INFLUENCE OF DIET AND EXERCISE INTENSITY ON SERUM LIPIDS AND LIPOPROTEINS IN YOUNG FEMALE RUNNERS

by

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B.S., New Mexico State University, 1983

A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

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KANSAS STATE UNIVERSITY
Manhattan, Kansas

1985

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TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

Cardiovascular disease (CVD) is one of the leading causes of death in the United States. The underlying cause of this disease is generally accepted as atherosclerosis, a process in which fatty deposits containing cholesterol thicken the walls and narrow the diameters of arteries. Available evidence suggests that the primary risk factors for CVD are smoking, hypertension, elevated blood cholesterol, and diabetes. Other risk factors include obesity, physical inactivity, personality patterns related to stress, genetic predisposition, and oral contraceptive use (26).

Attention has been focused on the potential contribution of physical activity to lowering the incidence of CVD. The basis for this concept hinges on beneficial alterations in blood lipids and lipoproteins that occur secondary to increased physical activity. An exercise program may significantly raise the high-density lipoprotein cholesterol (HDL-C) to low-density lipoprotein cholesterol (LDL-C) ratio, with or without changes in total cholesterol and triglycerides (48). These changes are desirable, as LDL may promote atheroma formation via its capacity for infiltrating the arterial intima (76), while HDL-C is believed to protect against atheroma formation by either transporting cholesterol to the liver for catabolism and excretion, or by inhibiting LDL-C uptake at extrahepatic tissues (19).

The effect of physical activity on lipid metabolism has been studied by a number of workers who used various exercise protocols and analytical methods. However, until very recently, few studies included consideration of variables such as diet, smoking, and adiposity.

Studies of physical activity and blood lipids in women are much less abundant in the literature than those of men, and require the consideration of additional variables such as oral contraceptive use and the menstrual cycle. Although the incidence of CVD in women is relatively low, future trends should be watched, as smoking and oral contraceptive use are on the rise (34).

The purpose of this research was to investigate the effects of diet and a consistent running program on serum lipid and lipoprotein-cholesterol levels in young women.

REVIEW OF LITERATURE

INFLUENCE OF DIET ON BLOOD LIPIDS.

Many dietary factors have an impact on blood lipids, including cholesterol, fat, carbohydrate, fiber, and alcohol. Advocates of the diet-heart theory believe that increased lipid levels in the blood are associated with dietary intake of lipids. As Americans consume approximately 10 billion pounds of visible fat per year (42) and have a high incidence of cardiovascular disease (26), various recommendations have been made concerning the reduction of dietary fat. Generally, cholesterol, total and saturated fat, and carbohydrate cause adverse alterations in blood lipids and/or lipoproteins, whereas polyunsaturated fat, fiber, and alcohol exert beneficial influences on these parameters.

Cholesterol intake. The effect of dietary cholesterol on blood lipids and lipoproteins is difficult to assess due to the impracticality of isolating the effect of cholesterol from that of total fat consumption or the ratio (P/S) of polyunsaturated fatty acids (PUFA) to saturated fatty acids (SFA). A number of dietary intervention studies have supported the theory that

alterations in cholesterol intake may alter the amount and distribution of blood total cholesterol (TC).

Schaefer et al. (60) found that a low cholesterol diet (250-300 mg/day) nonsignificantly lowered TC, LDL-C, and HDL-C levels by 5.9, 5.6, and 6.3%, respectively, compared to a normal hospital ad libitum diet in 8 male and 3 female subjects. No change in the LDL/HDL ratio, and slight, but nonsignificant decreases in very low-density lipoprotein cholesterol (VLDL-C) and triglycerides (TG) also were observed.

The removal of eggs from the diets of 44 male and female subjects by Bronsgeest-Schoute et al. (9) reduced cholesterol intake from 742 to 264 mg/day. This alteration resulted in a drop ($P \leq 0.05$) in serum TC from 244.2 to 218.8 mg/dL. That study also indicated that a highly variable response to dietary cholesterol may exist in a human population, and that the effects of dietary changes in a free living population are much smaller than in populations under controlled conditions.

Nonsignificant increases in TC and LDL-C were noted by Buzzard et al. (10) when daily cholesterol consumption was increased (addition of eggs) from 412 to 975 mg/day in 10 male subjects. HDL-C levels and TC/HDL-C remained relatively unchanged. Intakes of 400 to 800 mg/day cholesterol have been reported to cause "saturation" and increments above this amount only minorly affect TC levels (44). This may explain why the rise from 412 to 975 mg/day cholesterol failed to cause a dramatic increase in TC in the study by Buzzard et al. (10).

The examination of various population groups has led to even more inconsistent findings than the dietary intervention studies. Results from the Framingham Study (13) indicated that egg consumption was unrelated to serum TC; however, cholesterol from other sources was not considered. In

the Lipid Research Clinic's Program Prevalence Study (LRCPPS) there was a positive association ($P \leq 0.05$) between dietary cholesterol and HDL-C levels in women not taking gonadal hormones, but this relationship did not exist for other women or for men (15). Liebman and Bazzare (45) found no significant relationships between dietary cholesterol intake and plasma lipids and lipoproteins in 54 male vegetarian and nonvegetarian subjects.

Degree of fat saturation. Many studies have indicated that raising the P/S ratio of the diet resulted in lower blood cholesterol levels. And, it is generally believed that the hypocholesterolemic effect of polyunsaturated fat is due mainly to a decrease in the LDL-C fraction (33).

Studies of the effect of PUFA on HDL-C have yielded controversial results, with two showing no change (4, 77), one reporting a decrease (32), and another showing an increase (74) in HDL-C levels. Jackson et al. (32) stated that variations in HDL-C levels with the addition of PUFA may be related to differences in study protocols, types of subjects, the absolute level of PUFA intake, and the concurrent cholesterol intake, and they criticized researchers for comparing extremely high P/S ratio diets with very low ones. Jackson et al. (32) fed isocaloric diets containing 400 mg cholesterol and P/S ratios of 0.4, 1.0, and 2.0 to 6 healthy male and female subjects. Compared to the P/S = 0.4 diet, TC decreased by approximately 6 and 12% on the P/S = 1.0 and 2.0 diets, respectively. Decreases in LDL-C and HDL-C concentrations also were noted as P/S ratio increased. The mean levels of HDL-C were 49.0, 44.0, and 41.0 mg/dL for the P/S = 0.4, 1.0, and 2.0 diets, respectively. The ratios of HDL-C/TC and HDL-C/LDL-C were not significantly altered due to reductions in both HDL-C and LDL-C concentrations.

Studies such as that of Jackson et al. (32), which compared a diet high in PUFA with one high in SFA, failed to elucidate the separate effects of the two types of fatty acids. According to Brisson (8) many of the studies showing evidence of the beneficial effect of PUFA failed to isolate the influence of PUFA as the sole variable. To independently compare the effects of PUFA vs SFA on plasma lipids and lipoproteins, Becker et al. (4) isocalorically substituted equivalent amounts of PUFA and SFA for monounsaturated fatty acids (MUFA) in cholesterol-free formula diets, so that comparisons could be made of both PUFA and SFA with MUFA. TC and LDL-C decreased from baseline values of 166 and 103 to 133 and 81 mg/dL with the diet high in SFA, to 127 and 71 mg/dL with the diet high in MUFA, and to 123 and 65 mg/dL with the diet high in PUFA, respectively. These differences, along with decreases in TG levels with the PUFA diet, were statistically significant at the 5% level. No significant differences in HDL-C were noted. However, the LDL-C/HDL-C ratios were different ($P \leq 0.05$) from each other, with the lowest ratio observed on the PUFA diet, intermediate on the MUFA diet, and highest on the SFA diet. Thus, consuming a diet higher in polyunsaturates resulted in an improved lipoprotein pattern. Epidemiologically, a positive correlation ($P \leq 0.01$) between the SFA intakes and blood cholesterol levels has been observed (38), as well as a negative relationship ($P \leq 0.05$) between SFA intakes and blood HDL-C levels (16). Conversely, the LRCPPS showed no association between dietary SFA, MUFA, or PUFA intakes and blood levels of HDL-C (15).

Total fat intake. Diets providing a high percentage of energy from fat may induce rises in TC and change lipoprotein-cholesterol concentrations (23). Liebman and Bazzare (45) reported that vegetarians consuming a 23-33% fat diet had 11 and 21% lower TC and TG levels, respectively,

and 14% higher HDL-C levels than nonvegetarians consuming a 35-48% fat diet. In a population of 200 men, Kay et al. (37) observed that the percentage of calories from fat was independently and positively related to both blood TC ($P \leq 0.01$) and TG ($P \leq 0.001$) levels.

The LRCPPS showed no relationship between total fat intakes and HDL-C levels (15). However, a negative association ($P \leq 0.05$) between the percentage of energy from fat and HDL-C expressed as a percentage of TC was seen by Fehily et al. (16), who also observed a positive association ($P \leq 0.001$) between the percentage of energy from fat and LDL-C concentration.

Carbohydrate intake. Several studies have reported alterations in cholesterol metabolism in response to a high carbohydrate diet (15, 81). In the LRCPPS inverse relationships ($P \leq 0.05$) were found between HDL-C concentrations and intakes of total carbohydrate, starch, and sucrose (15). And, when a 40% fat diet was replaced by a 30% fat plus 10% glucose diet, six out of eleven subjects exhibited decreases ($P \leq 0.05$) in TC and LDL-C levels, while the whole group ($n = 11$) showed a nonsignificant increase (15%) in TG levels, and a 14% drop ($P \leq 0.05$) in HDL-C concentrations (81). Those results are consistent with Hartung's (23) observation that decreases in HDL-C might be expected with a high carbohydrate diet, as TG levels tend to be raised and HDL-C and TG levels are usually inversely related. There also may be a small increase in the HDL-C/LDL-C ratio, as there is a decline in LDL-C levels as well as in HDL-C levels. By necessity, a high carbohydrate diet is also a low fat diet, thus it is not clear whether the changes in cholesterol metabolism associated with a high carbohydrate diet are due to the carbohydrate alteration or to the fat alteration.

Fiber intake. Epidemiological as well as dietary intervention studies have demonstrated negative relationships between fiber consumption and blood lipid levels. In a multivariate analysis of 200 men, inverse correlations were found between dietary fiber intake and levels of TC ($P \leq 0.01$) and TG ($P \leq 0.05$) (37).

Recently, the administration of various types of gums to normal men for four weeks resulted in decreases ($P \leq 0.05$) in TC and LDL-C concentrations, with no significant changes in TG, VLDL-C, or HDL-C levels (5). A drop ($P \leq 0.05$) in TC and no changes in TG levels also were noted in 5 normal men who consumed gum arabic for 3 weeks (46). Anderson et al. (3) reported that oat bran and bean diets given to hypercholesterolemic men for 3 weeks lowered levels of serum TC and LDL-C by 19 ($P \leq 0.0005$) and 23% ($P \leq 0.0025$) and 19 ($P \leq 0.0005$) and 24% ($P \leq 0.0005$), respectively. No significant changes in TG concentrations were found with either the oat bran or the bean diets in subjects with normal TG levels. However, the oat bran diet lowered ($P \leq 0.001$) TG levels in hypertriglyceridemic subjects. The TG raising effect of a high-carbohydrate diet was previously reported (23), however, carbohydrate-induced hypertriglyceridemia does not occur if high carbohydrate diets are also rich in dietary fiber (2).

Alcohol intake. Strong dose-related associations between alcohol consumption and HDL-C levels have been found in both cross-sectional and longitudinal studies. The Cooperative Lipoprotein Phenotyping Study (CLPS) showed social drinkers to have 33% higher HDL-C levels than non-drinkers in five other study populations. Data from this study also indicated a direct positive relationship between alcohol intake and TG concentration, no association between alcohol and VLDL-C levels, and a tendency for LDL-C levels to be lower in men with alcohol intakes greater

than 20 ounces per week (11, 31). Cross-sectional analysis of data from the Multiple Risk Intervention Trial (MRFIT) yielded similar results (31). In the MRFIT study, men with no alcohol intake had 22% lower HDL-C levels than men consuming five or more drinks per day. This relationship held true even when confounding variables such as body weight and smoking were considered. Longitudinal analysis of MRFIT data also showed a small but significant independent association between change in alcohol intake and change in HDL-C concentrations. HDL-C levels were raised by 2.8 mg/dL for each ounce of alcohol consumed in an investigation of 164 premenopausal females (66). In the LRCPPS, a positive association ($P \leq 0.05$) was found between alcohol intake and HDL-C levels in 2473 men and 1530 women after adjustment for several confounding variables (21).

Clinical studies also have indicated that alcohol is a determinant of HDL-C levels. Hulley and Gordon (31) asked 15 long-term participants of the MRFIT study to decrease their usual alcohol intakes of 1.4 to 5.5 ounces (3 to 12 cans of beer) per day to zero for a two week period. The mean HDL-C level dropped ($P \leq 0.05$) 7 mg/dL during the abstinence period. As the men resumed their normal intakes, HDL-C concentrations increased to almost baseline values. Levels of VLDL-C also declined ($P \leq 0.05$) during the experimental period, and although a delayed decrease in LDL-C levels was observed during the second week, the change was not significant.

Haskell et al. (24) eliminated alcohol consumption by 24 healthy men for a period of 6 weeks. Decreases ($P \leq 0.05$) in concentrations of HDL-C and its subfraction, HDL_3, were observed, but no change was seen in the HDL_2 subfraction. Resumption of alcohol intake resulted in increases ($P \leq 0.05$) in both HDL-C and HDL_3 levels, but did not alter HDL_2 concentration. Haskell's (24) study indicated that the association of

alcohol with CVD is not correlated to increased HDL_2 levels and that the HDL_3 fraction may not be "inert," as previously believed. Furthermore, the relationship of alcohol and CVD may not be due to mechanisms related to HDL-C.

INFLUENCE OF OTHER FACTORS ON BLOOD LIPIDS.

Age and sex. Alterations in blood lipids and lipoproteins with age in North American populations are well established (24, 83), and population means for HDL-C for adult females are generally higher than those of their male counterparts (83). For example, in the CLPS the population means for HDL-C for males ranged from 45-49 mg/dL, while those among women were about 10 mg higher (31). Concentrations of TC, TG, LDL-C, and VLDL-C rise with age from puberty until age 60 in males, and at a slower rate and somewhat later age in females. Levels of HDL-C decrease by about 10% at puberty in males and then slowly increase at age 60-70 years. HDL-C levels in women, however, remain constant until about age 30, when they begin to rise, and then level off at age 60 (29). Stamford et al. (66) reported HDL-C levels to be 0.4 mg/dL higher and TG levels to be 1.4 mg/dL higher for each year of age in 164 premenopausal women.

Race. The LRCPPS showed black males aged 20-49 to have about 11 mg/dL higher HDL-C levels than their white counterparts when matched for TC levels. However, no differences in HDL-C levels were noted between black and white females of the same age group (28).

Adiposity. Plasma lipid and lipoprotein levels have been found to be negatively related to various indexes of adiposity in several cross-sectional studies. In the LRCPPS inverse relationships were found between the Quetelet index (W/H^2) and HDL-C levels in males and females aged 12-16

years ($P \leq 0.0001$ for males and $P \leq 0.05$ for females), 20-44 years ($P \leq 0.001$), and 45-65 years ($P \leq 0.0001$), after adjustments for age, cigarette smoking, alcohol intake, and gonadal hormone use (20). Kay et al. (37) and Stamford et al. (66) observed positive correlations between relative body weight and TC ($P \leq 0.05$) and TG ($P \leq 0.001$, and $P \leq 0.01$, respectively). Both cross-sectional and longitudinal analyses were performed on the Zutphen Study (41), in which data were collected three times at five year intervals on middle-aged men. Correlations ($P \leq 0.001$) were noted between body weight and serum cholesterol at all three observation times. Furthermore, changes in body weight during 5 and 10 years of follow-up were related ($P \leq 0.001$) to changes in serum cholesterol, and multivariate analysis demonstrated that a change of 1 kg in body fat was accompanied by a change of 2 mg/dL in serum cholesterol.

Smoking. Cigarette smoking has been found to be associated with changes in cholesterol metabolism in several studies. Smokers in the LRCPPS had lower HDL-C levels than nonsmokers when adjustments were made for age, adiposity, alcohol, and exercise level (28). The number of cigarettes smoked per day was positively correlated with cholesterol ($P \leq 0.05$) and TG ($P \leq 0.01$) in 200 healthy male subjects (37). Furthermore, Stamford et al. (66) noted that nonsmoking young women had HDL-C levels 10.1 mg/dL higher, and LDL-C levels 15.7 mg/dL lower than their smoking counterparts.

Oral contraceptives. Generally, the estrogen portion of oral contraceptive agents (OCA) has been shown to increase HDL-C and TG levels, and the progestin component to decrease HDL-C concentrations (40, 75). Reductions in the dosages of both the estrogen and progestin components of OCA have been tried in order to lessen the negative metabolic effects of these

steroids (39). An evaluation of the effects of such low-dose OCA showed that subjects using a combination of 35 μg estradiol and 0.4 mg of a weak progestin had increases ($P \leq 0.05$) in TC and HDL-C levels from baseline, and, there was no significant change in the TC/HDL-C ratio. However subjects using OCA containing 30 μg estradiol and 0.3 mg of a progestin which contained 1.5 mg of a strong progestin exhibited no significant changes in TC and HDL-C. No significant changes from baseline occurred in either treatment group for TG or LDL-C levels. Thus, the effect of OCA on lipid metabolism seems to depend on the type and dosage of the individual steroid components.

INFLUENCE OF PHYSICAL ACTIVITY ON BLOOD LIPIDS.

Participation in physical activity is on the rise in the United States, with an estimated 23 million Americans now jogging (47). In virtually every city around the country, people of all ages are pursuing rigorous exercise programs. Many publications have been written about the health benefits of exercise, but available scientific evidence of the effect of exercise on blood lipids remains unclear due to difficulties in eliminating other factors. In addition, findings of cross-sectional studies tend to differ from those of studies using a longitudinal design.

Cross-sectional studies. Long distance runners and other endurance athletes have exhibited elevated levels of HDL-C, decreased LDL-C levels, and sometimes decreased TC concentrations (23). Active men and women in the LRCPPS had higher ($P \leq 0.0001$ and $P \leq 0.02$, respectively) HDL-C concentrations than their sedentary counterparts, even after adjustments for age, body mass, alcohol intake, and smoking (25). Higher levels of physical activity also were associated with an 8.6 mg/dL higher HDL-C

level in 164 premenopausal women studied by Stamford et al. (66). Additionally, Stamford et al. (66) found a similarity in lipid and lipoprotein patterns between high-activity smokers and low-activity nonsmokers, and they suggested that chronic exercise combined with alcohol consumption may exert an additive effect on raising HDL-C levels.

Deshaies and Allard (14) examined 65 men and 31 women Olympic athletes from various countries. Elite world class athletes of both sexes had 20% higher HDL-C levels than average North Americans (65 vs 55 mg/dL in women, and 55 vs 45 mg/dL in men). Females had higher ($P \leq 0.01$) HDL-C levels than males, but no significant differences in HDL-C existed due to race or type of activity (rowing, $n = 30$; swimming, $n = 12$; track, $n = 9$, other, $n = 7$), and there were no significant correlations between HDL-C and Quetelet index (W/H^2) or maximal oxygen uptake.

Middle-aged and older males who had been jogging 3 or more times per week for 20 to 120 minutes per session for an average of 7.2 years had higher ($P \leq 0.01$) HDL-C levels and HDL-C/TC ratios than inactive control subjects (71.4 vs 57.9, and 0.30 vs 0.26, respectively), but no significant variations were observed in TG or TC concentrations (53). And, an exercise conditioning value, calculated from speed and frequency of jogging, was positively correlated ($P \leq 0.01$) with the HDL-C/LDL-C ratio, which indicated that the weekly running of long distances elevated antiatherogenic indices. Similarly, Wood et al. (83) found middle-aged men who ran more than 15 miles per week for at least one year to have lower ($P \leq 0.05$) plasma TC, LDL-C, and TG levels, and higher ($P \leq 0.05$) HDL-C levels than sedentary controls matched for age. Those differences were only partially explained by variations in adiposity between groups.

Comparisons of serum lipid, lipoprotein, and the major HDL apoprotein levels have been made between groups of active middle-aged and older men (n = 28), young women (n = 13), and middle-aged women (n = 10) and their respective sedentary controls (52). Active men, compared to controls, had higher TC ($P \leq 0.01$), HDL-C ($P \leq 0.05$), LDL-C ($P \leq 0.10$), apo A-I ($P \leq 0.05$), and apo A-II ($P \leq 0.05$) concentrations. Compared to controls, active young women had lower TC ($P \leq 0.05$), LDL-C ($P \leq 0.10$), and higher ($P \leq 0.01$) levels of apo A-I. The level of HDL-C in active young women was nonsignificantly lower than that of inactive young women (71.63 vs 75.75 mg/dL). Active middle-aged women had higher ($P \leq 0.10$) HDL-C and apo A-I concentrations compared to their inactive counterparts. Active participants in all 3 groups tended to have lower TC/HDL-C ratios than controls, which indicated a drop in atherogenic indices with increasing physical activity.

Rotkis et al. (59) established that HDL-C levels were positively related ($P \leq 0.001$) to average weekly running mileage, even when corrected for age, alcohol intake, and % body fat, in 90 experienced runners. Myhre et al. (51) suggested that increases in HDL-C with exercise were associated with both the amount and intensity of training. They found that serum HDL-C levels fell ($P \leq 0.02$) when cross-country skiers went from a period of low intensity, long duration training (LL) to one of high intensity and short duration (HS). Skiers had higher HDL-C concentrations than sedentary controls during both the LL ($P \leq 0.001$) and HS ($P \leq 0.025$) periods.

One of the most common criticisms of early studies of exercise in relation to blood lipids and lipoproteins has been failure to adequately consider the effects of diet. Recently, however, several comprehensive investigations have been conducted to study the effect of exercise and

diet on blood lipids. Reggiani et al. (57) studied 21 active and 21 inactive women in their early twenties, with active being defined as participation in various activities for at least 2 hours per day for 2 years. Results demonstrated nonsignificantly higher TC, TG, and LDL-C levels, and higher ($P \leq 0.01$) HDL-C levels in active compared to inactive women. Although the TC/HDL-C ratio was lower in active subjects, the difference was not significant. HDL-C levels were positively correlated ($P \leq 0.01$) with degree of physical fitness and dietary fat intake, however, no correlations were seen between HDL-C and calories, protein, P/S ratio, alcohol intake, or dietary cholesterol. After adjustment of HDL-C for dietary intake the variation between groups remained significant ($P \leq 0.01$).

A similar investigation by Moore et al. (50) included two levels of exercise intensity and duration. Female subjects, aged 24-58, consisted of groups of 45 long distance runners (LD) who ran a minimum of 26 miles per week, 49 joggers (J) who jogged a minimum of 6 miles per week, and 47 inactive women (I) who ran less than 2 miles per week. Findings indicated lower TG levels in LD compared to I ($P \leq 0.02$), no significant variation in TC among groups, higher ($P \leq 0.001$) HDL-C in LD (78 mg/dL) compared to J (70 mg/dL) or I (60 mg/dL), and a higher ($P \leq 0.05$) HDL-C/TC ratio in LD (0.42) compared to I (0.35). Fiber consumption was the only dietary constituent that differed significantly ($P \leq 0.01$) among groups, and pairwise comparisons showed that LD consumed more ($P \leq 0.05$) crude fiber (4.3 gm) than I (3.1 gm), but differences between intakes of J and I (3.6 gm) or J and LD were not significant. Further statistical analysis revealed that differences in dietary intake were not responsible for the observed alterations in blood lipids. The strongest determinants of HDL-C

in that study were miles run and % body fat. But, when results were adjusted for % body fat, significant differences remained only for HDL-C.

Results similar to those of Moore et al. (50) concerning duration and intensity of training were noted in a study of 19-20 year old female swimmers (65). Smith et al. (65) showed competitive swimmers (n = 7) who swam 6 days per week for 2 hours to have higher ($P \leq 0.05$) HDL-C levels (82 mg/dL) than synchronized swimmers (n = 6) who swam 3 days per week for 2 hours (70 mg/dL) and inactive controls (n = 6; 67 mg/dL). The only variation in diet between groups concerned caloric intake, which increased ($P \leq 0.05$) in proportion with physical activity. Whether or not results were adjusted for the differences observed in caloric intake was not reported.

Both Moore et al. (50) and Smith et al. (65) observed an increase in HDL-C with intensive exercise but not with moderate exercise, independent of dietary factors. Moore et al. (50), however, found HDL-C to be highly correlated with % body fat, while Smith et al. (65) found no significant correlations between any combination of the following variables: HDL-C, TC, % body fat, or relative weight.

A study of 20 male distance runners and 14 sedentary controls (71) also showed runners to have higher levels of HDL-C ($P \leq 0.001$), apo A-I ($P \leq 0.001$), and apo A-II ($P \leq 0.01$) concentrations than controls. And, although the runners had lower levels of TC, LDL-C, and TG than controls, the differences were not significant.

Even though no significant differences in intakes of carbohydrate, protein, or fat between groups were observed by Moore et al. (50) or Smith et al. (65), Thompson et al. (71) found active men to consume 40% more carbohydrate than inactive men, and suggested that the

hypertriglyceridemia associated with large carbohydrate intakes may not occur in lean individuals in a training program. Contrasting findings were reported by Reggiani et al. (57), however, who reported active female subjects to consume less ($P \leq 0.001$) carbohydrate than their sedentary counterparts.

Longitudinal studies. Training studies which have related exercise to blood lipids and lipoprotein patterns in men have generally revealed significant increases in HDL-C levels and variable changes (usually insignificant) in LDL-C, TC, and TG concentrations (30, 56, 63, 64, 67, 79, 82). Studies on initially sedentary middle-aged men have shown exercise thresholds of 8 miles per week for one year (82) and 10 miles per week for 9 months (78) necessary to cause beneficial lipoprotein changes. Nevertheless, several investigators (36, 43, 54, 70) detected no significant rise in HDL-C with exercise.

Studies of women are much rarer than those of men, and are even more contradictory. A few have shown increases in HDL-C levels with training (58, 61). For example, Rotkis et al. (58) found HDL-C levels to increase ($P \leq 0.01$) by 5 mg/dL in 19 women (average age = 29.3 years) when running mileage was increased from an average of 13.5 miles per week to an average of 44.9 miles per week over a 4-7 month period. Although those investigators noted that all subjects had normal menstrual cycles, other investigators (61) reported rises in HDL-C only in subjects who experienced menstrual irregularities during the course of the training period. The 9 subjects who developed menstrual irregularities showed increases ($P \leq 0.05$) in TC (from 156 to 173 mg/dL) and HDL-C concentrations (from 48.6 to 54.5 mg/dL), and an increase ($P \leq 0.05$) in the TC/HDL-C ratio (from 3.2 to 3.6).

Many longitudinal studies of female subjects, however, have detected no change or a slight decrease in HDL-C levels with training (17, 49). Failure of physical activity to raise HDL-C levels in 14 normal-weight women, aged 22-26 years, was reported by Moll et al. (49). The women participated in a 6 week program of 30-45 minutes of jogging, 5 days per week. Subjects were asked not to make any major changes in their diets, but adjustments in total caloric intake were made to maintain constant body weight. Training did improve the aerobic performance of the subjects as measured by a graded treadmill test, and a fall ($P \leq 0.05$) in TC was noted. But, HDL-C was not significantly altered (from 63 to 58 mg/dL), and the TC/HDL-C ratio decline was not significant (from 2.92 to 2.86). The researchers postulated that hormonal and other factors leading to higher baseline HDL-C concentrations in women counteracted the alterations in cholesterol metabolism expected with physical conditioning.

Similar results were reported by Frey et al. (17) who examined 16 women, aged 19-29 years, in a 10 week, 3 times per week, bicycle ergometer training program. The subjects were divided into two groups, with one group ($n = 12$) participating in an interval type program and the other ($n = 6$) in a continuous program. All subjects showed marked ($P \leq 0.0001$) improvement in maximal oxygen uptake and work capacity, and reductions in % body fat ($P \leq 0.002$) and resting heart rate ($P \leq 0.005$). Levels of TG and HDL-C did not change, although HDL-C concentrations were significantly lower at 2 and 5 weeks, but returned to baseline levels at 10 weeks (61.67 mg/dL at baseline, and 62.00 mg/dL after 10 weeks). TC and LDL-C levels, however, were increased slightly ($P \leq 0.10$) at the end of the conditioning period. Examination of food records showed no changes in eating patterns throughout the study, and alcohol consumption was fairly

uniform, with a few subjects consuming more or less than the average 10 week value of 24.5 gm per subject.

In a recent meta-analysis (statistical study of the findings of many studies) of 66 training studies (n = 2498 males and 427 females) conducted over a period of 26 years, Tran et al. (72) found that exercise reduced levels of TG by 15.8 mg/dL ($P \leq 0.01$), TC by 10 mg/dL ($P \leq 0.01$), LDL-C by 5.1 mg/dL ($P \leq 0.05$), and nonsignificantly raised HDL-C levels by 1.2 mg/dL. Those changes were most apparent in individuals who initially had exceptionally high TC, TG, and LDL-C levels and extremely low HDL-C levels. Other factors related to the magnitude of change included age, length and intensity of training, body weight, % body fat, and maximal oxygen consumption.

The inconsistent results of training studies are most often blamed on poor experimental design, specifically, failure to consider the weight loss accompanying an exercise program if additional calories are not consumed (30, 64, 79), and lack of adequate dietary examination and short training periods with various exercise intensities (82).

MATERIALS AND METHODS

Subjects. Twenty-six healthy female subjects between the ages of 20 and 32 years (mean = 24.5 years), were selected from a group of volunteer students and faculty at Kansas State University. Subjects were recruited by announcements in several nutrition classes, and distribution of a notice (which included a brief description of the study) to all faculty and graduate students in the College of Home Economics (see Appendix).

Interested persons completed a questionnaire, 'Exercise, Diet, and Medical Questionnaire' (see Appendix) which was used to eliminate persons

with health or dietary problems, to categorize the potential subjects into groups based on physical activity, and to quantify factors other than diet and exercise which may influence cholesterol metabolism.

Six subjects made up a group of high mileage runners, who ran from 40 to 50 miles per week, and ten subjects constituted a group of low mileage runners, who ran from 20 to 30 miles per week. All runners recorded mileage and time spent running each day during a 7-day period. The control group consisted of 10 subjects who were not engaged in a regular exercise program.

All subjects were within the ranges of desirable weights for heights suggested by the Committee on Dietary Allowances (12), and no one was actively trying to lose or gain weight by dietary means. All subjects were instructed regarding the procedures of the investigation and were provided informed consent information (see Appendix).

Dietary Analysis. Subjects were instructed on how to keep a 7-day diet record (see Appendix), and records were turned in daily and checked by researchers to facilitate accuracy in recording. Total calories, protein, carbohydrate, fat, saturated fat, alcohol, and fiber, and the percentage of calories from carbohydrate, protein, fat, saturated fat, and alcohol were quantified for each subject using a nutrient data program from the Agricultural Handbooks Number 456, Nutritive Value of American Foods, and 8 (1-9), Composition of Foods.

Body Composition. Percent body fat was determined for all subjects in the Exercise Physiology Laboratory by hydrostatic weighing, using the method of Behnke and Wilmore (6). Subjects were weighed on a balance beam scale (HOMS, model 150 TK) to the nearest ± 10 grams. Underwater weights were

measured using a 9 kg autopsy scale (Chatillon) accurate to ± 10 grams. Consecutive hydrostatic weights were recorded until a plateau could be identified, as described by Katch (35). Body density (D_b) was calculated using the average of three plateau values. The Siri equation was used to calculate % body fat from D_b (62): % body fat = $(\frac{495}{D_b} - 450)$.

Subjects wore nylon swim suits during all weighings, they were asked to fast for a minimum of 6 hours before reporting to the laboratory for testing. They were also asked to eliminate gas-forming foods from their diets prior to the fast.

Pulmonary residual lung volume was determined for each subject prior to hydrostatic weighing by the use of a nitrogen analyzer (model 47302 A, Hewlett Packard) using the oxygen dilution technique of Wilmore (80).

Blood samples. Fasting venous blood samples of 10 ml each were drawn into vacutainer clotting tubes in the Nutrition Laboratory approximately 3 weeks after the dietary data collection period. Subjects were asked to fast overnight (12-16 hours) before the test, and not to engage in any physical activity before coming to the laboratory in the morning to avoid acute post-exercise effects.

Serum was separated within two hours using a centrifuge (IFC Centra-7R) at room temperature and 2,500 rpm for 15 minutes. Serum from each sample was promptly transferred to 2 falcon tubes, one marked for triglyceride (TG) and the other for cholesterol analysis, providing approximately 5 ml for each of the two analyses. Serum was stored at -20°C for lipid analyses.

Serum total cholesterol (TC), alpha cholesterol (high-density lipoprotein cholesterol (HDL-C)), and beta cholesterol (low-density lipoprotein cholesterol (LDL-C) + very low-density lipoprotein cholesterol

(VLDL-C) were determined using the Isolab LDL-Direct Cholesterol Audit System (68), and a probe colorimeter (model PC80, Brinkman) (see Appendix for details of method).

Serum TG were assessed via the Sigma Quantitative Determination of Triglycerides in Serum or Plasma at 405-415 nm (69), and a spectrophotometer (Spectronic 20, Bausch and Lomb) (see Appendix for details of method).

In addition, LDL-C was calculated using the method of Friedwald et al. (18), as: LDL-C = TC - (HDL-C + $\frac{TG}{5}$). This method provides an accurate estimation of LDL-C if levels of TG are less than 400 mg/dL. The following two assumptions are made when using this equation: 1) the weight ratio of VLDL/TC is 1/4, and 2) in the fasting state the majority of TG are carried by VLDL rather than by chylomicrons.

Statistical analysis. Analyses of variance were used to evaluate group differences in physical characteristics, nutrient intake, and serum lipids and lipoprotein-cholesterol levels. Table 1 illustrates the model, which also provided a pooled estimate of the magnitude of sampling variation encountered in this study, as measured by standard deviations. Statistical significance was determined at the $P \leq 0.05$ level. Correlation coefficients were determined to assess the relationship between serum lipids and physical characteristic and dietary intake variables using the Pearson product moment correlation formula.

TABLE 1. Analysis of variance

SOURCE OF VARIATION	DF
Groups (fixed)	2
Subjects: Group	23
Total	25

RESULTS AND DISCUSSION

None of the subjects smoked, had menstrual irregularities, had recently undergone a weight change, or suffered from any known illness or disease. Although none of the runners reported taking any drugs that might influence serum lipids or lipoproteins, three subjects in the control group used low-dose oral contraceptives.

The studies referred to in discussing results used female subjects unless otherwise stated.

PHYSICAL CHARACTERISTICS.

Physical characteristics of inactive subjects (I), low mileage runners (LM), and high mileage runners (HM) are summarized in table 2.

Age. Subjects did not differ significantly in age, although HM tended to be slightly older than either I or LM.

Adiposity. The W/H index was higher ($P \leq 0.05$) for I than for HM, but differences between I and LM and LM and HM were not significant. Inactive subjects also exhibited a higher ($P \leq 0.05$) % body fat than either LM or HM.

TABLE 2. Physical characteristics of inactive subjects, low mileage, and high mileage runners.*

VARIABLE	INACTIVE (n = 10)	LOW MILEAGE (n = 10)	HIGH MILEAGE (n = 6)	POOLED S.D.**
Age (years)	23.9^{a}	23.6^{a}	26.8^{a}	3.6
W/H (kg/m)	36.4^{a}	33.2^{ab}	32.7^{b}	3.3
% Body Fat	32.7^{a}	21.9^{b}	18.8^{b}	3.6

* Values are means. Means sharing a common superscript within the same row are not different at $P \leq 0.05$.

** Pooled estimate of S.D. within activity groups using 23 D.F.

Other cross-sectional studies of women have yielded similar results. For example, Moore et al. (50) found a higher ($P \leq 0.001$) % body fat in inactive subjects (29.8%) than in long distance runners (22.5%). They also reported a higher ($P \leq 0.001$) % body fat in inactive subjects than in joggers (26.6%), and in joggers compared to long distance runners. Smith et al. (65) also found a tendency for competitive swimmers to have a lower % body fat than inactive subjects (19.4 vs 20.8%, respectively), but this difference was not significant. In both of those studies, as well as in the present one, subjects at lower exercise intensities had body fat values intermediate between inactive subjects and higher intensity exercisers.

DIETARY FACTORS.

Mean nutrient intakes of inactive subjects, low mileage, and high mileage runners are shown in table 3. All 3 groups were within the range of calorie intake recommended by the National Research Council for females 19-22 (1700-2200 kcals/day) and 23-50 years old (1400-2200 kcals/day) (12). The % of calories from protein in all three groups was higher than the 12% recommended by the U.S. Senate Select Committee (73), and the 11-12% reported as the average protein content of the U.S. diet (55). Intakes of carbohydrate, fat, and saturated fat, expressed as a % of calories were intermediate between the 42, 46 (55), and 16 (73) %, respectively, contributed to the national diet and the 58, 30, and 10% recommended by the U.S. Senate Select Committee (73). Subjects in all 3 groups also derived considerably less of their calories from alcohol than the 5-10% that is reported for social drinkers (22). This may be explained by the fact that 46% (12 out of 26) of the subjects in the present study consumed no alcohol at all. Subjects in the I and LM groups consumed less

TABLE 3. Nutrient intakes of inactive subjects, low mileage, and high mileage runners.*

VARIABLE	INACTIVE (n = 10)	LOW MILEAGE (n = 10)	HIGH MILEAGE (n = 6)	POOLED S.D.**
Total calories	1795[a]	1624[a]	1864[a]	478
% Protein	14.1[a]	15.9[ab]	17.9[b]	2.9
% Carbohydrate	50.5[a]	49.5[a]	48.8[a]	9.0
% Total Fat	36.5[a]	36.8[a]	32.6[a]	7.0
% Saturated Fat	14.5[a]	13.9[a]	13.3[a]	4.1
% Alcohol	0.7[a]	0.6[a]	3.1[b]	1.9
Protein (gm)	66.2[a]	62.1[a]	88.0[a]	29.7
Carbohydrate (gm)	228.1[a]	201.9[a]	219.4[a]	61.5
Total Fat (gm)	71.9[a]	66.4[a]	70.4[a]	28.0
Saturated Fat (gm)	27.5[a]	25.1[a]	29.9[a]	13.8
Cholesterol (mg)	292.1[a]	224.7[a]	623.8[a]	462.4
Crude Fiber (gm)	3.5[a]	4.8[a]	5.4[a]	2.2

* Values are means over a 7 day period. Means sharing a common superscript within the same row are not different at $P \leq 0.05$.
** Pooled estimate of S.D. within activity groups using 23 D.F.

cholesterol than both the 300 mg/day that is recommended (73) and the 600 mg/day commonly consumed by Americans (73). The HM group, however, consumed in excess of 600 mg/day. This large mean intake was due in part to the consumption of an average of 2591 mg/day cholesterol by one subject in that group (see Appendix).

Caloric intake. No significant differences were noted in caloric intake among the 3 groups. Moore et al. (50) observed that low intensity exercisers consumed fewer calories than either inactive subjects or high intensity exercisers. Those investigators observed inactive subjects to consume 1740 kcals, joggers, 1562 kcals, and long distance runners, 1765 kcals, whereas this study revealed that I consumed 1795 kcals, LM,

1624 kcals, and HM, 1864 kcals. Other studies (7, 57, 65) have shown that high intensity exercisers consumed a significantly greater amount of calories than inactive subjects. And Smith et al. (65) found that low intensity exercisers consumed an amount of calories intermediate between that consumed by inactive subjects and high intensity exercisers (2282, 2030, and 2468, respectively).

Protein intake. The percentage of calories from protein was higher ($P \leq 0.05$) for HM than for I (17.9 vs 14.1), but the difference was not significant when protein was expressed in grams. Other researchers (7, 50, 57, 65) have not noted differences in % of calories from protein between inactive and highly active female subjects.

Carbohydrate intake. The % of calories from carbohydrate, as well as carbohydrate expressed in grams, was similar for all groups. This is consistent with the findings of some investigators (7, 50, 65), whereas others (57, 71) reported variations in carbohydrate intake between highly active and inactive male and female subjects. Thompson et al. (71) found that active men consumed 40% more carbohydrate than inactive men, and Reggiani et al. (57) reported that active women consumed less ($P \leq 0.001$) carbohydrate than inactive women.

Fat intake. No significant group variations in total and saturated fat were found whether expressed as % of calories or in grams. Blair et al. (7) found no significant differences between runners and controls when saturated fat was expressed on a per 1000 calorie basis, or % of calories from fat. Smith et al. (65) showed groups to be similar in % of calories from fat and the P/S ratio of the diet. Additionally, Moore et al. (50) found that inactive subjects, joggers, and long distance runners consumed

41.4, 39.7, and 41.7% of calories from fat, respectively, and 25.8, 23.1, and 25.5 grams of saturated fat, respectively. However, Reggiani et al. (57) observed that active subjects consumed more total ($P \leq 0.001$) and saturated ($P \leq 0.05$) fat than inactive subjects, when expressed as % of calories (40.9 vs 30.4, and 16.5 vs 11.3, respectively).

Alcohol intake. Four out of 10, 6 out of 10, and 5 out of 6 subjects in the I, LM, and HM groups, respectively, reported consumption of alcohol on their food records. Most of the alcohol consumed was from beer (332 oz.), with smaller amounts from wine (54 oz.) and hard liquor (4.5 oz.). Groups I, LM, and HM consumed 65, 128, and 198 ounces of alcohol, respectively. The % of calories from alcohol was higher ($P \leq 0.05$) for HM compared to either I or LM (3.1, 0.7, and 0.6%, respectively). However, intakes for I and LM were much lower than, and intakes for HM about the same as those reported in the literature for subjects of the same age and sex. Reggiani et al. (57) reported no significant differences in alcohol intake between active and inactive subjects (3.5 and 2.0, respectively), although active subjects tended to consume more alcohol than inactive subjects. Likewise, Moore et al. (50) found that inactive subjects obtained 3.6% of calories from alcohol, while joggers and long distance runners consumed 2.9 and 4.1% of calories as alcohol, respectively.

Cholesterol intake. Groups did not differ significantly in grams of cholesterol consumed per day, although at first glance it may appear that HM consumed considerably more than either I or LM (623.8 vs 291.1 and 224.7 mg/day, respectively). An extremely high intake of cholesterol (2591 mg/day) for just one subject in the HM group may explain why the difference was not significant. Most other studies also have shown no

differences in cholesterol consumption between physically active and inactive groups (7, 50, 65, 71). Reggiani et al. (57) however, found active subjects to consume more ($P \leq 0.001$) cholesterol than their inactive counterparts (408.7 vs 245.7 mg/day, respectively).

Fiber intake. Although intake of crude fiber tended to increase with exercise intensity, differences between groups were not significant. Contrastingly, Moore et al. (50) found that crude fiber was the only nutrient that differed ($P \leq 0.01$) between groups. In their study inactive subjects, joggers, and long distance runners consumed 3.1, 3.6, and 4.3 gms/day crude fiber, respectively. Although these differences do not appear to be greater than those in the present study (I = 3.5, LM = 4.8, and HM = 5.4 gm/day) that study included a much larger subject population than ours (141 subjects vs 26 subjects), thus levels of significance could be reached with smaller group differences. Fiber was not included as part of the dietary analysis in various other studies (7, 57, 65, 71).

SERUM LIPIDS.

Table 4 summarizes mean serum levels of total cholesterol, triglycerides, and lipoprotein-cholesterol for the I, LM, and HM groups. The average range of total cholesterol for females 20-34 years old is 170.3-179.1 mg/dL (data from the Lipid Research Clinics Population Studies Data Book) (1). Both I and LM had lower than average TC value (154.6 and 162.2, respectively), but HM had higher than average TC concentrations 191.0 mg/dL. The average HDL-C concentration for females 20-34 years old ranges from 53.3-56.1 mg/dL (data from the Lipid Research Clinics Population Data Book) (1). Levels of HDL-C for I were at the upper end of this

range (56.2 mg/dL), and those for LM and HM were higher than average (61.1 and 61.2 mg/dL, respectively).

TABLE 4. Serum lipids of inactive subjects, low mileage, and high mileage runners (mg/dL).*

VARIABLE	INACTIVE (n = 10)	LOW MILEAGE (n = 10)	HIGH MILEAGE (n = 6)	POOLED S.D.**
Total cholesterol	154.6[a]	162.2[a]	191.0[b]	23.9
Triglycerides	63.1[a]	47.1[a]	65.2[a]	21.1
HDL-cholesterol†	56.2[a]	61.1[a]	61.2[a]	10.8
LDL-cholesterol†	85.5[a]	91.8[a]	115.7[b]	18.9
HDL-C/TC†	0.36[ab]	0.38[a]	0.32[b]	0.05

* Values are means. Means sharing a common superscript within the same row are not different at $P \leq 0.05$.
** Pooled estimate of S.D. within activity groups using 23 D.F.
† HDL-C = high-density lipoprotein cholesterol, TC = total cholesterol, and LDL = low-density lipoprotein.

Total cholesterol. The mean concentration of TC for HM (191.0 mg/dL) was higher ($P \leq 0.05$) than that of either I (154.6 mg/dL) or LM (162.2 mg/dL). Several other groups of investigators have reported similar levels of TC, but did not find significant differences between inactive and active subjects (50, 57, 65, 71). For example, Moore et al. (50) found that female long distance runners had nonsignificantly higher TC than either joggers or inactive subjects (185 vs 182 and 179 mg/dL, respectively). Decreases in TC with exercise also have been reported in both middle-aged men (83) and young women (52).

Triglycerides. No significant group differences were found in serum triglycerides (TG). Middle-aged males who had been engaged in an exercise program for an average of 7.2 years also showed no significant variations in TG when compared to sedentary controls (53). Wood et al. (83),

however, reported that middle-aged distance runners had lower ($P \leq 0.05$) TG than control subjects, and Moore et al. (50) found that long distance runners had lower TG levels than either joggers or inactive subjects. In contrast, Reggiani et al. (57) observed insignificantly higher TG levels in active compared to inactive subjects.

High-density lipoprotein cholesterol. No differences in HDL-C concentrations were observed among groups, although HM and LM tended to have slightly higher values than I (61.2 and 61.1 vs 56.2 mg/dL, respectively). Nagoa et al. (52) found no significant differences in HDL-C between active and inactive groups of young women, but the active group tended to have lower levels of HDL-C (71.7 mg/dL) than inactive subjects (75.5 mg/dL). Many similar cross-sectional studies, however, have reported increases in HDL-C levels with increasing exercise intensity (50, 65). For example, Moore et al. (50) found inactive subjects, joggers, and long distance runners to have 62, 70, and 78 mg/dL HDL-C, respectively.

About 70% (18 out of 26) of the participants in the present study and 80% (8 out of 10) of those in the inactive group were in the food science and/or nutrition field. Thus, our subjects, especially the inactive ones, may represent a more health conscious segment of the population than the general public. Furthermore, levels of HDL-C have been positively correlated ($P \leq 0.0002$) with educational achievement among females of all ages (27). Out of the 26 women in the present study, 18 were college graduates with 14 pursuing Master's degrees, 1 holding a Master's degree, and 3 holding Ph.D. degrees. The remaining 8 subjects were undergraduates who had completed at least 2 years of college. Therefore, our subjects may represent a more highly educated subpopulation compared to the general public.

Low-density lipoprotein cholesterol. In this study, HM had higher ($P \leq 0.05$) low-density lipoprotein cholesterol (LDL-C) levels than either I or LM (HM = 115.7, I = 85.5, and LM = 91.8 mg/dL). Other investigations have shown no significant differences in this parameter in inactive compared to active women (50, 57, 65) or in men (71).

HDL-C/TC ratio. The ratio of HDL-C to TC was higher ($P \leq 0.05$) in LM (0.38) compared to HM (0.32), with I showing an intermediate value (0.36). Others have demonstrated nonsignificant differences between groups (57, 65) or increasing ($P \leq 0.001$) ratios with increasing exercise (50). Moore et al. (50) found that inactive subjects, joggers, and long distance runners had ratios of 0.35, 0.39, and 0.42, respectively.

CORRELATIONS BETWEEN SERUM LIPIDS AND PHYSICAL CHARACTERISTICS.

Correlation coefficients between serum lipids and lipoproteins and physical characteristics are shown in table 5.

Age. A significant positive correlation was found between age and TC ($P \leq 0.05$) and age and LDL-C ($P \leq 0.10$). This may, at least in part, explain the higher levels of TC and LDL-C observed in HM compared to either I or LM, as HM tended to be older than I or LM. Although Stamford et al. (66) reported a rise in HDL-C and TG levels with age, correlations of these parameters with age were not significant in the present study. The ratio of HDL/TC was also unrelated to age.

Adiposity. No significant correlations were found between measures of body fat and serum lipids and lipoproteins. The Lipid Research Clinic's Program Prevalence Study (LRCPPS) however, showed an inverse correlation ($P \leq 0.001$) between Quetelet index (W/H^2) and HDL-C levels in young women,

TABLE 5. Correlation coefficients between serum lipids and physical characteristics of all subjects.

VARIABLE[1]	r
TC vs Age (years)	.422*
Wt/Ht Index (kg/m)	-.010
% Body Fat	-.231
TG vs Age (years)	.316
Wt/Ht Index (kg/m)	.322
% Body Fat	.087
HDL-C vs Age (years)	.145
Wt/Ht Index (kg/m)	.017
% Body Fat	-.091
LDL-C vs Age (years)	.384†
Wt/Ht Index (kg/m)	-.099
% Body Fat	-.262
HDL-C/TC vs Age (years)	-.252
Wt/Ht Index (kg/m)	.025
% Body Fat	.120

[1] TC = total cholesterol, TG = triglycerides, HDL-C = high density lipoprotein cholesterol, and LDL-C = low density lipoprotein cholesterol.

† Significant at $P \leq 0.10$ * Significant at $P \leq 0.05$

and others have observed positive correlations between relative body weight and TC and TG concentrations (37, 66). One of the strongest determinants of HDL-C found by Moore et al. (50) was % body fat, but Smith et al. (65) found no relationship between these measures.

CORRELATIONS BETWEEN SERUM LIPIDS AND DIETARY FACTORS.

Correlation coefficients between serum lipids and dietary factors of all subjects are shown in table 6.

Caloric intake. The amount of calories consumed was not significantly related to any of the lipid parameters. Moore et al. (50) found an inverse correlation ($P \leq 0.001$) between caloric intake and TC, but no relationship between calories and TG, HDL-C, or LDL-C levels, or the HDL-C/TC ratio.

TABLE 6. Correlation coefficients between serum lipids and dietary intake of all subjects.

SERUM VARIABLE[1]	DIETARY VARIABLE[2]										
	Kcals	Pro	CHO	Fat	S.F.	Alc	Pro	CHO	Fat	S.F.	C.F.	Chol
		(% of Kcals)					(gm)					(mg)
TC	.252	.445*	.244	.054	.179	.212	.441*	-.076	.293	.378†	.095	.471*
TG	.098	-.072	-.083	.131	.187	-.056	-.005	.078	.098	.120	.056	-.028
HDL-C	.177	.320	-.249	.128	.192	.261	.274	-.093	.264	.333†	-.022	.358†
LDL-C	.227	.410*	-.148	-.032	.074	.105	.428*	-.039	.226	.297	.138	.424†
HDL-C/TC	-.091	-.112	-.051	.129	.073	.038	-.175	-.064	-.012	-.024	-.162	-.098

[1] TC = total cholesterol, TG = triglycerides, HDL-C = high density lipoprotein cholesterol, and LDL-C = low density lipoprotein cholesterol.

† Significant at $P \leq 0.10$ * Significant at $P \leq 0.05$

[2] Pro = protein, CHO = carbohydrate, S.F. = saturated fat, Alc = alcohol, C.F. = crude fiber, and Chol = cholesterol.

And, Reggiani et al. (57) reported no significant correlation between HDL-C concentration and total caloric intake.

Protein intake. In this study, correlations ($P \leq 0.05$) existed between both the % of calories from protein and grams of protein and serum TC and LDL-C levels. Correlations between protein and TG, HDL-C, and the HDL-C/TC ratio were insignificant. Other researchers found no association between levels of HDL-C and protein intake (50, 57), and Moore et al. (50) reported no significant correlations between protein intake and TC, TG, LDL-C concentrations of the HDL-C/TC ratio.

The HM group had higher ($P \leq 0.05$) TC and LDL-C levels, and % of calories from protein than either I or LM. Thus, the finding of an association between TC and LDL-C levels and the % of calories from protein may partially explain why HM had higher levels of TC and LDL-C than the other 2 groups, although this association has not been substantiated by other researchers.

Carbohydrate intake. Although all serum variables except TC and TG were negatively associated with carbohydrate intake, none of the correlations were significant. Significant inverse correlations between % of calories from carbohydrate and HDL-C levels were noted by both Reggiani et al. (57) and Moore et al. (50) in their studies of female runners. Additionally, grams of carbohydrate was positively correlated ($P \leq 0.001$) with the HDL-C/TC ratio (50), and the LRCPPS showed an inverse relationship ($P \leq 0.05$) between HDL-C and carbohydrate intake (15).

Fat intake. Both total fat and the % of calories from saturated fat were not significantly related to any serum lipid measurement in this study, but grams of saturated fat was positively correlated ($P \leq 0.10$) with TC

and HDL-C levels. Other studies have suggested a positive relationship between total fat intake and levels of TC, TG (37, 45), and LDL-C concentrations (16), as well as a positive correlation between the intake of saturated fat and TC (38). Although a negative correlation ($P \leq 0.05$) between saturated fat intake and HDL-C concentration was noted by Fehily et al. (16), the LRCPPS showed no association between dietary saturated fat and levels of HDL-C (15). In female runners, Reggiani et al. (57) found a positive correlation ($P \leq 0.01$) between % of calories from fat and HDL-C concentration, however, the P/S ratio of the diet was unrelated to HDL-C levels. Negative associations ($P \leq 0.001$) between the % of calories from fat and TG levels, and between grams of saturated fat and the HDL-C/TC ratio were observed by Moore et al. (50). However, those investigators found no relationship between dietary total fat and TC, HDL-C, and LDL-C levels, or the HDL-C/TC ratio, or between dietary saturated fat and TC, TG, or HDL-C concentrations.

Alcohol intake. The amount of alcohol consumed was not significantly related to any of the serum variables in this study. Numerous population studies have demonstrated strong dose-related associations between alcohol consumption and HDL-C concentrations (21, 31, 66), and alcohol intake was related to levels of TG and inversely related to LDL-C levels in the Cooperative Lipoprotein Phenotyping Study (31, 11). Those studies, however, included much larger subject populations and a greater variety of alcohol intakes compared to the present one. Investigations similar to ours have reported no significant correlations between alcohol intake and HDL-C (50, 57), or TG levels, or the HDL/TC ratio (50), but correlations ($P \leq 0.001$) were noted between the % of calories from alcohol and TG levels, and grams of alcohol and LDL-C concentrations (50).

Cholesterol intake. Significant correlations were found between cholesterol intake and levels of TC ($P \leq 0.05$), HDL-C ($P \leq 0.10$), and LDL-C ($P \leq 0.10$). Similar studies have reported no significant correlations between dietary cholesterol and HDL-C levels (50, 57) or levels of TC, TG, LDL-C, or the HDL-C/TC ratio (50). Population studies have yielded inconsistent results. In the LRCPPS, a positive association ($P \leq 0.05$) was found between dietary cholesterol and HDL-C levels in women not taking gonadal hormones, but this relationship did not exist for other women or men. Another study, however, showed no correlation between cholesterol intake and plasma lipids or lipoprotein-cholesterol (45).

Fiber intake. Crude fiber was not significantly correlated with serum lipids or lipoprotein-cholesterol levels in this study. An amount of fiber considerably higher than that consumed in the present study would be necessary to induce lipid changes. While our intakes are comparable to the amount commonly consumed by Americans, Anderson et al. (3) used 3 to 6 times that amount to induce lipid changes in their subjects. Moore et al. (50) also looked at crude fiber and found similar results. In contrast, inverse correlations were found between dietary fiber and levels of TC ($P \leq 0.01$) and TG ($P \leq 0.05$) in a study of 200 healthy men (37). Various other studies also have demonstrated negative relationships between fiber intake and blood lipid levels (3, 5, 46).

SUMMARY

The influence of dietary intake and exercise intensity on serum levels of total cholesterol (TC), triglycerides (TG), and high- and low-density lipoprotein cholesterol was studied in 26 females between the ages of 20 and 32 years. Subjects were categorized into 3 groups

according to their habitual level of physical activity. Six subjects were included in a group of high mileage runners (HM) who ran 40-50 miles per week, and 10 subjects made up a group of low mileage runners (LM) who ran 20-30 miles per week, and 10 subjects not involved in any exercise program served as inactive controls (I).

Concentrations of TC and low-density lipoprotein cholesterol (LDL-C) were higher ($P \leq 0.05$) in HM than in either I or LM (191.0 vs 154.6 and 162.2 mg/dL, respectively). No significant differences in high-density lipoprotein cholesterol (HDL-C) existed between groups, although LM and HM tended to have higher HDL-C levels than I (61.1 and 61.2 vs 56.2 mg/dL, respectively). All groups were within average TC and HDL-C ranges for their age and sex, except that HM had higher than average levels of TC (191.0 vs 170-179 mg/dL). The HDL-C ratio was higher ($P \leq 0.05$) in LM (0.38) compared to HM (0.32), with I showing an intermediate value (0.36).

Age and protein intake both were correlated ($P \leq 0.05$) with levels of TC and LDL-C. These correlations may partially explain why HM had higher ($P \leq 0.05$) levels of TC and LDL-C than I or LM, as HM also had higher ($P \leq 0.05$) protein intakes and tended to be older than the other two groups (26.8 vs 23.9 and 23.6 years).

Both saturated fat and cholesterol intakes were correlated with TC ($P \leq 0.10$ and $P \leq 0.05$, respectively) and HDL-C ($P \leq 0.10$) concentrations, and cholesterol intake was also correlated ($P \leq 0.10$) with levels of LDL-C. However, no significant differences in these nutrients were found among groups. HM consumed more ($P \leq 0.05$) alcohol than either I or LM, but alcohol was not related to any serum lipid measurement. Furthermore, no significant correlations were found between any lipid parameter and any of the following variables: % body fat, weight/height, calories, carbohydrate, total fat, or fiber,

ACKNOWLEDGMENTS

The author is very grateful to Dr. Beth Fryer, Major Professor, for her guidance and encouragement throughout the author's graduate study, research and preparation of this manuscript.

Sincere appreciation is expressed to Dr. Robert Reeves for his proficiency in drawing blood samples and for serving on the supervisory committee, and to Dr. Holly Fryer for his expertise and patience in planning and performing the statistical analyses, and also for being on the supervisory committee.

The author is also grateful to Dr. Sue Bolze and Dr. Katharine Grunewald not only for their help and suggestions in the biochemical analyses, but also for their participation as subjects.

Thanks also are due to Dr. Tony Wilcox for his patience and expertise in taking body composition measurements.

A special thanks is expressed to Cindy Byfield, Jo Ann Cipriano, Julie Clark, Angela Di Georgio, Stephanie Dunshee, Bettina Grieb, Gail Gumminger, Cathy Hastings, Crystal Jenkins, Vilma Maldonado, Lori McIlhiney, Marty Oborg, Mary Jo Olberding, Bert Phillips, Letitia Piper, Sheryll Ramsey, Shari Rogers, Tami Rollins, Cristi Searls, Tammy Tucker, Laura Tunnel, and Kris Williams for their participation and cooperation as subjects. (The author also served as a subject.)

Deep and sincere appreciation and thanks are extended to my parents, Louis and Alice Wiese, and to my husband, Reza, for their sacrifices, patience, and steadfast encouragement throughout my studies.

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APPENDIX

FEMALE RUNNERS NEEDED!

For: a Master's research project investigating the effects of running on appetite. Your body composition and caloric cost of running will be determined.

CRITERIA:

- -female
- -20-30 years of age
- -must have been running (or jogging) a minimum of 2 miles per day, 5 times per week consistently for at least 1 year.

Controls who do not engage in any regular exercise are also needed.

_____ I would be interested in being a subject (runner).

_____ I would be interested in being a control.

Name _____

Dept. _____

Phone _____

Please return to: Kris Williams
Foods and Nutrition
Justin 209

EXERCISE, DIET AND MEDICAL QUESTIONNAIRE

This information is strictly confidential and will be used only by the researchers.

Please fill out the following form as completely as possible.

Name: _____ Date: _____ Age: _____ Birthdate: _____

Campus Address: _____ Phone: _____

Home Address: _____ Phone: _____

Occupation (student, faculty, etc.): _____ Sex: _____

Activity level: very sedentary ____
sedentary ____
light ____
moderate ____
heavy ____
very heavy ____

Height: _____

Weight: _____

Number of miles run per week: _____

Number of days run per week: _____

Minutes run per exercise bout: _____

How long (in months, years) have you been running?

Do you engage in any regular type of exercise other than running? If so, list the activities and the frequency of participation:

What are your reasons for running?

Are you trying to lose weight? gain weight?

Has your food intake increased or decreased since you have been running? If so, explain:

Do you notice a difference in your food intake on days you run versus days you do not? If so, explain:

Do you have any health problems that might limit you in some way when exercising?

Do you have any type of disease that might influence your food intake? (i.e. diabetes, hypoglycemia, etc.)

Do you smoke? If so, how much?

Do you consume alcohol? If so, how much and how often?

Questionnaire - continued

Do you take oral contraceptives?

Do you take any regular medications? If so, for what purposes?

List medications:

Check if you have any of the following:

____ diabetes mellitus

____ thyroid disorder

____ high blood pressure

____ chest pains

____ heart murmur

____ kidney disorder

____ any type of infection

____ any type of tumor

____ frequent colds

____ frequent sore throat

____ irregular menstrual cycle

____ chronic constipation or irregularity

____ diarrhea

____ gall bladder disease

____ gastric or duodenal ulcer

____ anemia

____ any nervous or emotional problems

____ recent weight change

____ poor appetite

____ excessive weakness or tiredness

____ vomiting

____ menstrual cramps

Questionnaire - continued

What do you consider a good weight for yourself?

What is the most you have ever weighed? At what age?

Weight now: Weight one year ago:

Number of meals you usually eat per day: _____

Number of snacks you usually eat per day: _____

List any vitamin-mineral or protein or other supplements taken on a regular basis. Also list the brand name and nutrient composition.

Number of times per week you usually eat:

____ beef

____ pork

____ fish

____ fowl

____ eggs

____ variety meats

____ cheese

____ milk

____ other dairy products - list items

____ bread

____ cereals

____ cakes, cookies, pastries

____ other desserts - list items

____ fruit or juices

____ vegetables

____ fats - oils, salad dressing, butter, margarine, etc.

____ legumes, beans, etc.

____ other (indicate)

Questionnaire - continued

____ regular soft drinks

____ diet soft drinks

____ beer

____ other alcoholic beverages - list

PROJECT TITLE: EFFECTS ON YOUNG WOMEN OF A REGULAR RUNNING PROGRAM

INVESTIGATORS: Beth Fryer, Project Director
Kris Williams, Graduate Assistant
Karen Wiese, Graduate Assistant

JUSTIFICATION: Running is one of the more popular forms of exercise for many young women. Reports of the effects of running on women have not always been in agreement. In this study, two groups of young female runners (20-30 miles/wk and 40-50 miles/wk) will be compared with each other and with a sedentary control group. Factors to be studied will include: 1) caloric intake and body composition; 2) iron status; and 3) plasma lipids and nutrient intake.

AGREEMENT AND RELEASE

1. I volunteer to participate in the study of "Effects on Young Women of a Regular Running Program" to be conducted during October and November, 1984 in the Department of Foods and Nutrition, Kansas State University by Beth Fryer, Project Director and Kris Williams and Karen Wiese, Graduate Assistants.

2. I will keep a record of all foods and beverages consumed for a period of 7 days.

3. I also will keep a record of the amount of time spent running and the estimated mileage during the same 7-day period.

4. I will allow the researchers to perform the following procedures to determine body composition and caloric cost of running:

   a. Hydrostatic (underwater) weighing. This involves sitting in a chair in a water tank, exhaling to a maximum, and bending over until your body is completely under water while your weight is recorded. Since the level of the water in the tank is not over your head, you need only raise your head to breathe once more.

   b. Skinfold thicknesses will be measured at the triceps, scapula, suprailiac, abdomen and thigh by means of a caliper.

   c. Oxygen consumption will be measured while you are running for 10 minutes on a treadmill at your accustomed running pace. The sedentary control subjects will not undergo this procedure.

5. I will allow the researchers to draw blood from a fingerprick for determination of hemoglobin, hematocrit, protoporphyrin and ferritin and from a venipuncture for determination of plasma lipids. Minor discomforts may be associated with blood collection but there will be only minor risks since trained persons will be drawing the blood and there will be a chair and/or a cot available should you feel faint.

6. I have been completely informed as to and understand the nature and purpose of this research. The researchers have offered to answer any further questions that I may have. I understand that I will be able to withdraw from the study at any time of my own accord.

7. I realize that reports will be made of this study and I consent to publication of such if strict confidentiality is maintained by identifying my data only by a number and not by my name.

8. I have been informed that this study should increase our knowledge of the benefits of running for young women. The benefits to me will include gaining information about my 1) caloric and nutrient intake, 2) caloric cost of running, 3) body composition, 4) iron status and 5) blood lipid levels.

Date _____ Signed _____

DAILY FOOD RECORD

Subject Identification: ________________________

Date: ____________ Day of the Week: ________________________

Vitamin, mineral or nutrient supplement ________________________
Name and amount of supplement ________________________

TIME	MEAL OR SNACK (M OR S)	DESCRIPTION OF FOOD/BEVERAGE	AMOUNT

DAILY RUNNING AND FOOD RECORD

Subject Identification: _____

Date: _____ Mileage: _____

Day of the Week: _____ Time of day: _____

Minutes run: _____

Vitamin, mineral or nutrient supplement _____
Name of supplement and amount _____

TIME	MEAL OR SNACK (M OR S)	DESCRIPTION OF FOOD/BEVERAGE	AMOUNT

ADDITIONAL GUIDELINES

1. PLEASE EAT AS YOU ORDINARILY DO.
   This is very important because we are interested in your usual food intake.

2. MORE EXAMPLES AND INSTRUCTIONS:

Describe foods eaten as accurately as you can. Use brand names to clarify descriptions. Indicate method of preparation. For example:

Fruit juice: Orange, grape, tomato, grapefruit, V-8, Hi-C, Tang; fresh, frozen and reconstituted, canned, reconstituted powder

Cereal: Oatmeal, Wheaties; cooked, dry; with sugar added (granulated or brown); with whole milk

Bread or toast: White, whole wheat, cracked wheat, rye; homemade; dry; with butter, margarine, grape jelly

Milk: Whole, skim, 2 percent, reconstituted non-fat dry milk; chocolate

Coffee or tea: Black, with cream (half and half, Coffee Mate) or sugar

Mixed Dishes and Drinks: Give name of dish or drink and list the amount of ingredients consumed. If you give the recipe, indicate how much it makes. Be specific (if condensed soup, was it diluted with milk or water, etc.)

Fruits and vegetables: Raw or fresh, canned, frozen; plain, with butter, margarine, white or cheese sauce, sugar

Meats and fish: Fried, breaded, broiled, baked; trimmed of separable fat; cod, haddock, etc.

Estimate amounts eaten as carefully as you can and record amounts in household measures as suggested below:

	Household amounts
Beverages (fruit juice, milk, tea, coffee, soft drinks, liquor, etc.)	Measuring cups or ounces
Breakfast cereals, cooked vegetables, canned fruits, gravies, sauces, ice cream, nuts, snack foods	Measuring cups or tablespoons
Meats, cheeses, cake, pizza	Ounces or inches Example: 4" × 2" × 1/2"
Foods in small amounts (margarine, butter, sugar, grated foods, cream, etc.)	Teaspoons or tablespoons 1 pat butter = 1 teaspoon
Bread, packaged luncheon meats	Slices
Rolls, cookies, crackers, fresh fruits, boiled potatoes, etc.	Small, medium, large or inches

3. PLEASE KEEP A FOOD RECORD FOR THE 7 DAYS STARTING _____ AND ENDING _____.

4. IF YOU HAVE ANY QUESTIONS REGARDING THE FOOD RECORDS FEEL FREE TO CALL ME, KRIS WILLIAMS, 539-0243 (home) or 532-5508 (office) OR KAREN WIESE, 539-1502 (home) or 532-5508 (office).

GUIDELINES FOR YOUR FOOD RECORD

These guidelines will help you to describe the foods and beverages that you eat. It is important to understand and follow the guidelines so that you can make an accurate record of food intake.

Measure amounts in cups, ounces, etc. of serving sizes you most often consume so you can accurately record amounts consumed (i.e. if you normally drink the same amount of milk or eat the same amount of cereal, measure the amount so you will know how much it is without having to measure each time you consume that food). By doing this beforehand you will also have a better idea of serving sizes and can thus better judge amounts of food that you don't normally consume.

Please start a new sheet for every day of your intake. Record the date and day of the week on each sheet.

GUIDELINES

1. Record all foods and beverages after each meal or snack. Do not expect to remember all that you have eaten at the end of the day.

2. Record name and description of the food or beverage. Please include how the food was prepared and brand names when possible.

   EXAMPLE: 3/4 c Campbell's tomato soup vs. 3/4 c homemade chicken noodle soup

   1 fried chicken leg vs. 1 baked chicken thigh

3. Record the amount of each food and beverage that is eaten in standard measuring units - i.e. cups, spoons, etc.

   EXAMPLE: 1/2 c grape juice vs. 1 c grape drink

   1 teaspoon butter vs. 1 tablespoon margarine

4. Remember to include and record any additional sauces, gravies, salad dressing, margarine or sugar.

   EXAMPLE: 1/2 c mashed potato/ 2 Tbsp gravy

   3/4 c Rice Krispies with 1/2 c whole milk and 1 tsp sugar

5. Remember to include ALL between meal snacks and beverages. Include also beverages such as coffee (with cream or sugar), tea, diet or regular sodas and beer or any other alcoholic beverages.

6. Record any nutritional supplements or vitamins as a part of the food intake and give brand names.

7. For homemade dishes, you can either estimate the amount of each ingredient in the portion size, or list ALL the ingredients and the number of servings in the TOTAL recipe. Record these homemade dish recipes and their ingredients on the back of the food record sheet for that particular day.

CHOLESTEROL DETERMINATION (TOTAL, HDL, LDL + VLDL) WITH

MICRO-SCALE AFFINITY CHROMATOGRAPHY COLUMNS

Column separation of alpha and beta fractions per Isolab instructions using Isolab columns and elution fluids.

Determination of cholesterol can be done with other enzyme methods as long as samples are diluted correctly and the ratio of serum to enzyme is maintained (appropriate to the reagent directions).

Sigma quantitative, enzymatic determination of total and HDL cholesterol in serum at 500 nm (procedure No. 351).

Sigma procedure modified as follows:

1. Macro-method (greater than 1 ml reaction volume) for total cholesterol used for all determinations so that Brinkmann probe colorimeter could be used.

2. Total cholesterol serum samples and standards (50 and 200 mg) diluted 1:6 to match dilution factor of alpha and beta eluates.
   i.e. 0.2 ml serum or standard
   1.0 ml saline
   1.2 ml total volume = eluate volume and concentration

3. Once all serum, eluates and standards are at similar dilution, 0.12 ml is added to 1.0 ml of reagent so that correct ratio of serum to reagent is maintained (i.e. original Sigma method: 0.02 ml serum to 1.0 ml reagent = 1:50, therefore, diluted serum and standards use 0.12 ml (6×) to 1.0 ml reagent = 1:50).

4. More consistent results obtained if reagent is reconstituted (50 ml deionized water/bottle) several hours (or overnight) before use.

PROCEDURE: Separation of alpha and beta fractions (HDL and LDL cholesterol)

Ioslab LDL-Direct Cholesterol Audit System
#QS-8160 (60 test)
Isolab Inc.
Innovative Biochemical Methodology
Drawer 4350
Akron, OH 44321 800-321-9632

Bentzen, C.L., Acuff, K.J., Marechal, B., Rosenthal, M.A. and Volk, M.E. (1982) Direct determination of lipoprotein cholesterol distribution with micro-scale affinity chromatography columns. Clin Chem 28(7): 1451-1456.

1. Remove first the column's top cap, then the bottom closure. This order of opening is important - otherwise air will enter the column tip, interfering with free liquid flow.

2. Use the wide end of a Pasteur pipette to push the upper disc down until it contacts the top of the resin bed. Do not compress bed.

3. Allow the column to drain until the liquid level reaches the top disc, where flow will automatically stop.

4. Check to determine whether air may have entered the column during shipment. A few small air bubbles will not affect its performance. However, large volumes of air should be removed by tilting the top disc until the bubble escapes, then returning the disc to its original position.

5. Equilibrate the column bed by adding 1.0 ml of Alpha Fraction Elution Agent (Reagent #1) to the column. Allow column to drain. Discard eluate.

6. With the column positioned over a test tube (12 × 75 mm, 5 ml), add 0.2 ml patient serum to the column, near or on the upper disc. Collect the eluate.

7. Add 1.0 ml of Alpha Fraction Elution Agent (Reagent #1) and collect the entire volume in the same test tube, for a total fraction volume of 1.2 ml. Mix well.

8. Place the column over a clean 12 × 75 mm tube.

9. Add 1.2 ml of Beta Fraction Agent (Reagent #2) and collect the entire volume. Mix well.
Fill column with saline or eluted Alpha Elution Reagent, recap and store for possible regeneration.

PROCEDURE: Determination of Cholesterol (total, HDL, LDL)

Cholesterol, Total and HDL Quantitative, Enzymatic Determination in Serum or Plasma at 500 nm (Procedure No. 351). Cholesterol Reagent, Catalog No. 351-50, 50 ml size
Sigma Diagnostics
P.O. Box 14508
St. Louis, MO 63178
800-325-3010 (order)
800-325-8070 (service and technical information)

Cholesterol Standards:
Dow Diagnostics
The Dow Chemical Company
Indianapolis, IN 46268
200 mg/dl - 212688 lot #3M4L
expiration Dec. 1985
50 mg/dl - 213967 lot #3M4M
expiration Dec. 1985
Cholesterol in solution of ethylene glycol monomethyl ether and stabilizer

Procedure

Incubator at 37°C

Reagent should be reconstituted (50 ml deionized distilled water/bottle) several hours or overnight before assay.

Disposable glass tubes 12 × 77 mm (5 ml culture tubes)

A. Dilution of standards and serum for total cholesterol (1:6 to match dilution of alpha and beta fractions during separation).

1. Pipette 0.2 ml of each STANDARD (50 and 200 mg/dl) or SERUM SAMPLE for total cholesterol into tubes.

2. Add 1.0 ml SALINE to each of the above tubes and vortex.

B. Cholesterol Determination

1. Pipette 0.12 ml saline for blank (or distilled water)
0.12 ml diluted serum for total cholesterol
0.12 ml diluted standards
0.12 ml alpha fraction
0.12 ml beta fraction
(run duplicates of each, except blank)

2. To each tube add 1.0 ml cholesterol REAGENT.

Cover with parafilm and invert several times to mix (gently).

Cholesterol Determination (continued)

3. Incubate all tubes at 37°C for 12-15 minutes.

4. Following incubation, add 1.0 ml SALINE to all tubes and vortex gently. (can add 2.0 ml saline to increase volume to 3 ml to read in Spec. 20).

5. Read BLANK - 100% transmittance, 0% absorbance as reference at 545 nm (direction indicate 500 ± 15 nm but Brinkmann probe has filter at 545 nm). Read and record absorbance of STANDARD and SAMPLES.

   COMPLETE ALL READINGS WITHIN 30 MINUTES OF INCUBATION

6. Calculate cholesterol as follows:

$$\text{Cholesterol (mg/dl)} = \frac{\text{Absorbance}_{\text{Sample}}}{\text{Absorbance}_{\text{Standard}}} \times \text{Concentration of standard (50 or 200)}$$

7. Calculate percent recovery of alpha and beta fractions:

$$\text{\% Recovery} = \frac{\text{HDL (mg/dl)} + \text{LDL (mg/dl)}}{\text{Total Cholesterol (mg/dl)}} \times 100$$

   (recovery generally 94-97%)

TRIGLYCERIDE DETERMINATION

Sigma Quantitative Determination of Triglycerides in Serum or Plasma at 505-515 nm
#405
Sigma Chemical Company
St. Louis, Missouri

Reagents

1. Triglyceride purifier. Activated alumina.
2. Isopropyl, anhydrous.
3. Triolein standard. Contains 300 mg triolein (glycerol trioleate) dissolved in 1 dl anhydrous isopropanol.
4. Potassium hydroxide solution, 1 N.
5. Sodium m-periodate solution. Prepared by reconstituting a vial (125 g) of sodium m-periodate with 50 ml 2 N acetic acid solution.
6. Ammonium acetate solution 2 M.
7. H' color reagent. Prepare by mixing 20 ml ammonium acetate solution, 40 ml isopropanol (anhydrous) and 0.15 ml acetylacetone. Age overnight.

Procedure

A. Extraction

1. Label 3 or more tubes, add (0.8 g (± 0.2) triglyceride purifier to each tube. To blank add 5.0 ml isopropanol and 0.2 ml water; to standard 4.8 ml isopropanol, 0.2 ml water and 0.2 ml triolein standard; to test 5.0 ml isopropanol and 0.2 ml serum or plasma.
2. a) Shake with a mechanical mixer, or manually, for at least 5 minutes. b) Allow vials to stand for a few seconds until absorbent starts to settle. To facilitate separation of fluid from solids in the following step, give each vial a single sharp snap with the wrist while grasping the capped vial from the top. This will tend to wash most of the solids down to the bottom of the vial.
3. Centrifuge at about 3,000 rpm for 5 minutes to obtain clear supernatant.

B. Saponification

1. Label 3 or more clean tubes: glank, standard, test 1, test 2, etc.

Triglyceride Determination (continued)

2. Carefully transfer 2.0 ml of clear supernatant to bottom of the correspondingly labeled tube.

3. Into each tube pipet just above the liquid level 0.5 ml potassium hydroxide solution. Mix by swirling, do not invert.

4. Incubate all tubes at 60° (± 4°) for 5 minutes.

5. Remove tubes from water bath and cool to room temperature with tap water.

C. Oxidation

1. To each tube add 0.5 ml periodate solution. Mix immediately after each addition. Start timer after addition to the first tube and note time interval between additions.

D. Color Development

1. Ten minutes after addition of periodate solution to first tube, add to each tube 3 ml reagent H'.

2. Cover tubes and place in a 60° (± 4°) water bath for 30 minutes.

3. Remove tubes from water bath and cool to room temperature with tap water.

4. Transfer contents of tubes to correspondingly labelled cuvettes. Read absorbance (A) of the standard and test versus blank as reference at the same wavelength used to prepare calibration curve. Reading should be completed within 20 minutes.

Calculate Triglycerides as follows:

Determine the value for both the standard and test from the calibration curve. The absorbance (A) of the standard should correspond to a value between 280 and 320 mg/dl as read on your calibration curve. If it does the following equation may be used:

$$\text{Triglycerides mg/dl} = \frac{\text{A (test)}}{\text{A (standard)}} \times 300$$

TABLE 7. Physical characteristics of each subject.

Subject Number	VARIABLE		
	Age (yrs)	W/H (kg/m)	% Body Fat
	Inactive Subjects		
1	24	34.5	26.9
2	22	44.5	38.2
3	22	35.0	39.6
4	30	33.5	27.4
5	25	34.0	29.7
6	22	41.8	31.6
7	21	32.3	32.2
8	29	34.4	35.4
9	22	36.7	37.3
10	22	36.8	28.5
	Low Mileage Runners		
11	29	34.6	17.2
12	21	29.0	22.5
13	21	34.6	20.2
14	25	31.9	26.2
15	26	32.3	22.7
16	24	33.3	19.6
17	21	39.0	22.1
18	21	31.7	23.1
19	21	28.8	18.7
20	27	37.0	26.4
	High Mileage Runners		
21	20	33.3	19.0
22	31	29.1	20.4
23	24	32.5	20.5
24	32	33.4	19.7
25	23	34.3	17.5
26	31	33.3	15.7

TABLE 8. Nutrient intakes of each subject.*

Subject Number	VARIABLE											
	Kcal	Pro	CHO	Fat	S.F.	Alc	Pro	CHO	Fat	S.F.	Chol	C.F.
		(% of Kcals)					(gm)				(mg)	(gm)
Inactive Subjects												
1	1866	16.0	49.8	33.1	12.1	2.2	74.4	232.1	68.6	25.0	354	3.8
2	1545	14.7	44.3	43.2	23.1	0.0	56.8	171.0	74.2	39.6	258	2.4
3	2082	10.7	55.6	33.6	15.6	1.4	54.9	288.6	72.8	26.3	267	2.9
4	1961	18.3	44.9	38.9	14.0	0.0	89.7	220.1	84.6	30.5	374	4.9
5	1547	14.0	45.8	41.8	16.2	0.0	53.9	177.0	71.8	27.8	152	3.2
6	1963	13.5	54.7	33.9	13.7	0.0	66.4	268.7	73.8	29.8	314	6.0
7	1517	14.5	54.9	30.3	12.2	2.0	55.1	208.0	51.1	20.6	174	3.0
8	1817	16.1	49.7	35.6	11.8	0.0	73.2	225.7	71.9	23.8	634	3.5
9	2206	9.9	57.6	33.5	11.5	1.1	54.4	317.8	82.1	28.1	233	2.2
10	1453	13.4	47.3	41.5	14.4	0.0	83.5	171.7	67.9	23.2	161	3.0
Low Mileage Runners												
11	2265	12.9	46.3	42.8	13.1	0.0	73.2	262.3	107.6	35.5	267	3.6
12	1716	16.5	52.4	32.0	12.2	1.5	70.8	224.9	61.0	23.3	182	6.0
13	1076	18.7	35.6	46.5	20.2	0.0	50.4	95.8	55.5	24.2	165	1.5
14	1381	22.1	62.1	20.7	6.3	0.0	62.3	234.4	27.8	9.6	352	8.6
15	1434	16.8	54.9	30.3	10.8	0.2	60.4	196.7	48.3	17.2	208	6.2
16	1695	12.6	58.0	33.5	13.2	0.0	53.3	245.9	63.1	24.9	142	5.9
17	1895	16.8	43.1	41.1	14.2	0.9	79.7	204.1	86.5	29.8	207	4.0
18	1550	15.9	37.3	44.7	19.5	2.5	61.6	144.5	76.9	33.5	342	1.8
19	1581	12.6	48.8	40.3	15.2	0.6	50.0	192.9	70.7	26.7	218	4.2
20	1642	14.5	52.9	36.5	14.2	0.0	59.7	217.3	66.6	25.9	164	5.7
High Mileage Runners												
21	1004	17.2	38.4	37.9	15.3	10.2	43.3	95.4	42.2	17.0	111	1.5
22	1332	18.5	50.6	31.8	13.4	0.9	61.7	168.4	47.0	19.9	367	3.0
23	1346	15.9	54.5	31.8	10.6	0.0	53.6	183.4	47.5	15.9	242	5.3
24	2309	14.9	60.4	25.2	9.3	2.7	85.9	348.7	64.6	23.8	280	10.8
25	1896	16.3	62.7	22.4	8.5	2.1	77.4	297.3	47.2	18.0	152	8.4
26	3358	24.5	25.9	46.6	22.7	2.7	205.8	217.3	173.9	84.7	2591	3.6

Values are means over a 7 day period.

* Pro = protein, CHO = carbohydrate, S.F. = saturated fat, Alc = alcohol, Chol = cholesterol, C.F. = crude fiber.

TABLE 9. Serum lipids of each subject.

Subject Number	VARIABLE (mg/dL) Total* Cholesterol	HDL* Cholesterol	LDL† Cholesterol	LDL + VLDL* Cholesterol	Triglycerides
			Inactive Subjects		
1	133.4	41.6	73.2	90.1	93.0
2	188.6	64.7	101.3	113.2	97.2
3	151.2	57.5	83.8	84.9	49.3
4	172.2	60.4	100.9	101.1	54.3
5	176.3	65.7	89.9	100.4	103.9
6	133.2	53.4	66.1	76.3	68.3
7	121.3	48.8	66.9	67.1	28.1
8	159.8	45.0	104.4	105.6	52.3
9	157.8	67.5	81.5	91.3	44.1
10	151.9	57.3	86.5	83.9	40.4
			Low Mileage Runners		
11	156.5	67.0	74.8	77.6	73.4
12	155.5	54.8	92.1	90.9	43.1
13	109.9	48.5	49.2	55.8	61.0
14	179.4	82.2	89.5	93.4	38.9
15	168.2	64.7	98.1	94.2	26.7
16	162.8	59.7	96.5	111.2	33.4
17	169.2	51.9	108.6	109.3	43.1
18	194.9	63.6	121.5	124.0	49.0
19	126.4	50.4	67.9	78.7	40.6
20	199.6	67.8	119.5	112.6	61.6
			High Mileage Runners		
21	188.7	77.1	95.7	93.3	49.8
22	192.1	56.2	122.5	125.6	67.1
23	191.4	50.4	126.4	131.4	73.1
24	166.8	56.6	89.8	102.7	102.1
25	178.1	46.5	121.9	114.7	48.7
26	228.9	80.6	138.1	144.6	50.6

* The sum of HDL-cholesterol and LDL + VLDL-cholesterol may not equal total cholesterol due to variation from 100 in the % recovery of these lipoprotein fractions.

† LDL-cholesterol was calculated using the equation of Friedwald et al. (18).

INFLUENCE OF DIET AND EXERCISE INTENSITY ON SERUM LIPIDS AND LIPOPROTEINS IN YOUNG FEMALE RUNNERS

by

KAREN WIESE SADEGHIAN

B.S., New Mexico State University, 1983

AN ABSTRACT OF A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Food Science and Nutrition

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1985

The influence of dietary intake and exercise intensity on serum levels of total cholesterol (TC), triglycerides (TG), and high- and low-density lipoprotein cholesterol was studied in 26 females between the ages of 20 and 32 years. Subjects were categorized into 3 groups according to their habitual level of physical activity. Six subjects were included in a group of high mileage runners (HM) who ran 40-50 miles per week, and 10 subjects made up a group of low mileage runners (LM) who ran 20-30 miles per week, and 10 subjects not involved in any exercise program served as inactive controls (I).

Concentrations of TC and low-density lipoprotein cholesterol (LDL-C) were higher ($P \leq 0.05$) in HM than in either I or LM (191.0 vs 154.6 and 162.2 mg/dL, respectively). No significant differences in high-density lipoprotein cholesterol (HDL-C) existed between groups, although LM and HM tended to have higher HDL-C levels than I (61.1 and 61.2 vs 56.2 mg/dL, respectively). All groups were within average TC and HDL-C ranges for their age and sex, except that HM had higher than average levels of TC (191.0 vs 170-179 mg/dL). The HDL-C ratio was higher ($P \leq 0.05$) in LM (0.38) compared to HM (0.32), with I showing an intermediate value (0.36).

Age and protein both were correlated ($P \leq 0.05$) with levels of TC and LDL-C. These correlations may partially explain why HM had higher ($P \leq 0.05$) levels of TC and LDL-C than I or LM, as HM also had higher ($P \leq 0.05$) protein intakes and tended to be older than the other two groups (26.8 vs 23.9 and 23.6 years).

Both saturated fat and cholesterol intakes were correlated with TC ($P \leq 0.10$ and $P \leq 0.05$, respectively) and HDL-C ($P \leq 0.10$) concentrations, and cholesterol intake was also correlated ($P \leq 0.10$) with levels of LDL-C. However, no significant differences in these nutrients were found

among groups. HM consumed more ($P \leq 0.05$) alcohol than either I or LM, but alcohol was not related to any serum lipid measurement. Furthermore, no significant correlations were found between any lipid parameter and any of the following variables: % body fat, weight/height, calories, carbohydrate, total fat, or fiber.