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Title: THE EFFECTS OF A PHYSICAL FITNESS PROGRAM ON THE PLASMA LIPIDS OF LATE ADOLESCENT MALES

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The effects of an 11-week jogging program on the plasma lipid fractions and clotting time of 19 late adolescent males (18 to 19 years) were investigated. Total lipids, triglycerides, total and free cholesterol, phospholipids, non-esterified fatty acids, and clotting time of plasma were determined. Statistical analysis was applied to the change in each parameter. Dietary patterns prior to and following the fitness program were examined.

The mean concentrations of total lipids, total and free cholesterol, and phospholipids were lower than those reported for young men; plasma clotting times were longer. The mean plasma triglycerides and non-esterified fatty acids were comparable to those observed in adults. Individual values of the plasma lipid fractions were within the ranges reported in the literature. Consistent relationships could not be found between dietary intake, changes in

weight, or any of the lipid parameters.

Significant changes in triglycerides, total and free cholesterol, and non-esterified fatty acids were not apparent as a result of exercise. There was a significant decrease in phospholipids; total lipids and clotting time increased significantly following the fitness program.

The Effects of a Physical Fitness Program on the Plasma Lipids of Late Adolescent Males

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THE EFFECTS OF A PHYSICAL FITNESS PROGRAM ON THE PLASMA LIPIDS OF LATE ADOLESCENT MALES

INTRODUCTION

The high incidence of cardiovascular disease among the people of the Western world has led to a concerted effort toward its prevention. In recent years, interest has focused on the effects of physical activity on coronary heart disease and on the hyperlipemia that usually accompanies it.

A number of studies indicate that men in sedentary occupations show a greater tendency to coronary heart disease than do men whose jobs require physical exertion. For example, Morris and coworkers (1953) reported that heart disease was more frequent among drivers of the London Transport system than among the more active conductors. The conductors not only showed a lower mortality rate but also a delayed onset of the disease. Taylor et al. (1962) observed a similar pattern among men employed by the railroad industry. The clerks had a higher mortality from arteriosclerotic heart disease than did the sectionmen (5.7/1000 v.s. 2.8/1000). Both Morris and Taylor attributed their findings to the differences in physical activity associated with occupation.

Physical fitness programs have produced sustained reductions in the plasma lipids of hyperlipemic individuals (Garrett, Pangle

and Mann, 1961). Montoye et al. (1957) attributed the lipid lowering effect of exercise to a concomitant loss of body weight. On the other hand, it is apparent that even subjects who are in caloric balance draw on plasma fatty acids for muscular energy (Friedberg et al., 1963). Thus one might anticipate that regular physical exercise, by increasing lipid metabolism, would tend to reduce the circulating lipids of healthy normolipemic subjects, also. However, Johnson and Wong (1961) reported that a controlled exercise program did not alter the plasma lipids of young athletes.

The influence of physical activity on blood lipids has been investigated chiefly in older men. A few studies of young men are available but little has been reported concerning the lipid profile of late adolescent boys. The purpose of this study was to evaluate the effects of an 11-week physical fitness program on the plasma lipids of 18 to 19 year old males.

REVIEW OF LITERATURE

Fat as a Source of Energy

The immediate source of energy for muscle contraction is adenosine triphosphate. Ultimately, however, energy is derived from the oxidation of either glucose or fatty acids. As early as 1928, Bock et al. concluded, from respiratory quotients (RQ) obtained during exercise at a steady state, that carbohydrate served as a primary source of energy. They found that prolonged exercise resulted in a reduction of the RQ, presumably due to an increased utilization of fat. More recently, Hermansen, Hultman, and Saltin (1967) showed that prolonged exercise induced a depletion of muscle glycogen followed by a decline in RQ. Trained athletes maintained a lower RQ during exercise than did untrained subjects. Åstrand (1967) concluded that the decrease in RQ indicated an increased utilization of fat for energy, and that trained athletes were more efficient in the use of fat.

A number of studies indicate that lipid energy is supplied to the cells in the form of non-esterified fatty acids (NEFA). Gordon and Cherkes (1956) showed that plasma NEFA are immediately available to the cells for energy demands. These authors demonstrated that the NEFA values of patients and healthy subjects decreased below fasting levels one to two hours after the ingestion of 100

grams of glucose. By four hours after the glucose meal, blood glucose had returned to fasting levels and NEFA had increased sharply. This suggested that, if energy requirements were satisfied by carbohydrate, the circulating NEFA would decrease.

Basu, Passmore, and Strong (1960) reported that plasma NEFA levels increased during exercise. There was a concurrent decrease of blood glucose, but a statistical correlation could not be found between the two parameters.

Carlson and Pernow (1959) measured the arteriovenous differences of NEFA in exercising and in resting leg muscles of young men. At moderate exercise, a positive arteriovenous difference was noted; at maximal exercise, a negative arteriovenous difference occurred. The authors concluded that during moderate exercise the leg muscles were extracting NEFA from the blood. At maximal exercise NEFA extraction declined, presumably because there was insufficient oxygen to support aerobic metabolism of NEFA.

A further study by Carlson and Pernow (1961) demonstrated the effect of different work loads on NEFA concentrations. The plasma NEFA decreased at the initial stage of a prolonged, but constant, work load. After ten minutes, NEFA increased to the pre-exercise levels. During an uninterrupted work series, consisting of an increased work load every six minutes, plasma NEFA

decreased with each increment of exertion. Within five minutes after cessation of exercise, the NEFA values doubled. It was concluded that the muscles removed NEFA from the blood during exercise to satisfy the energy requirements. An efflux of NEFA from tissue to plasma occurred at the end of exercise, probably due to a sudden decrease in utilization.

Direct evidence that fatty acids are oxidized by exercising tissue was provided by Friedberg and Estes (1962), who measured the conversion of radiopalmitate to $^{14}CO_2$. In a later study, Friedberg et al. (1963) showed that NEFA were continuously extracted from the plasma throughout exercise. These authors attributed the initial decline in plasma NEFA to tissue uptake. The subsequent rise in plasma NEFA was taken as evidence that fat mobilization had become fully operative.

Although the mechanism for NEFA mobilization from adipose tissue has not been clarified, several hormonal factors have been delineated. Gordon and Cherkes (1956) observed that the infusion of epinephrine to humans caused a sharp elevation of plasma NEFA, lasting approximately 20 minutes. Shafrir and Steinberg (1960), using dogs, implicated the adrenal cortex function in NEFA mobilization. Epinephrine injections in normal dogs caused NEFA levels to rise. After either adrenalectomy or hypophysectomy, the circulating NEFA failed to respond to epinephrine; however, treatment with

cortisone restored the NEFA response. The authors concluded that mobilization of NEFA by epinephrine is dependent upon the adrenal cortex function and on the availability of cortisone-like steroids.

Havel and Goldfien (1960) found that epinephrine and nor-epinephrine elevated plasma NEFA in humans and dogs. Furthermore, the two hormones efficiently abolished the depression of plasma NEFA resulting from a ganglionic block. Carlson et al. (1963) used nicotinic acid as an adrenergic blocking agent in resting and in exercising subjects. At rest, nicotinic acid decreased the rate of NEFA mobilization; this effect was magnified during exercise. When nor-epinephrine was injected into the subjects treated with nicotinic acid, NEFA increased. It was believed that nicotinic acid prevented the fat-mobilizing action of the catecholamines at their receptor sites in the adipose tissue.

Response of Plasma Lipids to Exercise

Cholesterol

An elevation in serum cholesterol has long been identified as a coronary risk factor. In the Framingham study (Kannel et al., 1964), both the incidence and severity of coronary heart disease were more closely correlated with hypercholesteremia than with any other single factor. Chapman and Massey (1964) reported

similar findings after a ten year study in Los Angeles. Myocardial infarction occurred most frequently among the men who had the highest serum cholesterols; neither the infarction nor the serum cholesterol could be related to overweight.

Garrett, Pangle and Mann (1961) attempted to modify the coronary risk factors (hypercholesteremia, hypertension, obesity) by a course of vigorous physical exercise. Graduate students displaying one or more of these factors engaged in an eight-week program of calisthenics and jogging. Despite an increased caloric intake, the coronary risk factors were lowered in all subjects. Particularly noteworthy was the reduction in plasma cholesterol. The cholesterol level did not increase during a four-week observation period following the exercise regimen.

Other reports support the theory that physical activity plays a role in moderating cholesterol levels. Stulb et al. (1965) studied the serum cholesterols of white males living in Evans County, Georgia. Occupation served as a criterion for physical activity. Statistical analysis showed an inverse relationship between the cholesterol levels and the amount of physical exertion required by the occupations. Correlation with dietary variables was not significant.

Similar conclusions may be drawn from studies of Air Force personnel. Shane (1966) reviewed the clinical records of all the flight personnel evaluated at the School of Aerospace Medicine during

1963-1964. The subjects studied included healthy individuals as well as those exhibiting some systemic disorders. All of the subjects had participated in treadmill exercises for varying periods. Serum cholesterols were lower in those men who had spent longer times on the treadmill. Hoffman (1967) studied healthy senior Air Force officers stationed at the Pentagon. The officers who had made a practice of performing dynamic physical exercises showed lower serum cholesterols than did the physically inactive officers.

The variability of plasma cholesterol during and after exercise has been noted. Rochelle (1961) studied 12 male subjects during a 15 week period. The subjects ran two miles a day for five weeks. Analysis of plasma cholesterol showed a decrease after the exercise period. A temporary rise of cholesterol was noted while exercise was in progress. Four weeks after the exercise had ended, the plasma cholesterols had returned to the pre-training levels.

Still other studies suggest that exercise may exert a lipid-lowering effect through the reduction of body weight. Trulson et al. (1964) compared Irish-born Boston residents with their brothers living in Ireland. Despite the fact that they consumed more calories with a higher percentage of fat, the brothers living in Ireland had significantly lower serum cholesterols than did their counterparts in Boston. Investigation into the living habits of the two groups revealed that the Irish Americans were heavier and less physically active.

The serum cholesterols of faculty members completing a moderate exercise regimen were studied by Montoye et al. (1957). The authors concluded that the effect of exercise was indirect, and that any decrease in cholesterol concentration was associated with the subjects' weight losses. Golding (1961) reported similar results.

A study on the combined effects of low caloric intake and exercise on lipid levels indicated that cholesterol levels were associated with weight (Carlson and Fröberg, 1967). The subjects walked 50 km. a day and consumed a diet containing 200 Calories. The serum cholesterols decreased steadily from the first to the tenth day of the study. The decrease correlated well with the steady weight loss of all the subjects.

Not all investigators agree that physical activity is a major factor in reducing serum cholesterol. Keys et al. (1956) studied populations differing in dietary habits and physical activity. The population samples were drawn from five foreign cities and one United States city. Serum cholesterols showed little relation to the physical activity of the subjects; however, as the percentage of fat decreased in the diets of each group, the serum cholesterols were lower. In a later study (Taylor, Anderson and Keys, 1957a) these investigators showed that serum cholesterol levels of young men remained unchanged at two levels of physical activity and caloric intake, so long as the type of fat and the

percentage of fat in the diet remained constant.

Reports concerning the effects of exercise on cholesterol levels in young men have revealed little correlation between the two. Johnson et al. (1959) studied the influence of controlled exercise on the serum lipids of college swimmers. Even after several months, there were no changes in the serum cholesterols. Johnson and Wong (1961) concluded, from a similar study, that greater exertion would have been necessary to produce any change since individual cholesterol levels were low initially. Calvy et al. (1963, 1964) conducted studies with young Marine recruits who were involved in the rigors of basic training. The results showed that the serum cholesterols did not differ statistically over a five and a half month period. The recruits' diets were unrestricted and averaged 4500-5200 Calories of which 45% was derived from fat.

An investigation by Campbell (1965) indicated that various types of physical activity had different effects on serum cholesterol. Subjects consisted of freshmen males enrolled in various physical education courses of ten week's duration. Campbell defined two types of physical activity as phasic and static. Phasic activity involved a rapid interchange of arms and legs with a rapid flexing and relaxing of the muscles (cross-country running and tennis). Static exercise occurred when muscles were held in a prolonged state of contraction with a slower rate of muscle flexing and

relaxing (wrestling, weight training, and tumbling gymnastics). Campbell found a decrease of serum cholesterols with phasic activities and little change with static activities.

Triglycerides

Serum triglycerides are also known to be elevated in patients with coronary heart disease. Albrink, Meigs and Man (1961) reported that 82% of the coronary patients whom they studied had serum triglycerides which were considerably higher than those of healthy individuals. The authors speculated that an error in the metabolism of triglycerides might be operative in coronary heart disease.

Several studies have indicated that plasma triglycerides are altered by exercise. Calvy et al. (1963) reported that the triglycerides of Marine recruits were the most variable of all the lipids analyzed. During the first months of basic training the triglyceride concentrations increased two-fold. Then, the values decreased sharply, only to rise again toward the end of the five and a half month period.

The effect of skiing on plasma lipid levels was studied by Carlson and Mossfeldt (1961). There was a definite decrease in plasma triglycerides immediately after skiing when compared to the triglyceride values of the same subjects obtained after normal activities. The authors suggested that the decline of plasma

triglycerides during exercise was caused by decreased formation of the triglycerides in the liver as a result of lowered hepatic uptake of plasma NEFA. This would result in a decreased influx of triglycerides to the plasma. They also implied that an increased removal of triglycerides from the plasma might occur because of an increased uptake of triglyceride fatty acids by the working muscles. This uptake might be regulated by the action of lipoprotein lipase which increases during exercise.

Shane (1966), in his review of clinical records of Air Force flight crews, showed an inverse correlation between treadmill time and serum triglycerides. The plasma triglycerides of senior Air Force officers were only slightly depressed by physical activity (Hoffman et al., 1967).

Holloszy et al. (1964) studied the effects of a six-month program of exercise on lipids of middle-aged men. By the end of the study, serum triglycerides had decreased to one half of the pre-training values. The reduction in triglycerides appeared to be an acute effect which occurred shortly after exercise and usually persisted approximately two days without exercise. It was concluded that regularly performed exercises would maintain a reduction in triglycerides.

Other Lipids

Less significance has been attributed to the effects of exercise on lipid phosphorus. Several reports have shown little or no change in blood phospholipids during or after exercise (Johnson and Wong, 1961; Calvy et al., 1963; Holloszy et al., 1964; Shane, 1966; Garrett, Pangle and Man, 1966; Hoffman, Nelson and Gross, 1967). Taylor, Anderson and Keys (1957b) reported that phospholipids declined after four weeks of increased physical activity. However, the level increased to pre-exercise values after one week of a sedentary period. The plasma phospholipids of skiers decreased immediately after ski racing, but no explanation for this observation was given (Carlson and Mossfeldt, 1964). Carlson and Fröberg (1967) also reported a marked lowering of phospholipids during a ten-day period of exercise and low caloric intake.

Total lipids have shown little difference after long term exercise programs (Fitzgerald, Heffernan and McFarlane, 1965; Hoffman, Nelson and Gross, 1967; Zauner and Swenson, 1967).

Clotting Time

The blood lipids are closely associated with the blood coagulation process. It is well known that lipemia will result in a shortened plasma clotting time and an inhibition of fibrinolysis (O'Brien, 1957;

Greig, 1956; Greig and Runde, 1957). The platelet lipids, phosphatidylserine and phosphatidylethanolamine, function in the stages of prothrombin activation (Merskey and Marcus, 1963; Marcus, 1966). The relationship of the coagulation process to coronary heart disease is less well established. Astrup (1959) proposed that a dynamic equilibrium exists between fibrin formation and fibrinolysis. He theorized that if this balance were shifted, the risk of coronary artery disease would be enhanced. Panchenko and Bazaz'yan (1965) found signs of increased blood coagulability and depressed fibrinolysis in patients suffering from coronary insufficiency. Similarly, Chakrabarti et al. (1968) reported that men who had suffered myocardial infarction showed persistently defective fibrinolysis.

Some evidence has indicated that muscular exertion might affect the blood coagulation and fibrinolysis sequence. Keys and Buzina (1956) observed that the blood of business and professional men coagulated more rapidly than did that of physically active railroad workers. On the other hand, Keeney (1959) found no significant changes in whole blood clotting time immediately after exercise. More recently, Burt, Blyth and Rierson (1964) reported a shortened clotting time but accelerated fibrinolytic activity in healthy college men after exercise. These authors concluded that exercise may alter the coagulation-fibrinolysis equilibrium to favor the fibrinolytic process. Cohen et al. (1968) also found a significant acceleration of

fibrinolysis at the conclusion of exercise. Similar observations were reported by Ogston and Fullerton (1961) who suggested that regularly performed exercise, by increasing fibrinolytic activity, may be a factor in the prevention of coronary artery disease.

Plasma Lipids of Healthy Adults

The plasma lipid concentrations vary widely among healthy individuals, and the values reported can vary with different analytical methods. In a compilation of data from several investigators, Henry (1964) has presented ranges of plasma lipids which have been observed in healthy adults. Total lipids may vary from 450-1000 mg/100 ml; triglycerides, 29-134 mg/100 ml; phospholipids, 125-300 mg/100 ml; NEFA, 0.45-0.90 mEq/l; and cholesterol, 110-356 mg/100 ml. Free cholesterol is estimated at approximately 22% to 30% of total cholesterol. The lipid values are reported to be slightly higher in females than in males; however, the differences are small (Kornerup, 1950). Most of the lipids tend to increase with age; this effect is most noticeable for total cholesterol levels (Kornerup, 1950; Keys et al., 1950; Aldersberg et al., 1956; Lopez-S, Krehl and Hodges, 1967).

In several studies the plasma lipid profiles of younger men have been investigated. The mean values obtained in two such studies (Hallgren et al., 1960; Svanborg and Svennerholm, 1961)

are presented in Table 1. In both cases the subjects were in the post-absorptive and non-active state. There appear to be no similar reports of plasma lipid concentrations of men under 20 years of age. One of the objectives of this study was to establish such values.

Table 1. Plasma lipids of young males.

Reference	Subject	Age	Total Lipid	Triglyceride	Cholesterol			Phospholipid	NEFA
					Total	Free	Free/Total		
	no.	yr	mg/100 ml	mg/100 ml	mg/100 ml		%	mg/100 ml	
Hallgren et al. (1960)	8	23-38	588	76	224	69	27	197	19 mg/100 ml
Svanborg & Svennerholm (1961)	62	16-35	609	83	191	64	34	207	0.75 mEq/l

EXPERIMENTAL PROCEDURE

Experimental Plan

Subjects

Twenty-two male students, who were enrolled in the Fitness Appreciation course (PE 150), volunteered for this study. The subjects were apparently healthy, and their ages ranged from 18 to 19 years. Three subjects were later dropped from the study: two had eaten prior to the drawing of the blood samples, and the third had limited participation in the fitness program because of an injury.

Physical Fitness Program

The 11-week program consisted chiefly of running or jogging, followed by swimming for relaxation. There were ten running courses, varying from 2 to 12 miles in length. Twice a week, each subject and a partner ran a selected course in 45 to 50 minutes. On the third day, the subjects participated in a planned group physical activity. The students were allowed to progress at their own speed, but by the end of the program all were expected to run a 10-mile course within 90 minutes.

Dietary History

Each of the subjects kept a three-day dietary record before and after the 11-week fitness program. Dietary interviews were

conducted to determine the accuracy of these records. The average daily intake of nutrients was estimated from data in standard food tables (Watt and Merrill, 1963; Church and Church, 1963).

Blood Samples

Approximately 20 ml of venous blood were drawn from fasting subjects at the beginning and again at the end of the fitness program. The blood was collected in 10-ml vacutainers which had been treated with EDTA (Ethylenediaminetetraacetic acid). The plasma was separated at once by centrifugation at 2800 rpm for ten minutes. A small portion of the plasma was reserved for clotting time determinations; the remainder was stored at -10°C for future analyses.

Methods of Blood Analysis

Total Lipid Extraction

The procedure of Smith (1965) was used to extract 2 ml of plasma into chloroform-methanol, 2:1, V/V. Lipid extracts were evaporated under nitrogen in tared 1-ml volumetric flasks. The weights of the dried lipid extracts were expressed as mg/100 ml plasma. The extracts were redissolved in chloroform and aliquots were taken for the analysis of triglycerides, free and total cholesterol, and phospholipids.

Triglycerides

Two hundred microliters of lipid extract were used for the determination of triglycerides by a modification of the procedure of Van Handel and Zilversmit (1957). Phospholipids were removed by adsorption onto silicic acid as described by Chiu (1969). The triglycerides were then saponified to release glycerol, glycerol was oxidized to formaldehyde, and the formaldehyde was treated with chromotropic acid. Optical density of the resulting colored solution was measured photometrically (Beckman Model DU Spectrophotometer). Triglycerides were expressed as mg/100 ml plasma.

Free and Total Cholesterol

The lipid extract was diluted 1:10 with chloroform. One hundred microliter aliquots of the diluted lipid extract were used for the determination of free cholesterol and 20 µl aliquots for total cholesterol. To obtain the total cholesterol, samples were saponified and neutralized prior to cholesterol determination. Cholesterol was precipitated as the digitonide; the digitonide was then treated with the Liebermann-Burchard color reagent and the colored complex was measured photometrically. The microprocedure described by Smith (1961) was used with two modifications. First, the optical density of the color produced was read at 45 minutes rather than 30

minutes after addition of the color reagent (Chiu, 1969). Second, cholesterol standards were carried through the entire procedure for both total and free cholesterol determinations. Samples were placed in microcuvettes and the optical density was measured in a Beckman Model DU Spectrophotometer. Cholesterol has been reported as mg/100 ml plasma.

Phospholipids

Lipid phosphorus was determined by the microprocedure of Lowry et al. (1954) as modified by Hawthorne, Smith and Pescador (1963). A sample of lipid extract corresponding to 16 μl plasma was used. Briefly, the method consisted of the oxidation of lipid phosphorus to inorganic phosphate, the formation of phosphomolybdic acid, and the reduction of phosphomolybdic acid to molybdenum blue which was measured photometrically. Samples were placed in microcuvettes and the optical density was measured in a Beckman Model DU Spectrophotometer. The plasma phospholipid concentration, in mg/100 ml plasma, was obtained by multiplying the phosphorus concentration by 25. Phosphorus constitutes about 1/25 of the weight of most phospholipids.

Non-esterified Fatty Acids

The non-esterified fatty acids were determined essentially by

the method of Dole (1965). Lipids were extracted from 2 ml of plasma with a mixture of isopropyl alcohol, heptane, and water, 39:10:1, V/V. Aliquots of the heptane phase were titrated with sodium hydroxide delivered from a digital readout pipet (Manostat Digi-Pet). Nile blue was used as the indicator rather than thymol blue as described by Dole, and the endpoint was violet to light pink in color. The non-esterified fatty acids were expressed as mEq/l of plasma.

Clotting Time

The modified Howell procedure, as described by Davidsohn and Wells (1962) was used for the determination of the clotting time for plasma. The test measured the time required for the fibrin clot to form in decalcified plasma after addition of calcium chloride. The time required for the fibrin clot to form was recorded in seconds.

RESULTS AND DISCUSSION

The total lipids and lipid fractions of plasma samples obtained prior to the exercise program are presented in Table 2; values obtained after the 11-week course are shown in Table 3. In order to determine the effect of exercise on the lipid parameters, the amount of change between the first sample and the second sample was calculated for each individual. Statistical analysis of the change in each parameter was performed by application of Student's t test. A two-tailed test was used with the critical value of t taken at $t_{.05}$ and at n-1 or 18 degrees of freedom. Standard deviations were also determined for each parameter.

With few exceptions, the plasma lipid concentrations of these late adolescent males were within the ranges which are typical of healthy adults (Henry, 1964). The overall mean triglycerides (76 mg/100 ml) and NEFA (0.66 mEq/l) approximated the values reported for men in the 16 to 35 year age range (Table 1). However, the mean concentrations of total lipids (486 mg/100 ml), total cholesterol (141 mg/100 ml), free cholesterol (49 mg/100 ml), and phospholipids (158 mg/100 ml) are considerably lower than those shown in Table 1. The finding of low concentrations of lipids in plasmas of 18 to 19 year old boys was not unexpected. Many investigators have noted that cholesterol values increase with age (Keys et al., 1950; Lopez-S, Krehl and Hodges, 1967). The data of Adlersberg et al. (1956)

Table 2. Plasma lipids before exercise program.

Subjects	Age	Height	Weight	Total Lipid	Triglyceride	Cholesterol			Phospholipid	NEFA	Clotting Time
						Total	Free	Free/Total			
	yr	in	lb	mg/100 ml	mg/100 ml	mg/100 ml		%	mg/100 ml	mEq/l	sec
B	18	71	150	408	55	160	60	38	165	0.45	172.3
C	19	72	160	428	74	133	48	36	138	0.31	159.3
D	18	70	147	457	61	150	57	38	176	0.39	200.7
E	18	72	155	467	69	133	53	40	118	0.60	175.0
G	18	68	141	336	41	99	36	36	181	1.01	180.3
H	19	72	167	411	62	124	42	34	160	1.18	157.3
I	18	70	175	442	66	149	55	37	150	0.57	179.7
J	18	72	157	516	119	142	52	37	179	0.79	203.0
K	18	72	168	455	50	125	47	38	164	1.00	189.3
L	18	70	164	507	73	147	49	33	179	1.08	167.7
N	19	68	169	530	116	147	52	35	177	0.35	137.3
Q	19	73	188	506	78	163	60	37	189	0.65	213.0
R	19	72	155	501	60	156	51	33	182	0.46	149.3
S	19	69	149	432	75	121	40	33	168	0.78	149.3
T	19	74	184	574	114	152	46	30	194	0.97	160.3
U	19	72	173	515	102	127	42	33	179	0.76	170.7
W	18	74	155	381	47	107	37	35	118	0.76	154.7
X	19	67	123	457	76	140	56	40	165	0.37	187.3
Y	19	70	162	497	53	151	55	36	167	1.15	172.3
Mean				464	73	138	49.4	36	163	0.72	172.6
Standard Deviation				± 56	±23	±17	±7.2		±21	±.28	±19.5

Table 3. Plasma lipids after exercise program.

Subjects	Weight	Total Lipid	Triglyceride	Cholesterol			Phospholipid	NEFA	Clotting Time
				Total	Free	Free/Total			
	lb	mg/100 ml	mg/100 ml	mg/100 ml		%	mg/100 ml	mEq/l	sec
B	157	662	52	170	55	32	195	0.65	187.7
C	161	456	120	137	48	35	109	1.27	169.0
D	147	492	100	123	38	31	145	0.33	166.7
E	165	452	37	145	53	37	141	0.54	190.0
G	129	377	64	131	45	34	122	0.72	174.0
H	154	708	114	111	35	32	133	0.46	212.7
I	171	459	67	142	46	32	144	0.44	183.0
J	157	507	85	163	53	33	169	0.55	198.0
K	166	610	43	163	52	32	200	0.41	209.7
L	162	600	84	153	46	30	157	0.88	207.3
N	165	525	99	164	54	33	175	0.70	210.7
Q	185	494	72	173	57	33	150	0.56	227.7
R	158	462	54	137	47	34	170	0.50	147.7
S	151	469	66	139	47	34	158	0.89	208.3
T	183	558	164	151	54	36	145	0.46	208.3
U	170	512	95	149	57	38	163	0.65	203.7
W	157	420	71	124	41	33	145	0.72	197.7
X	130	443	61	137	52	38	142	0.34	198.3
Y	164	438	61	134	47	35	144	0.47	140.0
Mean		508	79	145.	48.8	34	153	0.61	191.6
Standard Deviation		± 83	± 30	±17	±6.0		±22	± .22	± 22.5

indicate that serum cholesterol and phospholipid concentrations of males remain relatively constant until the age of about 22 years, at which time both lipid fractions begin to increase.

Of all the lipid fractions, the triglycerides and NEFA showed the greatest intersubject variation. Coefficients of variability[1] for these fractions on the two sampling dates ranged from 32 to 39. For other plasma lipids, the coefficients of variability ranged from 11 to 16. This observation is in accord with the statement of Calvy et al. (1964) that triglycerides were the most variable of the lipid fractions. Calculation of the coefficients of variability from data on the 16 to 35 year old males of Svanborg and Svennerholm (1961) lends further support to the conclusion that total lipids, cholesterol, and phospholipids vary with age. The triglycerides and NEFA of the Svanborg and Svennerholm men showed a variation which was similar to that observed in the 18 to 19 year-old subjects of this study. On the other hand, for the 16 to 35 year-old men, the coefficients of variability for phospholipids, total lipids, and cholesterol ranged from 17 to 30. Presumably, the larger variation was a function of the greater age range of the subjects.

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[1] $CV = \frac{100 \times \text{standard deviation}}{\text{mean}}$

Most of the individual concentrations of lipids were within two standard deviations of the means. High concentrations of NEFA were found in several plasma samples; these may have resulted from lipolysis during storage (Davis, 1947). Subject G had low concentrations of total lipid and cholesterol in the pre-exercise sample. This would suggest an incomplete extraction of plasma lipids, although the phospholipids, determined on the same extract, were above the mean. Furthermore, the triglycerides of Subject G, determined on a separate extract, were low. Subject H showed a high total lipid and triglyceride level in second plasma sample, although other lipids were not excessive. It was suspected that this subject may have eaten prior to having his blood drawn because there was a decrease in NEFA, concurrent with the increase in total lipids and triglycerides.

The clotting times of plasmas before and after the exercise program are presented in Tables 2 and 3. The clotting times are greater than those usually observed with this method (Davidsohn and Wells, 1962). Intersubject variation was small; coefficients of variability for the two sampling periods were 11 and 12.

Analysis of the dietary intakes taken before the beginning of the exercise regimen showed a mean caloric consumption of 2900 Calories. At the end of the program, the average intake was 3100 Calories. Protein intake was approximately 15% and 14% of total

calories; carbohydrate was 45% and 46%. Fat intake remained constant at about 40% for both analyses. Much of the fat calories was derived from animal sources such as meat, milk, and butter. According to the criteria on which the Recommended Dietary Allowances (National Research Council, 1968) are based, the intakes of the individual subjects were, on the whole, adequate to meet their needs. Weight loss or gain of the subjects could not be related to any changes in the parameters or to the dietary intakes. No consistent relationship could be found between the dietary intakes and any of the lipid parameters. The calculated dietary intakes before and at the end of the exercise program appear in Tables 1 and 2 of the Appendix.

The changes in plasma lipids and in clotting times, resulting from the fitness program, appear in Table 4. Statistical analysis of the differences in total lipid values showed a significant increase after exercise ($P \leq 0.05$). These results do not agree with the findings reported by other investigators (Fitzgerald, Heffernan and McFarlane, 1965; Hoffman, Nelson and Gross, 1967; Zauner and Swenson, 1967).

The mean triglyceride concentrations changed only slightly after the exercise program. Statistical analysis revealed no significant difference upon completion of exercise. These results are in accordance with those reported by Carlson and Mossfeldt (1961)

Table 4. Changes in plasma lipids resulting from exercise program.

Subjects	Weight	Total Lipid	Triglyceride	Cholesterol			Phospholipid	NEFA	Clotting Time
				Total	Free	Free/Total			
	lb	mg/100 ml	mg/100 ml	mg/100 ml		%	mg/100 ml	mEq/l	sec
B	+ 7	+254	- 3	+ 10	- 5	-6	+30	+0.20	+15.4
C	+ 1	+ 28	+46	+ 4	0	-1	-29	+0.96	+ 9.7
D	0	+ 35	+39	- 27	-19	-7	-31	-0.06	-34.0
E	+10	- 15	-32	+ 12	0	-3	+23	-0.06	+15.0
G	-12	+ 41	+23	+ 32	+ 9	-2	-59	-0.29	- 6.3
H	-13	+297	+52	- 13	- 7	-2	-27	-0.72	+59.0
I	- 4	+ 17	+ 1	- 7	- 9	-5	- 6	-0.13	+ 3.3
J	0	- 9	-34	+ 21	+ 1	-4	-10	-0.24	- 5.0
K	- 2	+155	- 7	+ 38	+ 5	-6	+36	-0.59	+20.4
L	- 2	+ 93	+11	+ 6	- 3	-3	-22	-0.20	+39.6
N	- 4	- 5	-17	+ 17	+ 2	-2	- 2	+0.35	+73.4
Q	- 3	- 12	- 6	+ 10	- 3	-4	-39	-0.09	+14.7
R	+ 3	- 39	- 6	- 19	- 4	+1	-12	+0.04	- 1.6
S	+ 2	+ 37	- 9	+ 18	+ 7	+1	-10	+0.11	+59.0
T	- 1	- 16	+50	- 1	+ 8	+6	-49	-0.51	+48.0
U	- 3	- 3	- 7	+ 22	+15	+5	-16	-0.11	+33.0
W	+ 2	+ 39	+24	+ 17	+ 4	-2	-27	-0.04	+43.0
X	- 7	- 14	-15	- 3	- 4	-2	+23	-0.03	+11.0
Y	+ 2	- 59	+ 8	- 17	- 8	-1	+23	-0.68	-32.3
Mean	- 1.3	+ 43	+ 6.2	+ 6.3	- 0.58		-12.7	-0.11	+18.9
Standard Deviation	± 5.5	± 95	±26.2	± 17.5	± 7.8		±26.3	±0.39	±29.8

and Holloszy et al. (1964). Calvy et al. (1964) found that serum triglycerides of young Marines doubled after an 11-week training program.

Total and free cholesterol did not differ significantly after the exercise class. Johnson and Wong (1961) and Calvy et al. (1963, 1964) also found that exercise had little effect on the cholesterol values of young men. On the other hand, Campbell (1965) noted that serum cholesterols of young men declined after a 10-week program of phasic activity similar to the jogging regimen of the present study.

A significant decrease in phospholipid concentration occurred ($P \leq 0.05$). Other investigators have reported little or no relationship between exercise and phospholipids (Johnson and Wong, 1961; Calvy et al., 1963; Holloszy et al., 1964; Shane, 1966).

Although the mean NEFA levels decreased from the first analysis to the second analysis, statistical significance could not be attributed to the change. NEFA are affected while exercise is in progress and shortly after an exercise is terminated (Basu, Passmore and Strong, 1960; Carlson and Pernow, 1961; Friedberg et al., 1963). Since this study investigated the long term effects of exercise, it was not expected that NEFA would be affected by this program.

An increase in the mean clotting time was noted after completion of the fitness program. This change was significant even at the critical value of $t_{.01}$ ($P \leq 0.01$). This finding is not in agreement with the results of Keeney (1959) or Burt, Blyth and Rierson (1964) who worked with the whole blood clotting times. Keys and Buzina (1956), however, found that the blood of physically active men clotted less rapidly than did that of physically inactive men. In light of the evidence that blood coagulability is increased in cases of coronary insufficiency (Panchenko and Bazaz'yan, 1965), the results of the present study would suggest that exercise could be a preventive measure against coronary thrombosis.

Since the phospholipids are intimately concerned with the coagulation process (Merskey and Marcus, 1963; Marcus, 1966), a correlation coefficient was determined on the differences for the two parameters; no relation could be found. A relationship between clotting time and other lipid fractions was not apparent.

SUMMARY

Nineteen apparently healthy late adolescent males participated in an 11-week fitness program which consisted primarily of jogging. Dietary intakes were calculated prior to and after the fitness regimen. Venous blood was drawn from fasting subjects before the exercise program and again at the end of the program. Plasma concentrations of total lipids, triglycerides, total and free cholesterol, phospholipids, and non-esterified fatty acids were determined. Clotting times were determined soon after the plasmas were separated. The change in each parameter, resulting from the exercise program, was subjected to statistical analysis.

The mean concentrations of total lipids, total and free cholesterol, and phospholipids in the plasmas of these 18 to 19 year old boys were lower than the values reported for young men. Plasma clotting times were greater. The mean plasma triglycerides and non-esterified fatty acids, on the other hand, were similar to those found in adults. Individual measurements of all lipid fractions were essentially within the ranges observed in healthy males. The greatest intersubject variation was observed in triglycerides and non-esterified fatty acids.

Exercise had no significant effect on triglycerides, total and free cholesterol, or the non-esterified fatty acids. Phospholipids

decreased significantly after exercise; total lipids and clotting times increased significantly. Comparison of the phospholipids to clotting time revealed no significant correlation. Consistent relationships could not be found between the dietary intakes, weight change, or any of the lipid parameters.

It is concluded that the plasma of late adolescent males is relatively low in lipids, and that it coagulates more slowly than does that of the adult male. The major effect of the physical fitness program was a prolongation of the coagulation time.

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APPENDIX

Appendix Table 1. Dietary intakes before exercise program.

Subject	Food Energy	Protein	Fat	Total Carbohydrate	Calcium	Iron	Vitamin A	Thiamin	Riboflavin	Niacin	Ascorbic Acid
	Calorie	gm	gm	gm	mg	mg	IU	mg	mg	mg	mg
B	2427	82	113	282	953	15.3	3728	1.1	2.1	16.0	64
C	2587	128	128	245	1675	14.7	1113	1.4	3.0	22.1	171
D	2824	121	103	360	2117	15.0	8921	2.3	3.8	29.5	99
E	3285	127	157	337	882	19.7	3691	1.9	2.0	23.8	36
G	2946	91	104	343	1255	10.9	6326	1.2	2.4	15.0	39
H	2491	84	116	284	1326	8.2	8376	1.2	2.3	9.1	43
I	3774	124	168	455	1296	18.1	7703	2.3	2.5	19.4	80
J	1653	65	60	216	1139	8.5	4898	1.0	1.9	13.4	21
K	3112	108	137	328	1336	14.8	8202	2.3	2.5	18.6	192
L	3135	102	158	334	1238	13.9	7442	2.2	2.2	16.0	128
N	1849	58	73	264	871	8.7	5107	1.1	1.5	10.1	132
Q	2856	117	139	276	1763	16.8	5278	1.6	3.2	14.5	63
R	3913	133	188	429	2277	15.3	8606	2.5	3.5	14.9	146
S	3177	126	157	327	2098	12.8	7495	1.6	3.7	12.7	48
T	3201	133	141	373	1693	13.7	10203	1.4	3.1	23.3	39
U	4083	153	202	416	1612	20.5	12666	1.6	3.3	25.3	115
W	3640	121	147	470	1455	17.6	10116	1.6	2.5	18.2	121
X	2809	88	117	360	1408	12.5	4962	1.0	2.5	12.4	21
Y	2027	68	86	243	482	7.5	4228	0.8	0.9	14.3	79
Mean	2936	107	131	334	1415	13.9	6793	1.6	2.6	17.3	86

Appendix Table 2. Dietary intake at end of exercise program.

Subject	Food Energy	Protein	Fat	Total Carbohydrate	Calcium	Iron	Vitamin A	Thiamin	Riboflavin	Niacin	Ascorbic Acid
	Calorie	gm	gm	gm	mg	mg	IU	mg	mg	mg	mg
B	2917	119	143	280	1248	15.2	4296	1.8	2.7	28.1	77
C	2297	81	92	275	468	11.1	2843	1.1	1.2	19.9	28
D	2698	130	110	304	1362	19.1	8595	2.3	2.5	13.3	84
E	4220	116	198	514	1532	15.9	7903	1.8	2.6	18.8	111
G	3539	138	175	357	2045	16.6	8197	1.9	3.6	21.5	154
H	2740	111	134	279	1446	12.3	4806	2.3	2.5	10.9	64
I	2658	98	107	332	320	13.8	4250	1.9	1.1	14.7	57
J	1662	69	62	208	1010	8.6	6636	0.9	1.4	10.3	82
K	3966	149	178	410	1463	19.3	6159	3.0	3.1	23.8	137
L	2747	77	114	361	1001	12.7	4845	1.2	1.7	11.7	125
N	3055	117	126	367	1373	15.9	4966	1.7	2.8	19.0	97
Q	2523	100	113	285	1399	14.1	4878	1.3	2.3	12.1	86
R	3023	117	114	391	2814	11.5	4130	1.7	3.9	17.6	120
S	3777	151	172	414	2156	20.3	7954	2.4	4.0	17.4	50
T	3254	91	132	407	1543	9.5	3145	1.2	2.6	15.3	34
U	3407	95	153	416	1134	15.1	6884	1.5	2.2	15.8	74
W	4519	159	240	435	1737	24.1	12970	2.9	3.3	24.7	140
X	3577	92	155	453	1423	13.7	5170	1.4	2.8	11.3	20
Y	2519	69	116	275	526	10.9	2234	1.7	1.4	23.5	26
Mean	3110	109	139	356	1368	14.7	5824	1.8	2.5	17.4	82