24 weeks. In the control group, total cholesterol levels remained unchanged, while LDL-C decreased significantly at 12 weeks ($p \leq 0.0179$).

After 6 weeks, HDL-C rose significantly in the exercise group ($p \leq 0.0428$), but subsequent values showed a progressive decrease to below baseline. Values for the ratio of HDL-C to total cholesterol showed a similar trend. Control group values for HDL-C and HDL-C:total cholesterol at 24 weeks were significantly lower than initial values ($p \leq 0.0004$ and $p \leq 0.0124$, respectively).

VLDL-C levels did not vary significantly from baseline in the experimental group. A significant rise in VLDL-C at 24 weeks was observed for the controls ($p \leq 0.0430$).

3.2.2 Fitness Variables

Table 2 reveals no significant changes in any of the fitness parameters for the exercise group.

In the control group, a small but statistically significant rise in mean body weight was evident between Weeks 12 and 24 ($p \leq 0.0047$). As a result of this change, BMI also increased significantly during this time ($p \leq$

TABLE 2

Fitness Variables for the Exercise and Control Groups over 24 Weeks

Variable	Week 0	Week 6	Week 12	Week 18	Week 24
Exercise group:					
Weight (Kg)	58.8±10.1	-	58.7±10.8	-	58.6±11.5
SOS	81.7±21.3	-	80.1±24.5	-	84.2±27.6
VO_2 max (ml/kg/min)	36±3	-	36±5	-	35±5
Resting heart rate (bpm)	75±11	-	77±11	-	77±5
Body density (g/cc)	1.034±0.012	-	1.034±0.014	-	1.031±0.013
BMI	22.3±3.1	-	22.3±3.4	-	22.2±4.0
Control group:					
Weight (Kg)	58.2±6.4[a]	-	58.6±5.9[a]	-	59.8±6.4[b]
SOS	80.6±14.3[ab]	-	79.6±16.7[b]	-	87.3±21.5[a]
VO_2 max (ml/kg/min)	35±6	-	35±7	-	34±6
Resting heart rate (bpm)	73±8	-	76±12	-	71±10
Body density (g/cc)	1.021±0.019	-	1.021±0.015	-	1.019±0.016
BMI	22.8±1.4[a]	-	22.9±1.4[a]	-	23.4±1.5[b]

Means with the same letter are not distinguished by the Student's t-test for paired differences at the 0.05 significance level.

Values are mean ± SD.

0.0031). The SOS value became significantly elevated between 12 and 24 weeks of the study ($p \leq 0.0392$). Measures of VO_2, resting heart rate and body density remained stable throughout the experiment.

3.2.3 Dietary Variables

Dietary intake of exercise and control subjects is shown in Table 3.

With the exception of a decrease at Week 12 ($p \leq 0.0526$), there were no significant differences in total caloric intake (kcal/day) for the exercise subjects over the study period. The controls showed a slight but nonsignificant increase in total caloric intake (kcal/day) over the 24 weeks.

In the exercise group, a slight reduction in per cent carbohydrate was observed at 24 weeks, but this change was not significant. Carbohydrate intake, as a percentage of total caloric intake, remained constant for the control group.

While no change in per cent protein was revealed in the experimental group, the values for control subjects displayed a significant drop at 12 weeks ($p \leq 0.0352$), and remained depressed below baseline at 24 weeks.

TABLE 3

Dietary Variables for the Exercise and Control Groups over 24 Weeks

Variable	Week 0	Week 6	Week 12	Week 18	Week 24
Exercise group:					
Total caloric intake (Kcal/day)	1615±479[a]	1665±573[a]	1378±418[b]	1369±376[ab]	1503±411[ab]
Carbohydrates (% of total Kcal)	46.0±6.1	45.0±8.1	48.1±11.7	47.9±11.4	41.0±9.2
Protein (% of total Kcal)	16.5±1.6[ab]	17.8±2.6[ab]	17.7±5.1[ab]	17.9±2.3[ac]	17.4±2.5[b]
Fat (% of total Kcal)	36.8±3.6	37.4±5.4	33.6±7.6	34.8±9.7	41.4±11.4
Saturated fatty acids (% of total Kcal)	13.2±2.7[a]	12.5±3.4[a]	10.4±2.9[ab]	10.0±3.0[b]	13.5±4.0[ab]
Polyunsaturated fatty acids (% of total Kcal)	5.8±1.4	5.5±2.1	5.6±2.1	6.5±4.5	7.3±4.6
Monounsaturated fatty acids (% of total Kcal)	13.8±1.2[a]	14.9±2.6[a]	11.8±2.4[b]	13.1±4.8[ab]	15.3±7.1[ab]
Control group:					
Total caloric intake (Kcal/day)	1602±228	-	1789±334	-	1810±499
Carbohydrates (% of total Kcal)	45.8±4.8	-	47.8±3.6	-	46.9±3.6[ab]
Protein (% of total Kcal)	15.5±2.2[a]	-	14.1±1.6[b]	-	14.1±2.4
Fat (% of total Kcal)	36.3±5.5	-	37.3±5.2	-	38.4±2.6
Saturated fatty acids (% of total Kcal)	12.6±2.6	-	12.4±2.1	-	13.7±3.0
Polyunsaturated fatty acids (% of total Kcal)	5.4±2.0	-	5.4±1.3	-	5.3±1.6
Monounsaturated fatty acids (% of total Kcal)	14.5±3.2	-	15.4±4.5	-	15.4±2.0

Means with the same letter are not distinguished by the Student's t-test for paired differences at the 0.05 significance level.

Values are mean ± SD.

The percentage of total fat in the diet of the exercise subjects remained stable to 18 weeks, then rose significantly at 24 weeks ($p \leq 0.0298$). Reductions in saturated fatty acids and monounsaturated fatty acids (expressed as % of total kilocalories) were statistically significant at 18 weeks ($p \leq 0.0246$) and 12 weeks ($p \leq 0.0500$), respectively. Although no significant alterations in polyunsaturated fatty acid levels (% total kilocalories) were demonstrated, a slight rise was observed between 12 and 24 weeks of the study. For all measures of fat, the control group values remained constant throughout the study period.

Chapter IV

DISCUSSION

The present study investigated the effects of aerobic exercise and diet modification on serum lipid and lipoprotein levels in adult sedentary women. Following the 24-week program, levels of serum TG, total cholesterol, and LDL-C in the experimental group had not changed significantly from baseline. However, transient decreases in these variables were detected at 6 weeks, and were found to be significant for total cholesterol and LDL-C only. While HDL-C levels showed a significant increase at 6 weeks, this change was transitory and levels had dropped to below baseline by 24 weeks. The observed change in HDL-C is considered to be unfavorable, as reduced levels of HDL-C have been associated with an increased risk of CHD (Miller and Miller, 1975). The HDL-C:total cholesterol ratio, an indicator of the risk of developing CHD, also showed an unfavorable drop to below baseline at 24 weeks.

Cross-sectional studies have shown the association between endurance exercise and lowered serum TG concentrations to be quite consistent. Lehtonen and Viikari (1978) compared the serum lipid levels of 23 regularly training men (average exercise 83 km running or skiing

weekly) and 15 healthy men who did not participate in any regular exercise. The physically active group displayed significantly lower TG concentrations than the sedentary group, a finding which has been demonstrated in more recent studies (Wood and Haskell, 1979; Vodak et al, 1980). In the current study, 24 weeks of aerobic training failed to produce a decrease in the TG concentrations of the exercise subjects, and values were not significantly different from those of the control subjects.

The effects of exercise training on total cholesterol levels are limited and variable (Haskell, 1984), with the majority of researchers reporting no difference between active and sedentary groups of individuals (Vodak et al, 1980; Smith et al, 1982; Moore et al, 1983; Durstine et al, 1987). The results of the present study are in agreement with these findings, with no significant difference in total cholesterol reported between the exercise and control groups.

The impact of habitual exercise on serum LDL-C concentration appears to be quite small, and the magnitude of change highly variable in cross-sectional comparisons (Haskell, 1984). While some researchers have found lower LDL-C values for active versus inactive subjects (Wood et al, 1976; Lehtonen and Viikari, 1978; Hartung et al, 1980), others have found no differences among the groups (Vodak et

al, 1980; Smith et al, 1982; Moore et al, 1983; Durstine et al, 1987). The LDL-C levels of the exercisers in the current study were not significantly different from control levels.

Cross-sectional data suggest that a relationship exists between high levels of chronic physical activity and elevated HDL-C concentrations. This relationship is of interest due to the possible role of HDL-C as a "protective agent" against CHD (Miller and Miller, 1975; Gordon et al, 1977). Wood et al (1977) found that male and female long distance runners, who regularly ran more than 15 miles per week, had HDL-C levels greater than matched groups of nonexercising controls. More recent studies comparing long distance runners and sedentary controls have yielded similar results (Hartung et al, 1980; Moore et al, 1983; Durstine et al, 1987). Elevated levels of HDL-C have also been reported in swimmers (Smith et al, 1982), cross-country skiers (Enger et al, 1977), and tennis players (Vodak et al, 1980) when compared with relatively inactive controls. In contrast to the results of cross-sectional studies, the present investigation showed a significant decrease in the HDL-C levels of the exercise subjects following 24 weeks of training.

The results of longitudinal training studies measuring the effect of increased physical activity on serum

lipoproteins have been inconsistent. Studies in men have repeatedly shown an increase in HDL-C with training, along with decreases in LDL-C and TG (Altekruse and Wilmore, 1973; Lopez et al, 1974; Huttunen et al, 1979). No such rise in HDL-C has been demonstrated in similar studies conducted in young (Moll et al, 1979; Wynne et al, 1980; Frey et al, 1982) and middle-aged (Cauley et al, 1987) women. As well, no change in TG concentration has been observed (Wynne et al, 1980; Frey et al, 1982). The present study, investigating the effects of aerobic exercise on serum lipoprotein levels in women, actually showed a decrease in HDL-C, with no change in TG.

Improvement in physical fitness level has been associated with increases in HDL-C and decreases in TG, changes believed to reduce the risk of developing CHD. Level of fitness is commonly expressed in terms of maximal aerobic power (VO_2 max), with an increased VO_2 max reflecting an increase in aerobic fitness (Gaesser and Rich, 1984). Elevations in VO_2 max with physical conditioning are known to be a function of the type, duration, intensity and frequency of conditioning, as well as the initial level of fitness (Gaesser and Rich, 1984; Dowdy et al, 1985).

Aerobic exercise programs appear to have the potential to provide the quantity and quality of exercise necessary for developing and maintaining cardiorespiratory fitness

(ACSM, 1978), yet few investigations have been performed to verify this. A study by Dowdy and co-workers (1985) examined the effects of aerobic exercise on physical work capacity and cardiorespiratory function of young, middle-aged women. Twenty-eight sedentary females participated in the 10-week program, which consisted of 3-45 minute exercise sessions per week at an intensity of 70-85% of the heart rate reserve. The fitness program was designed in accordance with the guidelines set out by the ACSM (1986) for improved physical fitness. Results of the study revealed a significant increase in VO_2 max and a significant decrease in RHR, indicating that a program of aerobic exercise does indeed improve cardiorespiratory fitness.

The exercise program employed in the current study was similar in design to that of Dowdy and associates (1985), with an extended length of 24 weeks. However, unlike the results of Dowdy, no significant change in VO_2 max was observed at the conclusion of the study period. This lack of change is surprising, and we can only speculate as to the cause. Training intensity is a critical factor in the improvement of aerobic fitness (ACSM, 1986), and intensities greater than or equal to 70% of maximum heart rate have been documented to result in improvements in cardiorespiratory fitness (Gaesser and Rich, 1984). Our subjects were encouraged to exercise at an intensity between 70 and 85% of their maximum heart rate. To accomplish this, subjects were

asked to monitor their pulse rates twice during the aerobic segment of each exercise session. While this method provides a reasonable estimate of exercise intensity, it is not completely accurate (Fox and Mathews, 1981). Due to the lack of rigorous control, exercise intensity in the current study may have been insufficient to produce an increase in VO_2 max, and thus any positive changes in serum lipoprotein concentrations. Frequency of exercise is also important, with a minimum of three sessions per week contributing to an improved level of aerobic fitness (ACSM, 1986). Subjects in the present study were asked to exercise 3 times per week for 24 weeks. Compliance in this study was approximately 75%, making the frequency of exercise less than optimal for increasing physical fitness.

The initial level of fitness may affect the extent to which VO_2 max is altered following an aerobic exercise program (Dowdy et al, 1985). The exercise subjects in the present investigation had not participated in any regular physical activity for the 6 months prior to the study. In spite of this, the initial VO_2 max of our exercise subjects was above average relative to the norms for women in the same age group (Canadian Standardized Test of Fitness Operations Manual, 1986). Following exercise, a significant increase in VO_2 max may have occurred had our subjects possessed a lower initial level of fitness.

Failing to observe a significant rise in VO_2 max, we expected no change in the lipoprotein concentrations of the exercise subjects over the 24 weeks. However, our findings revealed a transitory decrease in serum total cholesterol and LDL-C in both the exercise and the control groups between 0 and 12 weeks of the study. As well, a significant decrease in HDL-C was observed in both groups at 24 weeks. Lipoprotein changes occurring in both the experimental and control groups of previous studies have been attributed to seasonal variation (Aromma et al, 1975; Huttunen et al, 1979), and this may well account for the changes observed in the present study.

While physical activity has been correlated with increased HDL-C levels and decreased LDL-C and total cholesterol levels (Lopez et al, 1974; Huttunen et al, 1979), a causal relationship has yet to be established. Due to the proposed "anti-atherogenic" effect of HDL-C (Miller and Miller, 1975), the association between increased exercise and elevations in HDL-C has been of particular interest to researchers. Some investigators have cited an increase in VO_2 max as the link between exercise and a rise in HDL-C (Huttunen et al, 1979; Farrell and Barboriak, 1980). Other investigators have disagreed, stating that it is the exercise-induced change in body weight that leads to alterations in HDL-C. Lipson and associates (1980) administered a 6 week program of 30 minutes of daily

treadmill exercise to 10 subjects, controlling for dietary intake and body weight. Following the study, VO_2 max rose significantly, while HDL-C displayed no significant change. Lipson concluded that a decrease in body weight was necessary to produce increases in HDL-C, and that it was not exercise per se that induced increases in this lipoprotein fraction. This conclusion is based on the negative correlation between body weight and HDL-C observed by Avogaro et al (1978).

An investigation by Williams et al (1983) supported Lipson's conclusion and revealed a strong association between weight loss and increases in HDL-C following a 1-year endurance training program. Contrary to this, the results of Huttunen's (1979) study indicated that weight reduction was not the cause of the increase in HDL-C levels of exercising individuals. A highly significant elevation in HDL-C was seen in subjects who lost greater than 1 kg body weight and also in those who maintained a constant body weight during the intervention. To further confound the issue, a study by Despres and co-workers (1985) displayed increases in VO_2 max and decreases in body weight but no significant change in the HDL-C levels of 13 healthy men after 20 weeks of aerobic training.

In the present study, no change in body weight was observed following 24 weeks of aerobic exercise. Whether

this lack of change played a role in the failure to produce an increase in HDL-C is unclear, due to the contradictory nature of the data currently available.

The mechanism responsible for exercise-induced increases in HDL-C is currently unknown. Increasing evidence suggests that an adaptive increase in the activity of the lipoprotein lipase (LPL) enzyme following exercise training may be of major importance. Nikkala and associates (1978) found that long distance runners had higher LPL activity in muscle and adipose tissue than did sedentary men. As well, initially sedentary individuals have shown increases in muscle and adipose tissue LPL following endurance training (Svedenhag et al, 1983; Stubbe et al, 1983). LPL catalyzes the degradation of chylomicrons and VLDL to LDL, a process resulting in the formation of HDL (Nilsson-Ehle et al, 1980). Increased levels of LPL lead to increased formation of HDL (Witztum and Schonfeld, 1979). Thus, it has been suggested that exercise training leads to elevations in HDL via increases in LPL activity. The lecithin:cholesterol acyl transferase (LCAT) enzyme also plays a role in the formation of functional HDL particles (Witztum and Schonfeld, 1979). The activity of LCAT has been shown to be greater in exercising individuals (Wood and Haskell, 1979), and may contribute to the observed increase in HDL-C following exercise.

While exercise training in men has consistently led to increases in HDL-C (Lopez et al, 1974; Huttunen et al, 1979), a similar trend has not been demonstrated in physically active women (Moll et al, 1979; Wynne et al, 1980), including the present study. This disparity may, at least in part, be related to the effects of sex steroids on lipoprotein metabolism (Goldberg et al, 1985; Sherwin et al, 1987). HDL-C has exhibited a sensitivity to sex steroids (Tikkanen et al, 1982). Specifically, estrogens have been associated with elevated levels of HDL-C (Frey et al, 1983; Zonderland et al, 1986) and androgens have been associated with either increases (Mendoza et al, 1981; Webb et al, 1984) or decreases (Semmens et al, 1983) in this lipoprotein fraction. Based on these observations, researchers have speculated that exercise-induced changes in HDL-C may be mediated by changes in sex hormone concentrations, and that these hormone responses are gender-specific (Frey et al, 1983; Hazzard et al, 1984). A mechanism of action, however, has yet to be proposed.

Several dietary factors have been linked to changes in serum lipoprotein concentrations. Investigations into the effect of dietary cholesterol on serum cholesterol levels in humans have been numerous (Tan et al, 1980; Flaim et al, 1981; Beynen and Katan, 1985), due to the strong positive correlation between serum cholesterol level and risk of CHD (The Lipid Research Clinics Study, 1984). Despite this

research, the role of dietary cholesterol in the alteration of serum lipoproteins remains a topic of controversy (Connor and Connor, 1986). A number of human studies have shown that ingestion of saturated fat leads to an elevation of serum total cholesterol and LDL-C, whereas polyunsaturated fat produces the opposite effect (Shepherd et al, 1980; Ehnholm et al, 1984). An increased carbohydrate consumption has led to increases in TG and decreases in HDL-C (Gonen et al, 1981; Coulston et al, 1983), changes deemed unfavorable due to their association with an increased incidence of CHD.

Current dietary recommendations include suggestions for the public to decrease the intake of total fat (including saturated fat), cholesterol, salt and sugars, and to increase the intake of polyunsaturated fat and complex carbohydrates, including fibre (Committee on Diet and Cardiovascular Disease, 1976; Committee on Nutrition, 1978). These recommendations have been set forth in an effort to reduce the incidence of CHD in the general population. In a recent investigation by Hallfrisch and associates (1985), minimal modifications in the typical American diet were made to incorporate the current recommendations. Following 13 weeks of diet modification, plasma total cholesterol, LDL-C and VLDL-C were significantly reduced, while plasma TG and HDL-C levels remained unchanged in 53 healthy men and women. Thus, a diet which conforms to current dietary guidelines

appears to produce positive changes in the lipoprotein profiles of individuals.

In the present study, analysis of food records revealed no significant change in the diet composition of the subjects over 24 weeks. Total fat intake remained slightly above the recommended intake of 30-35% of total calories, while carbohydrate consumption was slightly less than the 50% recommendation. The lack of change in dietary habits, combined with an unaltered aerobic capacity, resulted in little improvement in the lipoprotein profiles of the subjects. The key to change may lie, not merely in the improvement of knowledge, but in the alteration of an individual's attitude toward a desired change (Matheny et al, 1987). Subjects in the current study may have increased their knowledge of the current dietary guidelines, but they seemingly did not alter their attitude toward the need for dietary change. Because all subjects were apparently healthy, the recommended changes in diet, leading to a decreased risk of CHD, may have been of little concern to them. Emphasis on the short- and long-term benefits of a modified fat diet may be necessary to elicit the desired behavioral change.

Chapter V

CONCLUSION

The results of the present study suggest that a 24-week program of dietary modification and aerobic exercise does not lead to favorable changes in the serum lipid profiles of adult sedentary women. Serum concentrations of TG, total cholesterol, VLDL-C, and LDL-C remained unchanged in the exercise group following the experiment. Serum levels of HDL-C decreased signigicantly in both the exercise and the control groups after 24 weeks, suggesting that seasonal variation might be responsible for this decline. Previous investigations in women have also failed to produce increases in HDL-C or decreases in TG. Conversely, studies in men have repeatedly shown an increase in HDL-C with exercise training.

An improvement in physical fitness level, previously associated with increases in HDL-C and decreases in TG, was not demonstrated in the present study. The intensity and frequency of exercise training may have been insufficient to produce an increase in aerobic capacity (VO_2 max), and thus any positive changes in serum lipoprotein levels. High initial level of fitness and a stable body weight may also have contributed to the lack of change in VO_2 max.

No significant change in the diet composition of the subjects was observed throughout the 24-week study. Diet counselling and instruction were based on current dietary recommendations, which have been shown to improve the lipoprotein profiles of individuals. It is suggested that the alteration of an individual's attitude might be a necessary step leading to dietary change.

Twenty-four weeks of dietary modification and aerobic exercise did not appear to produce positive changes in the lipoprotein profiles of adult women. Further investigations are needed to elucidate the metabolic changes linking diet, exercise and alterations in serum lipoproteins. Such research is warranted in light of the association between changes in lipoprotein levels and the risk of CHD development.

REFERENCES

Allain CC, Poon LS, Ghan CSG. 1974. Enzymatic determination of total serum cholesterol. Clin Chem. 20:470.

Altekruse EN, Wilmore JH. 1973. Changes in blood chemistries following a controlled exercise program. J Occ Med. 15:110.

American College of Sports Medicine. 1978. The recommended quantity and quality of exercise for developing and maintaining fitness in healthy adults. Med Sci Sports. 10:vii-x.

American College of Sports Medicine. 1986. Guidelines for Exercise Testing and Prescription. Lea and Febiger, Philadelphia.

American Heart Association. 1978. Committee on Nutrition. Circulation. 58:762A.

Anderson JW, Chen W, Sieling B. 1980. Hypolipidemic effect of high-carbohydrate, high-fiber diet. Metabolism. 29:551.

Aromma A, Bjorksten F, Eriksson AW, Maatela J, Kirjarinta M, Fellman J, Tammenin M. 1975. Serum cholesterol and triglyceride concentration of Finns and Finnish Laps. Acta Med Scand. 198:13.

Astrand I. 1960. Aerobic work capacity in men and women with special reference to age. Acta Physiol Scand. 49 (Suppl 169).

Astrand P-O, Ryhming I. 1954. A nomogram for calculation of aerobic capacity (physical fitness) from pulse rate during submaximal work. J Appl Physiol. 7:218.

Avogaro P, Gazzolato G, Bon GB. 1978. HDL-cholesterol, apolipoprotein A, and B - age and index body weight. Atherosclerosis. 31:85.

Beynen AC, Katan MB. 1985. Effect of egg yolk feeding on the concentration and composition of serum lipoproteins in man. Atherosclerosis. 54:157.

Blum CB, Levy RI, Eisenberg S, Hall M, Goebel RH, Berman M. 1977. High density lipoprotein metabolism in man. J Clin Invest. 60:795.

Brozek J, Grande F, Anderson JT. 1963. Densitometric analysis of body composition: revision of some quantitative assumptions. Ann NY Acad Sci. 110:113.

Cahalan D, Cisen IH, Crossley HM. 1969. American Drinking Practices. Monograph. Rutgers Centre of Alcohol Studies, New Brunswick, New Jersey.

Carew TE, Hayes SB, Koschinsky T, Steinberg D. 1976. A mechanism by which high-density lipoproteins may slow the atherogenic process. Lancet. i:1315.

Carlson LA, Bottiger LE, Ahfeldt PE. 1979. Risk factors for myocardial infarction in the Stockholm prospective study: a 14-year follow-up focusing on the role of plasma triglycerides and cholesterol. Acta Med Scand. 206:351.

Castelli WP, Doyle JT, Gordon T, Hames CG, Hjortland MC, Hulley SB, Kagan A, Zukel WJ. 1977. HDL cholesterol and other lipids in coronary heart disease. The cooperative lipoprotein phenotyping study. Circulation. 55:767.

Cauley JA, LaPorte RE, Kuller LH, Black-Sandler R, Bates M. 1982. Exogenous estrogen use in the menopause: relationship to high density lipoprotein cholesterol subfractions. Circulation. 66(suppl ii):237.

Cauley JA, Kriska AM, LaPorte RE, Sandler RB, Pambianco G. 1987. A two year randomized exercise trial in older women: effects on HDL-cholesterol. Atherosclerosis. 66:247.

Chenoweth W, Ullmann M, Simpson R, Leveille G. 1981. Influence of dietary cholesterol and fat on serum lipids in men. J Nutr. 111:2069.

Conner WE, Connor SL. 1986. Dietary cholesterol and fat and the prevention of coronary heart disease: Risks and benefits of nutritional change. In Diet and Prevention of Coronary Heart Disease and Cancer. Ed. B Hallgren. Raven Press, New York.

Coulston AM, Liu GC, Reaven GM. 1983. Plasma glucose, insulin and lipid responses to high carbohydrate, low fat diets in normal humans. Metabolism. 32:52.

Criqui MH, Wallace RB, Heiss G, Mishkel M, Schonfeld C, Jones G. 1980. Cigarette smoking and plasma high density lipoprotein cholesterol. (The Lipid Research Clinics Program Prevalence Study). Circulation. 62(suppl iv):70.

Crouse JR, Grundy SM. 1984. Effects of alcohol on plasma lipoproteins and cholesterol and triglyceride metabolism in man. J Lipid Res. 25:486.

Despres JP, Bouchard C, Savard R, Tremblay A, Allard C. 1985. Lack of relationship between changes in adiposity and plasma lipids following endurance training. Atherosclerosis. 54:135.

Dowdy DB, Cureton KJ, Duval HP. 1985. Effects of aerobic dance on physical work capacity, cardiovascular function and body composition of middle-aged women. Res Q. 56:227.

Durrington PN. 1980. High-density lipoprotein cholesterol: methods and clinical significance. CRC (Critical Reviews in Clinical Laboratory Science). 18:31.

Durstine JL, Pate RR, Sparling PB, Wilson GE, Senn MD, Bartoli WP. 1987. Lipid, lipoprotein, and iron status of elite women distance runners. Int J Sports Med. 8:119.

Ehnholm C, Huttunen JK, Pietinen P, Leino U, Mutanen M, Kostiainen E, Iacono J, Dougherty R, Puska P. 1984. Effect of a diet low in saturated fatty acids on plasma lipids, lipoproteins, and HDL subfractions. Arteriosclerosis. 4:265.

Enger SC, Herbjornsen K, Erikssen J, Fretland A. 1977. High density lipoproteins (HDL) and physical activity: The influence of physical exercise, age and smoking on HDL-cholesterol and on the HDL-c/total cholesterol ratio. Scand J Clin Lab Invest. 37:251.

Farrell PA, Barboriak J. 1980. The time course of alterations in plasma lipid and lipoprotein concentrations during eight weeks of endurance training. Atherosclerosis. 37:231.

Fisher EA, Blum CB, Zannis VI, Breslow JL. 1983. Independent effects of dietary saturated fat and cholesterol on plasma lipids, lipoproteins, and apolipoprotein E. J Lipid Res. 24:1039.

Flaim E, Ferreri LF, Forrest WT, Hill JE, Sanford JR. 1981. Plasma lipid and lipoprotein cholesterol concentrations in adult males consuming normal and high cholesterol diets under controlled conditions. Am J Clin Nutr. 34:1103.

Flynn MA, Nolph GB, Flynn TC, Kahrs R, Krause G. 1979. Effect of dietary egg on human serum cholesterol and triglycerides. Am J Clin Nutr. 32:1051.

Food and Nutrition Board, National Research Council. 1980. Toward healthful diets. National Academy of Sciences, Washington, D.C.

Fossati P, Lorenzo P. 1982. Serum triglycerides determined calorimetrically with an enzyme that produces hydrogen peroxide. Clin Chem. 28:2077.

Fox EL, Mathews DK. 1981. The Physiological Basis of Physical Education and Athletics. Saunders College Publishing, Philadelphia.

Frey MAB, Doerr BM, Laubach LL. 1982. Exercise does not change high-density lipoprotein cholesterol in women after 10 weeks of training. Metabolism. 31:1142.

Frey MAB, Doerr BM, Srivastava LS, Glueck CJ. 1983. Exercise training, sex hormones, and lipoprotein relationships in men. J Appl Physiol: Respirat Environ Exercise Physiol. 54:757.

Gaesser GA, Rich RG. 1984. Effects of high- and low-intensity exercise training on aerobic capacity and blood lipids. Med Sci Sports Exerc. 16:269.

Goldberg AC, Schonfeld G. 1985. Effects of diet on lipoprotein metabolism. Ann Rev Nutr. 5:195.

Goldberg RB, Rabin D, Alexander AN, Doelle GC, Getz GS. 1985. Suppression of plasma testosterone leads to an increase in serum total and high density lipoprotein cholesterol and apoproteins A-I and B. J Clin Endocrinol Metab. 60:203.

Gonen B, Patsch W, Kuisak I, Schonfeld G. 1981. The effects of short term feeding of a high carbohydrate diet on HDL subclasses in normal subjects. Metabolism. 30:1125.

Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. 1977. High density lipoprotein as a protective factor against coronary heart disease. Am J Med. 62:708.

Hallfrisch J, West S, Fisher C, Reiser S, Mertz W, Prather ES, Canary JJ. 1985. Modification of the United States' diet to effect changes in blood lipids and lipoprotein distribution. Atherosclerosis. 57:179.

Hartung GH, Foreyt JP, Mitchell RE, Vlasek I, Gotto AM. 1980. Relation of diet to high-density-lipoprotein cholesterol in middle-aged marathon runners, joggers, and inactive men. N Engl J Med. 302:357.

Haskell WL, Taylor HL, Wood PD, Schrott H, Heiss G. 1980. Strenuous physical activity, treadmill exercise test performance and plasma high density lipoprotein cholesterol. The Lipid Research Clinics Program Prevalence Study. Circulation. 62(suppl iv):53.

Haskell WL. 1984. The influence of exersise on the concentrations of triglyceride and cholesterol in human plasma. In Exercise and Sport Sciences Reviews, vol 12, 205-224. DC Heath Co., Lexington, MA.

Hazzard WR, Haffner SM, Kushwaha RS, Applebaum-Bowden D, Foster DM. 1984. Preliminary Report: Kinetic studies on the modulation of high-density lipoprotein, apolipoprotein, and subfraction metabolism by sex steroids in a postmenopausal woman. Metabolism. 33:779.

Health and Welfare Canada. 1986. Canadian Nutrient File.

Health and Welfare Canada. 1976. Committee on Diet and Cardiovascular Disease.

Hurley BF, Seals DR, Hagberg JM, Goldberg AC, Ostrove SM, Holloszy JO, Wiest WG, Goldberg AP. 1984. High-density-lipoprotein cholesterol in bodybuilders v powerlifters: Negative effects of androgen use. JAMA. 252:507.

Huttunen JK, Lansimies E, Voutilainen E, Ehnholm C, Hietanen E, Pentilla I, Sutonen O, Rauramaa R. 1979. Effect of moderate physical exercise on serum lipoproteins. A controlled clinical trial with special reference to serum high density lipoproteins. Circulation. 60:1220.

Jackson RL, Kashyap ML, Barnhart RL, Allen C, Hogg E, Glueck CJ. 1984. Influence of polyunsaturated and saturated fats on plasma lipids and lipoproteins in man. Am J Clin Nutr. 39:589.

Kuusi T, Ehnholm C, Huttunen JK, Kostiainen E, Pietinen P, Leino U, Uusitalo U, Nikkari T, Iacono JM, Puska P. 1985. Concentration and composition of serum lipoproteins during a low-fat diet at two levels of polyunsaturated fat. J Lipid Res. 26:360.

Lehtonen A, Viikari J. 1978. The effect of vigorous physical activity at work on serum lipids with special reference to serum high-density lipoprotein cholesterol. Acta Physiol Scand. 104:117.

Lennon DLF, Stratman FW, Shrago E, Nagle FJ, Hanson PG, Madden M, Spennetta T. 1983. Total cholesterol and HDL-cholesterol changes during acute, moderate-intensity exercise in men and women. Metabolism. 32:244.

Lewis B. 1983. Relation of high-density lipoproteins to coronary artery disease. Am J Cardiol. 52:5B.

Lewis S, Haskell WL, Wood PD, Manoogian N, Bailey JE, Pereira M. 1976. Effects of physical activity on weight reduction in obese middle-aged women. Am J Clin Nutr. 29:151.

Lindgren FT. 1975. Preparative ultracentrifugal laboratory procedures and suggestions for lipoprotein analysis. In Analysis of Lipids and Lipoproteins, 204-224. Ed. EG Perkins. American Oil Chemists' Society, Champaign, Illinois.

Lipid Research Clinics Program Prevalence Study. 1979. Plasma lipid distributions in selected North American populations. Circulation. 60:427.

Lipid Research Clinics Program. 1984. The Lipid Research Clinics Coronary Primary Prevention Trial Results. II. The relationship of reduction in incidence of coronary heart disease to cholesterol lowering. JAMA. 251:351.

Lipson LC, Bonow RO, Schaefer EJ, Brewer HB, Lindgren FT. 1980. Effects of exercise conditioning on plasma high density lipoproteins and other lipoproteins. Atherosclerosis. 37:529.

Lopez SA, Vial R, Balart L, Arroyave G. 1974. Effect of exercise and physical fitness on serum lipids and lipoproteins. Atherosclerosis. 20:1.

Mahley RW, Bersot TP, Innerarity TL, Lipson A, Margolis S. 1978. Alterations in human high-density lipoproteins, with or without increased plasma cholesterol, induced by diets high in cholesterol. Lancet. i:807.

Matheny RJ, Picciano MF, Birch L. 1987. Attitudinal and social influences on infant-feeding preference. J Nutr Educ. 19:21.

Mattson FH, Grundy SM. 1985. Comparison of effects of dietary saturated, monounsaturated, and polyunsaturated fatty acids on plasma lipids and lipoproteins in man. Lipid Res. 26:194.

McGowan MW, Artiss JD, Strandberg DR, Zak B. 1983. A peroxide coupled method for the colorimetric determination of serum triglycerides. Clin Chem. 29:538.

Mendoza SG, Osuna A, Zerpa A, Gartside PS, Glueck CJ. 1981. Hypertriglyceridemia and hypoalphalipoproteinemia in azoospermic and oligospermic young men: relationships of endogenous testosterone to triglyceride and high density lipoprotein cholesterol metabolism. Metabolism. 30:481.

Metropolitan Life Insurance Company Height-Weight Data, Revised 1983.

Miller GH, Miller NE. 1975. Plasma high density lipoprotein concentration and development of ischemic heart disease. Lancet. i:16.

Miller NE. 1978. The evidence for the antiatherogenicity of high density lipoprotein in man. Lipids. 13:915.

Moll ME, Williams RS, Lester RM, Quarfordt SH, Wallace AG. 1979. Cholesterol metabolism in non-obese women: Failure of physical conditioning to alter levels of high density lipoprotein cholesterol. Atherosclerosis. 34:159.

Moore CE, Hartung GH, Mitchell RE, Kappus CM, Hinderlitter J. 1983. The relationship of exercise and diet on high-density lipoprotein cholesterol levels in women. Metabolism. 32:189.

Nikkala EA, Taskinen MR, Rehunen S, Harkonen M. 1978. Lipoprotein lipase activity in adipose tissue and skeletal muscle of runners: Relation to serum lipoproteins. Metabolism. 27:1661.

Nilsson-Ehle P, Garfinkel AS, Schotz MC. 1980. Lipolytic enzymes and plasma lipoprotein metabolism. Ann Rev Biochem. 49:667.

Nordoy A, Aakvaag A, Thelle D. 1979. Sex hormones and high density lipoproteins in healthy males. Atherosclerosis. 34:431.

Nye ER, Carlson K, Kirstein P, Rossner S. 1981. Changes in high density lipoprotein subfractions and other lipoproteins induced by exercise. Clin Chim Acta. 113:51.

Paffenbarger RS, Wing AL, Hyde RT. 1978. Physical activity as an index of heart attack risk in college alumni. Am J Epidemiol. 108:161.

Pietinen P, Huttunen JK. 1987. Dietary determinants of plasma high-density lipoprotein cholesterol. Am Heart J. 113:620.

Pyorala K. 1987. Dietary cholesterol in relation to plasma cholesterol and coronary heart disease. Am J Clin Nutr. 45:1176.

Reiser S, Hallfrisch J, Michaelis O, Lazar F, Martin R, Prather E. 1978. Isocaloric exchange of dietary starch and sucrose in humans. I. Effects on levels of fasting blood lipids. Am J Clin Nutr. 32:1659.

Rifai N, King ME, DeMeersman R, Merrill JR, Holly RG. 1987. Apolipoprotein and lipoprotein cholesterol concentrations in trained and sedentary males. Clin Chim Acta. 163:113.

Schaefer EJ, Levy RI, Ernst ND, Van Sant FD, Brewer HB Jr. 1981. The effects of low cholesterol, high polyunsaturated fat, and low fat diets on plasma lipid and lipoprotein cholesterol levels in normal and hypercholesterolemic subjects. Am J Clin Nutr. 34:1758.

Schaefer EJ, Foster DM, Zech LA, Lindgren FT, Brewer HB, Levy RI. 1983. The effects of estrogen administration on plasma lipoprotein metabolism in premenopausal females. J Clin Endocrinol Metab. 57:262.

Schonfeld G, Weidman SW, Witztum JL, Bowen RM. 1976. Alterations in levels and inter-relations of plasma apolipoproteins induced by diet. Metabolism. 25:261.

Schwandt P, Janetschek P, Weisweiler P. 1982. High density lipoproteins unaffected by dietary fat modification. Atherosclerosis. 44:9.

Semmens J, Rouse I, Berlin LJ, Masarei JRL. 1983. Relationship of plasma HDL-cholesterol to testosterone, estradiol and sex-hormone-binding globulin levels in men and women. Metabolism. 32:428.

Shepherd J, Packard CJ, Patsch J, Gotto CA, Taunton OD. 1978. Effects of dietary polyunsaturated and saturated fat on the properties of high density lipoproteins and the metabolism of apolipoprotein A-I. J Clin Invest. 61:1582.

Shepherd J, Packard CJ, Grundy SM, Yeshurun D, Gotto AM, Taunton OD. 1980. Effects of saturated and polyunsaturated fat diets on the chemical composition and metabolism of low density lipoproteins in man. J Lipid Res. 21:91.

Sherwin BB, Gelfand MM, Schucher R, Gabor J. 1987. Postmenopausal estrogen and androgen replacement and lipoprotein lipid concentrations. Am J Obstet Gynecol. 156:414.

Smith MP, Mendez J, Druckenmiller M, Kris-Etherton PM. 1982. Exercise intensity, dietary intake, and high density lipoprotein cholesterol in young female competitive swimmers. Am J Clin Nutr. 36:251.

Solyom A. 1972. Effect of androgens and serum lipids and lipoproteins. Lipids. 7:100.

Stamford BA, Matter S, Fell RD, Sady S, Papanek P, Cresanta M. 1984a. Cigarette smoking, exercise and high density lipoprotein cholesterol. Atherosclerosis. 52:73.

Stamford BA, Matter S, Fell RD, Sady S, Cresanta MK, Papanek P. 1984b. Cigarette smoking, physical activity, and alcohol consumption: relationship to blood lipids and lipoproteins in premenopausal females. Metabolism. 33:585.

Stamler J, Wentworth D, Neaton JD. 1986. Is the relationship between serum cholesterol and risk of premature death from coronary heart disease continuous and graded? (Findings in 356,222 primary screenees of the Multiple Risk Factor Intervention Trial [MRFIT]). JAMA. 256:2823.

Stanbury JB, Wyngaarden JB, Fredrickson DS, Goldstein JL, Brown MS. 1983. The Metabolic Basis of Inherited Disease, 591-592. McGraw-Hill Book Company, New York.

Standardized Test of Fitness Operations Manual. 1986. Fitness and Amateur Sport, Ottawa (Third edition).

Stubbe I, Hansson P, Gustafson A, Nilsson-Ehle P. 1983. Plasma lipoproteins and lipolytic enzyme activities during endurance training in sedentary men: Changes in high-density lipoprotein subfractions and composition. Metabolism. 32:1120.

Svedenhag J, Lithell H, Juhlin-Dannfelt A, Henriksson J. 1983. Increase in skeletal muscle lipoprotein lipase following endurance training in man. Atherosclerosis. 49:203.

Taggart HM, Applebaum-Bowden D, Haffner S, Warnick GR, Cheung MC, Albers JJ, Chestnut CH, Hazzard WR. 1982. Reduction in high density lipoproteins by anabolic steroid (stanozolol) therapy for postmenopausal osteoporosis. Metabolism. 31:1147.

Tan MH, Dickenson MA, Alders JJ, Havels RJ, Cheung MC, Vigne J. 1980. The effect of a high cholesterol and saturated fat diet on serum high density lipoprotein cholesterol, apoprotein A-I, and apoprotein E levels in normolipidemic humans. Am J Clin Nutr. 33:2559.

Thompson P, Bortz WM. 1978. Significance of high-density lipoprotein cholesterol. J Am Geriatr Soc. 26:440.

Thompson GR, Trayner I, Danford W, Mark F, Harrison R. 1980. Plasma lipids and lipoproteins after modified fat diet. Lancet. ii:421.

Tikkanen MJ, Nikkala EA, Kuusi T, Sipinen S. 1982. High density lipoprotein-2 and hepatic lipase: reciprocal changes produced by estrogen and norgestrel. J Clin Endocrinol Metab. 54:1113.

Vodak PA, Wood PD, Haskell WL, Williams PT. 1980. HDL-cholesterol and other plasma lipid and lipoprotein concentrations in middle-aged male and female tennis players. Metabolism. 29:745.

Wallentin L, Sundin B. 1985. HDL_2 and HDL_3 lipid levels in coronary artery disease. Atherosclerosis. 59:131.

Webb OL, Laskarzewski PM, Glueck CJ. 1984. Severe depression of high-density lipoprotein cholesterol levels in weight lifters and body builders by self-administered exogenous testosterone and anabolic-androgenic steroids. Metabolism. 33:971.

Weisweiler P, Drosner M, Janetschek P, Schwandt P. 1983. Changes in very low and low density lipoproteins with dietary fat modification. Atherosclerosis. 44:9.

Williams PT, Wood PD, Krauss RM, Haskell WL, Vranizan KM, Blair SN, Terry R, Farquhar JW. 1983. Does weight loss cause the exercise-induced increase in plasma high density lipoproteins? Atherosclerosis. 47:173.

Wilmore, JH. 1983. Body composition in sport and exercise: directions for future research. Med Sci Sports Exer. 15:21.

Witztum J, Schonfeld G. 1979. High density lipoproteins. Diabetes. 28:326.

Wood PD, Haskell W, Klein H, Lewis S, Stern MP, Farquhar JW. 1976. The distribution of plasma lipoprotein in middle aged runners. Metabolism. 25:1249.

Wood PD, Haskell WL. 1979. The effect of exercise on plasma high density lipoproteins. Lipids. 14:417.

Wood PD, Haskell WL, Blair SN, Williams PT, Krauss RM, Lindgren FT, Albers JJ, Ho PH, Farquhar JW. 1983. Increased exercise level and plasma lipoprotein concentrations: a one-year randomized, controlled study in sedentary, middle-aged men. Metabolism. 32:31.

Wood PD, Terry RB, Haskell WL. 1985. Metabolism of substrates: diet, lipoprotein metabolism, and exercise. Fed Proc. 44:358.

Wynne TP, Bassett-Frey MA, Laubach LL, Glueck CJ. 1980. Effect of a controlled exercise program on serum lipoprotein levels in women on oral contraceptives. Metabolism. 29:1267.

Zonderland ML, Erich WBM, Peltenburg AL, Bernink MJE, Havekes L, Thijssen JHH. 1986. Plasma lipoprotein profile in relation to sex hormones in premenarcheal athletes. Int J Sports Med. 7:241.

Appendix A

SUBJECT REQUIREMENTS

Subjects were selected for the study based on the following:

1. Body weight - a subject must be within the normal weight range for height, according to the Metropolitan Life Insurance Company Height-Weight Tables (1983).

2. Physical fitness level - a subject must not have exercised regularly during the past 6 months.

3. Serum lipid levels - a subject must have fasting total TG and total cholesterol within normal lipid values for age (The Lipid Research Clinics, 1980).

4. Smoking habits - a subject must not have smoked for the past 12 months.

5. Alcohol consumption - a subject must have light to moderate alcohol intake (Cahaban et al., 1969).

6. Oral contraceptives/pregnancy - a subject must not have taken oral contraceptives or been pregnant in the last 12 months.

7. Other - a subject must be pre-menopausal and have not had a hysterectomy nor have been on hormone therapy.

Appendix B

INFORMED CONSENT AND RELEASE FOR RESEARCH STUDY

Explanation of Tests

The following data will be collected from each subject during the study:

1. Height.
2. Weight.
3. Densiometry - determination of body density by submersion in a tank.
4. Five skinfold measurements.
5. Twelve minute submaximal bicycle test - determination of cardiovascular endurance.
6. Venous blood samples - determination of serum lipid and lipoprotein levels.
7. Three-day diet record.

During a 24-week period you will have the above tests three times. The test battery is administered by trained personnel who will explain each test item in detail. Results will be confidential.

Risks

While it is highly unlikely that a subject would be injured or take ill during the tests, emergency procedures would be implemented immediately. I have read the above and understand that if any test causes pain or discomfort, I am obliged to immediately inform the tester. I may stop or delay any further testing if I so desire and the testing may be terminated by the tester upon observation of any symptoms of distress or abnormal response.

Inquiries

We invite your questions about the procedures to be used in the test. If you have any doubts as to what to expect, please ask us for further information and explanation.

Waiver and Release

I understand that participating in this research study, I do so at my own risk and I undertake to indemnify the University of Manitoba against any and all liability that may arise as a result of my participating.

_____ _____

Signature Date

_____ _____

Witness Date

Note: This form must be witnessed at the time of signing and the witness must be of the age of majority.

Appendix C

SEPARATION OF LIPOPROTEINS

1. Preparation of serum (modified method of Lindgren (1975)).

   a) Each serum sample required 2 tubes:

      i) "Run 1" (density 1.0063 g/ml) = 0.6 ml serum + 1.8 ml Na Cl (density 1.0063 g/ml)

      ii) "Run 2" (density 1.0630 g/ml) = 0.6 ml serum + 1.8 ml Na Cl (density 1.0819 g/ml)

   b) All tubes were mixed on a Vortex mixer and placed in the ultracentrifuge head.

2. Separation of lipoprotein fractions

   a) Lipoproteins were fractionated by ultracentrifugation (rotor type 40.3) at 18°C and 34,000 rpm (calculated 103,578G) for 18 hours (Beckman L5-50B ultracentrifuge).

   b) Ultracentrifugation in a medium of density 1.0063 g/ml separated the VLDL (top layer) from the HDL+LDL (bottom layer). Ultracentrifugation in a medium of density 1.0630 g/ml separated the VLDL+LDL (top layer) from the HDL (bottom layer). Only the bottom layers, consisting of: i) HDL and LDL, and
      ii) HDL, were retained.

   c) Using a syringe, 1.5 ml was removed in one smooth continuous motion from the top fraction of each tube. Care was taken to not disturb the bottom fraction, or touch the sides of the tube. The bottom fraction was then carefully transferred to a 1.0 ml volumetric tube with a Pasteur pipette. Tubes were rinsed several times with 3-4 drops of distilled water, and the rinsings added to the volumetric tube to bring the volume up to 1.0 ml.

Appendix D

CHOLESTEROL DETERMINATION

1. Principle of method - cholesterol was determined enzymatically using a colorimetric assay kit (Fisher Diagnostics) based on the method of Allain et al (1974). The principles of the reactions are summarized below:

a) cholesterol esters $\xrightleftharpoons{\text{cholesterol ester hydrolyzate}}$ cholesterol + fatty acids

b) cholesterol + 02 $\xrightleftharpoons{\text{cholesterol oxidase}}$ cholesten-3-one + H202

c) 2 H202 + 4-aminoantipyrene + phenol $\xrightleftharpoons{\text{peroxidase}}$ quinoneimine* + 4 H20

*a chromagen with maximum absorbance at 505 nm.

2. Procedure

   a) 0.025 ml each of distilled water, of cholesterol standard and of samples to be assayed were pipetted into separate 4 ml test tubes.

   b) 1.0 ml of cholesterol reagent was added to each tube and all tubes were mixed in a Vortex mixer.

   c) All tubes were incubated in a 37°C waterbath for 15 minutes.

   d) 1.5 ml of 0.89% saline solution was added to each tube and all tubes were mixed on a Vortex mixer.

   e) Absorbance of standard and samples were read at 505 nm on a spectrophotometer (Model SP6-300), employing the distilled water sample as the blank.

   f) Mean absorbance for the duplicate measurements of each serum cholesterol sample were determined, and compared with the absorbance of a cholesterol standard of known concentration.

Appendix E

TRIGLYCERIDE DETERMINATION

1. Principle of method - Triglyceride was determined enzymatically using a colorimetric assay kit (Fisher Diagnostics), based on the methods of Fossati and Lorenzo (1982) and McGowan et al (1983). The principles of the reactions are summarized below:

a) triglycerides $\xrightleftharpoons{\text{lipase}}$ glycerol + fatty acids

b) glycerol + ATP $\xrightleftharpoons{\text{glycerol kinase}}$ glycerol-1-phosphate + ADP

c) glycerol-1-phosphate + O2 $\xrightarrow{\text{glycerol phosphate oxidase}}$ H2O2 + dihydroxyacetone phosphate.

d) H2O2 + 4-aminoantipyrene + 3,5-dichloro-2-hydroxy-benzenesulfonic acid

$\xrightleftharpoons{\text{peroxidase}}$ quinoneimine* + HCl + 2 H2O

* a chromagen with maximum absorbance at 515 nm.

2. Procedure

a) 0.020 ml each of distilled water, of triglyceride standard and of samples to be assayed were pipetted into separate 4 ml test tubes.

b) 1.0 ml of triglyceride color reagent was added to each tube and all tubes were mixed on a Vortex mixer.

c) All tubes were incubated in a 37^{o}C waterbath for 10 minutes.

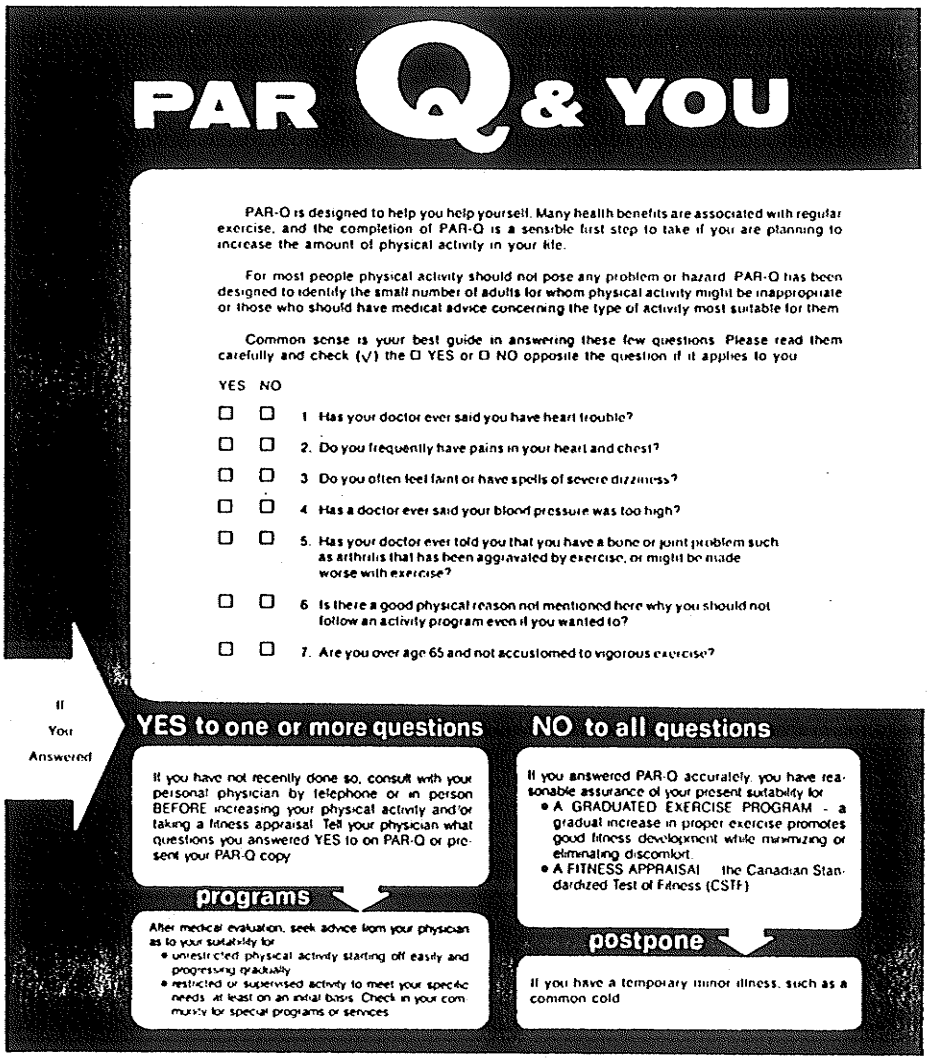
d) 1.5 ml of distilled water was added to each tube and all tubes were mixed on a Vortex mixer.

e) Absorbance of standard and samples were read at 515 nm on a spectrophotometer (Model SP6-300), employing the distilled water sample as the blank.

f) Mean absorbance for the duplicate measurements of each serum triglyceride sample were determined, and compared with the absorbance of a triglyceride standard of known concentration.

Appendix F

PHYSICAL ACTIVITY READINESS QUESTIONNAIRE (PAR-Q)



PAR Q & YOU

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advice concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read them carefully and check (√) the ☐ YES or ☐ NO opposite the question if it applies to you.

YES	NO	
☐	☐	1. Has your doctor ever said you have heart trouble?
☐	☐	2. Do you frequently have pains in your heart and chest?
☐	☐	3. Do you often feel faint or have spells of severe dizziness?
☐	☐	4. Has a doctor ever said your blood pressure was too high?
☐	☐	5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?
☐	☐	6. Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
☐	☐	7. Are you over age 65 and not accustomed to vigorous exercise?

If You Answered

YES to one or more questions

If you have not recently done so, consult with your personal physician by telephone or in person BEFORE increasing your physical activity and/or taking a fitness appraisal. Tell your physician what questions you answered YES to on PAR-Q or present your PAR-Q copy.

programs

After medical evaluation, seek advice from your physician as to your suitability for:
- unrestricted physical activity starting off easily and progressing gradually
- restricted or supervised activity to meet your specific needs, at least on an initial basis. Check in your community for special programs or services

NO to all questions

If you answered PAR-Q accurately, you have reasonable assurance of your present suitability for:
- A GRADUATED EXERCISE PROGRAM - a gradual increase in proper exercise promotes good fitness development while maximizing or eliminating discomfort.
- A FITNESS APPRAISAL - the Canadian Standardized Test of Fitness (CSTF)

postpone

If you have a temporary minor illness, such as a common cold

[illegible] Developed by the British Columbia Ministry of Health. Conceptualized and critiqued by the Multidisciplinary Advisory Board on Exercise (MABE).
[illegible] Translation, reproduction and use in its entirety is encouraged. Modifications by written permission only. Not to be used for commercial advertising in order to solicit business from the public.
[illegible] PAR-Q Validation Report. British Columbia Ministry of Health, 1978.
[illegible] Produced by the British Columbia Ministry of Health and the Department of National Health & Welfare.

Appendix G

LIFESTYLE QUESTIONNAIRE

NAME OF PARTICIPANT _____ DATE _____

1. Indicate the physical activities in which you have participated over the last month during your leisure time.

	FREQUENCY: Number of occasions over the last month	DURATION: Average number of activity minutes spent on each occasion				INTENSITY		
		1–15	15–30	31–60	60+	Slight change from normal state: LIGHT	Some perspiration, faster than normal breathing: MEDIUM	Heavy perspiration, heavy breathing: HEAVY
Walking for Exercise	____	☐	☐	☐	☐	☐	☐	☐
Bicycling	____	☐	☐	☐	☐	☐	☐	☐
Swimming	____	☐	☐	☐	☐	☐	☐	☐
Jogging/Running	____	☐	☐	☐	☐	☐	☐	☐
Gardening	____	☐	☐	☐	☐	☐	☐	☐
Home Exercises	____	☐	☐	☐	☐	☐	☐	☐
Ice Skating	____	☐	☐	☐	☐	☐	☐	☐
Cross Country Skiing	____	☐	☐	☐	☐	☐	☐	☐
Tennis	____	☐	☐	☐	☐	☐	☐	☐
Golf	____	☐	☐	☐	☐	☐	☐	☐
Popular Dance	____	☐	☐	☐	☐	☐	☐	☐
Baseball/Softball	____	☐	☐	☐	☐	☐	☐	☐
Alpine Skiing	____	☐	☐	☐	☐	☐	☐	☐
Ice Hockey	____	☐	☐	☐	☐	☐	☐	☐
Bowling	____	☐	☐	☐	☐	☐	☐	☐
Exercise Classes	____	☐	☐	☐	☐	☐	☐	☐
Racquetball	____	☐	☐	☐	☐	☐	☐	☐
Curling	____	☐	☐	☐	☐	☐	☐	☐
Others – Please specify:								
_____	____	☐	☐	☐	☐	☐	☐	☐
_____	____	☐	☐	☐	☐	☐	☐	☐
_____	____	☐	☐	☐	☐	☐	☐	☐

Appendix H

DIET RECORD INSTRUCTIONS

Directions for Completing the Three-Day Food Record

- Please use the attached pages to record your food and beverage intake for the days indicated.
- It is important that you record everything you eat and drink, at home and away from home.
- Describe each food item and the amount eaten. Some suggested ways of measuring foods are included on the next page.
- Indicate if the food was eaten raw or cooked (e.g. fried, boiled, baked) and how it was served (e.g. with gravy, sauces, dressings).
- Do not change your eating pattern during the three days.
- Record any vitamin and mineral supplement you consume.

Suggested Way of Measuring Foods

Food	Measurement
- Milk, etc. (whole, 2%, skim, in tea, coffee, on cereal)	- cups, tablespoons, teaspoons or ml.
- Cereals (dry, cooked, presweetened)	- cups tablespoons or ml.
- Potatoes (mashed, boiled fried, chips)	- cups, small or large size, number of fries.
- Bread (white, brown, whole wheat, rye)	- slices, large or small loaf.
- Biscuits, rolls, buns	- number, size and type.
- Meat	- slices, ounces, dimensions.
- Fruit	- number and size, or cups.
- Vegetables	- cups or number, eg. 1 carrot

Appendix H continued...

Food	Measurement
- Sugar	- teaspoons or tablespoons or ml.
- Condiments (jam, jelly, ketsup, etc.)	- tablespoons or teaspoons, ml.
- Sweets (candies, chocolate)	- number of pieces or size of package.
- Beverages (soda pop, alcoholic beverages, juices)	- cups or ounces, ml. (low calorie produce)

Appendix I

DIETARY RECOMMENDATIONS FOR CANADIANS

1. The consumption of a nutritionally adequate diet, as outlined in Canada's Food Guide.

2. A reduction in calories from fat to 30 to 35% of total calories, mainly as a decrease in saturated fat.

3. A partial substitution of polyunsaturated for saturated fat.

4. The consumption of a diet which contains less alcohol, salt and refined sugars, and which emphasizes more whole grain products, fruits and vegetables.

5. The prevention and control of obesity through reducing excess calories and increasing physical activity. Precautions should be taken that no deficiency of vitamins and minerals occurs when total calories are reduced.

NOTE: These recommendations should not be considered as a replacement for specific or therapeutic diets prescribed by a physician for treatment of a particular disease or condition.

SOURCE: Report of the Committee on Diet and Cardiovascular Disease (Canada: Department of Health and Welfare, 1976), pp. 81-82.

Appendix J

DIETARY PRINCIPLES AND GUIDELINES

I. BASIC PRINCIPLES FOR LOWERING BLOOD CHOLESTEROL (through diet)

1. Decrease total fat intake, especially saturated fat, as foods high in saturated fat tend to elevate the cholesterol concentration in the blood.

   Foods of animal origin are high in saturated fat. Therefore, decrease intake of such foods as: meat, lard, butter and whole milk products.

2. Decrease cholesterol intake. Cholesterol is found in foods of animal origin, eg: eggs, organ meats.

3. Substitute some saturated fat in the diet with polyunsaturated fat. Polyunsaturated fat tends to decrease blood cholesterol levels.

   Polyunsaturated fat is found in vegetable oils such as: safflower, soybean, sunflower, corn, cottonseed and sesame seed; and, in margarines made from these oils.

4. Increase the intake of complex carbohydrates, for example, fresh fruits and vegetables, and whole grain products (breads and cereals).

Appendix J continued ...

II. GUIDELINES: to control the amount and type of fat in the diet.

1. Use fish, chicken, turkey and veal more often than beef, lamb, pork and ham.

2. Choose lean cuts of meat; trim off all visible fat; discard fat which cooks out of meat.

3. Limit the use of organ meats, and luncheon/variety meats such as bacon, sausage, salami, frankfurters, liverwurst.

4. Avoid foods which are deep-fried (except, occasionally, those fried in polyunsaturated oils).

5. Use cooking methods which help to remove fat, such as baking, oiling, broiling, poaching or roasting.

6. Remove skin from poultry before cooking.

7. Refrigerate soup stocks, pan juices and casseroles; fat will solidify and can then be easily removed.

8. Limit intake of high-fat snack foods such as potato chips, French fries, chocolate and coconut.

9. Limit intake of breads, pastries and desserts containing animal fats, (such as lard), dried egg and/or whole milk; eg: commercial rolls and biscuits, doughnuts, pies, cakes, cookies.

10. Decrease the use of whole milk, cream, cream cheese, high-fat cheeses and ice cream.

11. Increase the use of skim or 2% milk, low-fat cottage cheese, low-fat yogurt and low-fat cheeses (such as farmers cheese; also, parmesan and mozzarella).

12. Use lipid vegetable oils and margarines, rich in polyunsaturated fats, instead of butter, shortening or lard.

13. Choose margarines which: a) list liquid oil as the first ingredient, followed by one or more partially hydrogenated vegetable oils; b) contain twice as much polyunsaturated as saturated fat.

14. Read labels when buying packaged foods. Keep in mind that ingredients must be listed in descending order of their content in the product. For example, a label which lists "Gravy, beef, peas, carrots" contains more gravy than anything else.

Appendix K

SAMPLE MENU

Fat-controlled Diet
(1800 Kilocalories per Day)

I. Total daily food exchanges (exchange = serving):

Meat, fish, poultry	2
Breads, cereals	8
Fruit	4
Vegetables	3
Milk	2
Fat	6

II.

	Daily Exchanges, Per Meal	Sample Menu
Breakfast	1 fruit	3/4 c. orange juice, unsweetened
	2 bread	1 c. bran flakes
	½ milk	½ c. skim milk
	1 fat	1 sl. rye bread, toasted
		1 tsp. margarine
		1 tsp. marmalade
		coffee or tea
Mid-morning Snack	1 free beverage	coffee
	1 bread	1 bran muffin
Lunch	1 milk	1 c. skim milk
	2 bread	2 sl. wholewheat bread, ½ c. tuna in water
	2 fat	1 tsp. margarine
	1 meat	1 tsp. mayonnaise
	1 fruit	½ c. tuna in water
	1 vegetable	2 lettuce leaves
		1 med. carrot
		1 apple.
Mid-afternoon Snack	1 fruit	1 sm banana
	1 bread	2 graham crackers
Supper	1 meat	1 sm. chicken breast, broiled
	1 bread	½ c. rice
	2 vegetables	1 c. lettuce
	3 fat	1 med. tomato
	1 fruit	1 T. salad dressing
		½ med. cantaloupe
		coffee or tea
Evening Snack	½ milk	1 oz. low-fat cheese
	1 bread	2 soda crackers
	1 free beverage	tea

III. Food Exchange List (some suggestions for substitutions):

Meat, Fish, Poultry, (1 serving = 3 oz. = approx. 150-200 Kcals):

lean ground beef	
lean roast beef	
meat <u>without</u> skin of chicken, turkey, cornish hen	
fish, any fresh or frozen (cod, halibut, flounder, etc.)	
tuna, canned, in water	½ cup
salmon, canned	½ cup
uncreamed cottage cheese	½ cup
dried peas or beans	1 cup
cheese, < 5% butterfat	3 oz.

Bread, Cereals (1 serving = approx. 70 Kcals):

bread, white	1 slice
bagel, small	½
English muffin, small	½
plain bread roll	1
ready-to-eat, unsweetened cereal	1 cup
cooked cereal	½ cup
pasta, cooked - spaghetti	½ cup
macaroni	½ cup
sweet roll, bun	½

III. Food Exchange List (some suggestions for substitutions): cont'd.

Fruit (1 serving = approx. 40 Kcals):

apple juice	1/3 cup
applesauce, unsweetened	½ cup
blueberries	½ cup
grapefruit	½
grapefruit juice	½ cup
orange	1 sm.
peach	1 med.
pear	1 sm.
pineapple	1/3 cup
raisins	2 T.

Vegetables (1 serving, raw or cooked = ½ cup = approx. 25 Kcals):

asparagus
beans, green string
beets
broccoli
brussel sprouts
cabbage
carrots
cauliflower
mushrooms
onions
radish
tomatoes
turnips
zucchini
vegetable juice, cocktail
tomato juice
(corn - ½ cup)
(peas - ½ cup)

The following vegetables can be used as desired, raw:

celery	escarole
chinese cabbage	lettuce
cucumbers	watercress
endive	

Milk (1 serving = approx. 80 Kcals):

skim milk	1 cup
powdered milk, nonfat, dry	1/3 cup
buttermilk, from skim milk	1 cup
yogurt, plain, from skim milk	1 cup

Fat (1 serving = approx. 35 Kcals);

margarine, soft, tub or stick (made from corn, cottonseed, soy, sunflower or safflower oil only)	1 tsp
oil (made from same as above)	1 tsp.
walnuts, small	6
mayonnaise, salad dressing	1 tsp.

Free Beverages - the following beverages can be used as desired:

coffee, black
tea, black
diet drinks/diet pop.