LIPOPROTEIN AND TOTAL PLASMA CHOLESTEROL: INFLUENCE OF AGE, RACE, DIET AND EXERCISE IN 150 FEMALE ADOLESCENTS

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PREFACE

This study examines relationships of diet and exercise practices with plasma cholesterol in a racially mixed population of 150 adolescent females in North Central Oklahoma. The primary objective is to see if plasma cholesterol changes between 12 and 16 years of age and to explore possible associations between plasma cholesterol, age, race, diet and exercise.

Many people have generously contributed to this research endeavor. First of all, the author extends heartfelt thanks to her major adviser, Dr. Mary Alice Kenney, who unselfishly donated her time and provided invaluable assistance throughout this study. Also, appreciation is expressed to other committee members, Dr. Donald Abbott, Dr. Lea Ebro and Dr. Esther Winterfeldt, for their advice and helpful comments in the preparation of this manuscript.

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CHAPTER I

INTRODUCTION

In recent years, researchers have related lipoprotein fractions to coronary heart disease (CHD) and, from the information amassed, have drawn some widely accepted conclusions. Among the least expected findings was an inverse relationship between coronary heart disease and high-density lipoprotein cholesterol (HDLC) (Miller and Miller, 1975; Gordon et al., 1977; Castelli et al., 1977). This discovery spurred a number of studies, but most are limited to older populations in cultures with high incidence of CHD. Since the disease may commence in childhood and progress into adulthood, the need for studies on young populations to explore the effect of known risk factors on total and lipoprotein cholesterol is indicated.

Background and Value of the Study

The function of HDL in cholesterol metabolism has not been clearly elucidated, although evidence points to a role for HDL in the removal of cholesterol from various tissues and its transport to the liver for catabolism (Tall and Small, 1978; Havel, 1979). Hence, HDL may aid in the breakdown of excess cholesterol, while the other fractions,

namely low-density lipoprotein (LDL) and very-low-density lipoprotein (VLDL), are involved in the transport of cholesterol to extrahepatic tissues. These findings demonstrate the need to examine lipoprotein fractions separately. In one extensive investigation, the Bogalusa Heart study (Frerichs et al., 1976), lipids were assayed in 3446 children in Louisiana. Cholesterol was estimated based on measurement of LDL and VLDL fractions. HDLC, however, was determined by subtraction from the total serum cholesterol (Srinivasan et al., 1976). In this adolescent sample, a slight decrease in total cholesterol was noted in females, usually occurring between their eleventh and fourteenth years, but was surmised to be dependent on level of sexual maturation rather than on age. The results of the Bogalusa study suggested this decrease to be due largely to a reduction in the LDL fraction, with little change in the HDL. If so, constancy over age makes this one of only a few measurements valid for girls at various stages of maturation. Unfortunately, most studies employ the method used by the Bogalusa investigators for HDLC determination, which is calculated by difference from serum cholesterol. Therefore, a quantitative study on the HDL fraction, specifically, is needed to support this finding.

Diet can influence plasma cholesterol. Not only specific nutrient intakes, but eating patterns may play a role. For instance, eating span, defined as the time interval between the first thing eaten in the morning and

the last thing consumed at night, and eating frequency, which is the number of times a person consumes food or beverage in a 24-hour period (Frank et al., 1978), were evaluated for a fifth grade subsample of the Bogalusa study. Results established a significantly higher total serum cholesterol for those children with an eating span longer than thirteen hours. Furthermore, the group eating the least frequently (fewer than or equal to five times) had significantly higher HDL levels than groups eating more often than five times a day (Frank et al., 1978).

Other potential factors cannot be overlooked. First of all, studies have determined that physical activity decreases total cholesterol levels, but increases HDLC (Berger, 1978; Wood and Haskell, 1979). However, with the exception of one study by Farrell (1980), who showed that HDL increased with a concomitant decrease in plasma triglycerides after four weeks of endurance training, reports of increased HDLC involved men only. Recent evidence (Frey et al., 1982) suggests that women may not exhibit increased HDLC levels with exercise, possibly due to difference in hormonal release with physical activity.

Purpose and Objectives of the Study

The purpose of this study of a racially mixed population of approximately 150 adolescents throughout North Central Oklahoma is to determine if total cholesterol and HDLC (both measured directly) or other plasma cholesterol

(OPC)(calculated by difference) change between twelve and sixteen years of age. In addition, the influence of different dietary practices and exercise on cholesterol concentrations will be investigated.

To establish dietary influences on HDLC levels the relationship of this lipoprotein fraction and of total plasma cholesterol to eating span and eating frequency will be carefully examined. Also, intakes of specific dietary components, such as cholesterol, protein, fat, carbohydrate and sodium will be related, if possible, to total plasma cholesterol and HDLC concentrations. Energy sources of the diet will be of particular interest because of contradictory reports that saturated fat and cholesterol intakes are or are not associated with elevated plasma cholesterol and that high-sucrose diets effect higher plasma cholesterol levels than diets high in other sources of carbohydrates.

Additional variables will be evaluated. In light of the conflicting evidence regarding the influence of exercise on total cholesterol and HDLC levels, the effect of activity on teenage girls in the midst of pubertal changes is an intriguing consideration. Also, oral contraceptives have been associated with increased HDLC in adolescent females. Thus, information about these practices will be obtained.

CHAPTER II

REVIEW OF THE LITERATURE

Atherosclerosis and Coronary Heart Disease

Atherosclerosis often leads to coronary heart disease (CHD) (Benditt and Gown, 1980). This disease is progressive, beginning as fatty streaks that initially develop at forks of major arteries, perhaps in childhood (Sabine, 1977). Later, fibrous plaques evolve as collagen and elastic tissue build up on the endothelium. Consequently, the intima does not get needed nutrients. The cells die, elasticity is lost and an accumulation of additional lipid, fiber and minerals occurs, gradually becoming what is termed a complicated lesion (Gotto et al., 1977). The event or events that initiate this process are unclear. One current hypothesis, supported by Ross and Harker (1976), is that endothelial injury of a mechanical, chemical or immunological nature may be the underlying cause of atherosclerosis. But, additional evidence is needed to substantiate this finding.

The contribution of serum cholesterol as a risk factor in the development and extent of these lesions and eventual CHD has been established in studies spanning at least three

decades. Keys et al. (1963) conducted a study on 281 middle-aged men in Minnesota. The subjects were followed for fifteen years. Thirty-two developed CHD. Serum cholesterol was significantly associated with risk of developing the disease. Kannel et al. (1971) of the Framingham study determined risk of CHD in 2282 men and 2845 women to be proportional to serum cholesterol. Moreover, a role for dietary lipids in the elevation of serum cholesterol levels in man has been extensively investigated. Based on dietary experiments, Keys et al. (1959) proposed a formula by which they ascertained one could predict serum cholesterol alterations caused by dietary changes of monounsaturated, polyunsaturated and saturated fatty acids. In 1961, Wilcox and Galloway published their findings that serum cholesterol levels in university students (four men and four women) were highest while consuming butter and lowest while ingesting corn oil. And most recently, in 1981, Shekelle et al., of the Western Electric study, presented the results of a 20-year longitudinal study of men, 40 to 55 years of age at their initial visit. Serum cholesterol levels were found to depend on fat intake.

Although the precise interplay between dietary lipids, serum cholesterol and other dietary factors in the development of CHD remains clouded, the likelihood that CHD can be slowed if dietary intervention begins early enough cannot be ignored. It must be mentioned that some studies

have failed to find a dietary relationship to serum lipids. Gordon and Kannel (1968) of the Framingham study found no association between dietary cholesterol and serum cholesterol levels. Neither was a link between exogenous cholesterol and serum levels found by Tecumseh study researchers (Nichols et al., 1976).

HDL: Definition, Structure and Function

Definition

Tall and Small (1978, p. 1232) define high-density lipoproteins as "very small aggregates of lipids and proteins that circulate in the lymph and blood plasma." By weight they are approximately 50% protein. The remaining 50% is comprised of lipids, specifically phospholipids, cholesterol, cholesterol esters and triglycerides (Jackson et al., 1976). Of the lipoprotein classes, HDL has the most protein and phospholipids and the least triglyceride (Sabine, 1977).

Structure

HDL exists as a bilayered structure, similar to a micelle (Tall and Small, 1978). The protein, being hydrophilic, serves to solubilize the lipids. Cholesterol and lecithin have polar and nonpolar ends. They constitute the bilayer; the polar ends face the water, while the nonpolar ends line up parallel to one another compactly on the inside of the complex. Cholesterol esters, which are hydrophobic, seek a nonpolar environment; thus, they

migrate inward. The bilayer surrounds the esters, forming a sphere. As a sphere, the complex measures only ten nanometers (Tall and Small, 1978).

Function

Among the lipoprotein classes, only the HDL are considered nonatherogenic (Havel, 1979). Up until the last decade, this class was overlooked, even though Barr et al. (1951) reported that HDL values of healthy men were higher than those with CHD. Instead, interest has focused primarily on the low-density lipoprotein and very-low-density lipoprotein fractions, which are positively correlated with CHD (Lewis, 1974). In the past several years, studies have provided substantial evidence that the HDL fraction exerts a protective effect against CHD (Miller and Miller, 1975; Castelli et al., 1977; Goldbourt and Medalie, 1979).

The mechanism by which this effect is realized is still a mystery. Currently, the most promising model for the role of HDL involves cholesterol removal from peripheral tissues and transport of the cholesterol to the liver for catabolism and subsequent excretion (Tall and Small, 1978; Havel, 1979). The source of HDL may be chylomicrons and VLDL. Upon their breakdown in peripheral tissues, liver and intestine, HDL molecules may be re-synthesized from their end products (Havel, 1979).

Adolescent Studies

General Description

A number of bold undertakings on large adolescent populations have of late provided information about serum lipids in the previously neglected younger segment of American society. Despite the influx of data, results have provided less than definitive conclusions. As a matter of fact, reports on total cholesterol and the HDL and LDL fractions of the teenager vary considerably with respect to age, sex and race. This fact alone points up the need to continue this line of research, while concomitantly striving for better biochemical methods for assaying blood lipids, so that more accurate and precise data can be collected.

The most comprehensive lipid studies to date involving adolescents include the Bogalusa Heart study (Frerichs et al., 1976; Srinivasan et al., 1976; Frank et al., 1978), the Evans County study (Hames and Greenberg, 1961), the Princeton study (Morrison et al., 1977, 1978, 1979, 1980a, 1980b), the Muscatine study (Lauer et al., 1975) and the Child Research Council study (Lee, 1967). Even among these research endeavors, accumulated data are insufficient and inconclusive, especially those relating to lipoprotein fractions. For example, the Muscatine and Child Research Council studies report total serum cholesterol values only; cholesterol in lipoprotein fractions was not

measured. In the Princeton study, lipoproteins were determined in only 30% of those appearing for a second visit and included exclusively those participants with high cholesterol and triglycerides from the previous visit plus a 15% subsample selected randomly from the original population (Morrison et al., 1978). Conducted in 1960, the Evans study employed now outdated methods for quantitating lipoproteins. Hames and Greenberg reported beta ratios, which are determined colorimetrically and reflect the quotient of total dye uptake and the dye content of each fraction after separation by paper electrophoresis. Comparing these values with cholesterol values recorded for the other studies is difficult.

Of the research cited above, only the Bogalusa Heart study and Princeton study conducted lipoprotein analyses. Although the total numbers involved in the Princeton study (6775) were about double those of the Bogalusa study (3446), only 15% of the Princeton population was evaluated for lipoproteins. The Bogalusa study, on the other hand, restricted the age of their participants to fourteen, so no information on the late-adolescent teenager is available from this study. In addition, HDLC was determined indirectly by difference, subtracting beta plus pre-beta lipoprotein cholesterol from total cholesterol, a less than ideal method for HDLC quantitation.

A summary of these studies is presented in Table I. For each of the five studies, statistics regarding female

TABLE I

SUMMARY OF ADOLESCENT STUDIES

Study	Study Statistics			Total Cholesterol mg/dl (SD)				Lipoprotein Cholesterol mg/dl (SD)								
	Age Span (yr)	%		Age (yr)	n	Females		n	%		HDLC[a]		LDLC[b]		VLDLC[c]	
		White	Black			White	Black		White	Black	White	Black	White	Black	White	Black
Bogalusa (1975)	5-14															
		63	37	12	206	162.9 (28.1)	163.7 (24.0)	196	62	38	63.7 (20)	72.9 (18)	85.8 (19)	84.0 (18)	11.8 (8.4)	7.10 (4.9)
		59	41	14	152	157.7 (26.8)	166.6 (28.5)	142	58	42	60.7 (19)	68.8 (17)	85.4 (21)	89.1 (23)	12.4 (7.3)	8.88 (6.0)
		63	37	5-14	635	163.5 (27.1)	170.9 (29.6)	1540	63	37	63.7 (21)	72.5 (22)	90.0 (22)	90.5 (23)	9.55 (8.0)	7.55 (5.8)
Princeton (1978)	6-17															
		73	27	12	452	160 (24.5)	162 (26.0)	88[d]	64	36	52 (10)	56 (11)	98 (20)	102 (24)		
		74	26	14	298	157 (23.7)	161 (29.3)	77[d]	73	27	50 (10)	56 (11)	91 (19)	102 (19)		
		57	43	16	169	155 (26.6)	164 (29.9)	77[d]	84	16	55 (12)	54 (10)	93 (22)	89 (30)		
		74	26	12-17	1555	157 (24.3)	165 (27.9)	242	73	27	52 (11)	56 (12)	94 (21)	100 (24)		
Muscatine[e] (1975)	6-18	96.4	0.6		2483											
Child Research Council (1967)	4-18	f	f	4-18	66											
Evans County (1961)	6-20	67	g		648											
		60	g	12	36	177.1										
		69	g	14	58	189.4										
		53	g	16	57	193.6										

[a]For Bogalusa Study, calculated: α-LP ÷ 5.9.

[b]For Bogalusa Study, calculated: β-LP x 0.469.

[c]For Bogalusa Study, calculated: pre-β-LP x 0.222.

[d]Includes 12 and 13 year-olds.

[e]No breakdown by race or sex was provided in literature. Total number of participants = 4829. Mean total cholesterol for all = 182 mg/dl (SD±29).

[f]Race not reported. Mean total cholesterol for 66 females = 166.1 mg/dl.

[g]Reported as white or non-white only.

teenagers between twelve and sixteen is presented. Mean total cholesterol values, along with HDLC, LDLC and VLDLC are tabulated, as are the number of subjects in each category.

Findings

Age Differences

Total Cholesterol. Reports of total cholesterol differences with age are contradictory. Lauer et al. of the Muscatine study (1975) found no total cholesterol changes in girls six to eighteen years old; neither did Bogalusa investigators find significant changes for the age span five to fourteen, although a tendency for a decrease in total cholesterol after age ten was noted. The Evans County contributors (Hames and Greenberg, 1961) noted a tendency for total cholesterol to increase from age six to twenty, but not significantly. To the contrary, Princeton researchers found significantly lower total cholesterol levels for ages twelve through seventeen among their 6775 participants than for the six through eleven group (Morrison et al., 1977). They attribute these lower values to a reduction in total cholesterol at around puberty, followed by an increase at about age seventeen. For the 15% random sample described previously, on which lipoprotein studies were conducted, this pattern was less evident for white females (P=.1) and absent in black females. Finally, in the longitudinal Child Research Council study (Lee, 1967), sixteen girls participated. Twelve of them exhibited an average 9% decrease during adolescence, which was defined as

the time between the beginning of their adolescent growth spurt and the onset of menstruation. This particular study has one great advantage over the others discussed; namely, the subjects were followed over a ten-year time span. Thus, adolescence was determined and evaluated on an individual basis, rather than depending solely on age and the homogeneity of the group.

HDLC. Neither the Princeton nor Bogalusa study found a change in HDLC values with age.

LDLC and VLDLC. Srinivasan et al. (1976) noted a definite decrease in LDLC levels between ages eleven and fourteen for females of both racial groups. Morrison et al. (1978) determined that LDLC decreased during adolescence in white females, but this did not hold true for black females, where no differences in LDLC were discovered. Those investigators ascribed the decrease in total cholesterol in white females to the LDL fraction. Of particular interest from the Bogalusa study is that the VLDLC increased significantly with age ($P < .0001$). This is the only study in which this fraction was measured.

Racial Differences

Total Cholesterol. Frerichs et al. (1976) found that blacks had higher serum cholesterol levels than whites based on the mean cholesterol value for female participants at all ages. However, the difference was not significant within

most age-groups. In fact, between ten and fourteen years, only girls fourteen showed a significant difference ($P < .05$). On the other hand, black females ages six through seventeen had higher total cholesterol than whites in all age-groups in the Princeton study (Morrison et al., 1977). The Evans study reports no racial differences in the adolescent age-groups. Since the Child Research Council did not consider race, no data are available from which to delineate racial distinctions.

HDLC. Bogalusa investigators (Srinivasan et al., 1976) noted that HDLC values of blacks tended to be higher than those of whites, but only for some age-groups, including the twelve through fourteen year-olds, where the difference proved significant ($P < .05$). Similarly, Princeton researchers (Morrison et al., 1978) found that white females, twelve to seventeen, had lower HDLC values than blacks ($P < .05$).

LDLC and VLDLC. No adolescent studies established racial variation in any of their age-groups for LDLC. However, the Bogalusa study, which is the sole adolescent research endeavor that pursued the measurement and analysis of VLDLC, reports a marked increase in VLDLC levels in white females during adolescence (Srinivasan et al., 1976).

Eating Span and Frequency

Investigating the role of eating patterns in the regulation of metabolic pathways is extremely complex. Over the years, Leveille in particular has addressed this topic through intensive study of laboratory animals. In a series of experiments (Leveille, 1967, 1969; Leveille and O'Hea, 1967; Chakrabarty and Leveille, 1968), he restricted rats to one meal a day rather than allowing them to nibble in their normal manner. The results indicated that rats consequently synthesized more lipids. Leveille hypothesized that the nibbler uses more carbohydrate than fat as a source of energy because this foodstuff is always readily available. Conversely, meal eaters store their food as fat upon digestion and use the fat for energy as needed. The idea was not a new one. Cohn and Joseph (1960) also contributed to this endeavor in the late fifties and early sixties, and concluded that meal eating by rats does stimulate lipogenesis. Hence, it is clear that feeding patterns can influence lipid levels in the blood. Whether or not this holds true for humans and what bearing this has on future risk of CHD are unknown and warrant further study.

In only one of the adolescent studies discussed, the Bogalusa study, were eating patterns investigated. Frank et al. (1978) randomly selected one-half of the ten year-olds as a subsample of their population to examine the effect of diet on cholesterol levels as well as its

relationship to other CHD risk factors. Eating span, the time interval between the first thing eaten in the morning and the last thing consumed at night, and eating frequency, the number of times a person consumes food or beverage in a 24-hour period, were among the parameters selected. The children were divided into three groups in each case. Group A had an eating span less than ten hours; Group B, ten to thirteen hours; and Group C, more than thirteen hours. Eating less than five times a day defined Group 1; six to eight times a day, Group 2; nine to twelve times a day, Group 3. Regarding eating span, Group C consumed more carbohydrate, protein, fat, energy and sodium than the other two groups. Moreover, Group C had significantly higher serum cholesterol than either Group A or B ($P < .05$). No other differences in lipids were found. Those with the greatest eating frequency (Group 3) had higher protein, carbohydrate, fat, sodium and energy intakes than the other frequency groups. In addition, Group 3 also consumed more sucrose, starch, saturated and polyunsaturated fatty acids, iron, calcium and cholesterol than Groups 1 and 2. In this case, Group 2 had the greatest VLDLC values. Group 1 had the highest HDLC. No differences were found, however, in total cholesterol levels.

The Bogalusa investigators reported lipoprotein profiles for a total of 3182 children, five through fourteen. Yet, in only one-half of the ten year-olds were eating patterns evaluated. Thus, all together, 185 fifth graders

were involved. The only sex-race difference reported for eating span or frequency was that black boys ate fewer meals compared to the other sex-race groups. It is clear from these facts that additional information would be useful in establishing possible associations between eating patterns and risk factors for CHD.

Dietary Factors

Cholesterol

The diet health controversy surrounding cholesterol and fats is based on inconclusive evidence linking diet to hypercholesterolemia, atherosclerosis and CHD via epidemiological and animal studies (Keys, 1967; Hodges, 1968; 1968; Garcia-Palmieri et al., 1980; Shekelle et al., 1981; Gordon et al., 1981), which is valuable but subject to error in interpretation and extrapolation.

The current controversy is an outgrowth of preceding stands on the issue. In 1961, the American Heart Association (AHA) recommended a decrease in saturated fatty acid intake and an increase in polyunsaturated vegetable oil consumption. This was followed in 1968 by eight dietary guidelines released by the AHA (AHA, 1968), which included the recommendation to reduce cholesterol intake. The American Medical Association and Food and Nutrition Board, in 1972, voiced their support for diet modification as a means to lessen the risk of CHD. The Dietary Goals of the Select Committee on Nutrition and Human Needs of the

U.S. Senate, 1977, placed actual limits on fat and cholesterol intake. Partly in response to criticism for having recommended specific figures, the government tempered its position in 1980 and issued a report, "Nutrition and Your Health---Dietary Guidelines for Americans." The same year, the Food and Nutrition Board released "Toward Healthful Diets," in which, to the chagrin of many, no specific recommendations were made regarding cholesterol intake for the healthy person. This decision was based on what the Board considered inconclusive evidence of the diet/coronary heart disease link and lack of information as to other effects of dietary changes which would produce recommended cholesterol intakes.

The controversy has been amplified by many outside the scientific community. For example, politicians argue that if accepted as public policy, the hypothesis will eventually lead to a comprehensive national health care plan (Richmond, 1980).

The controversy continues. In January, 1981, the results of the Western Electric study appeared in the New England Journal of Medicine (Shekelle et al., 1981). Subjects were evaluated in 1957, 1958, and again twenty years later. The findings support the diet/heart hypothesis that diet affects cholesterol levels and, consequently, the risk of coronary heart disease.

Of the adolescent studies outlined herein, only the Bogalusa study (Frank et al., 1978) and Princeton study

(Morrison et al., 1980) collected and evaluated dietary data. Few simple correlations with serum cholesterol or lipoprotein fractions ensued. Bogalusa investigators reported a weak positive correlation ($P < .05$) between LDLC and dietary cholesterol, while Princeton researchers noted that dietary cholesterol was positively correlated with HDLC.

Protein

The effect of dietary protein on serum cholesterol has been tested in humans (Olson et al., 1958; Walker et al., 1960; Hodges et al., 1967). In these instances, it has been demonstrated that changing the source of protein in the diet from animal to vegetable can effect a decrease in serum cholesterol independent of levels of carbohydrate or fat. Sirtori et al. (1977) substantiated this proposition. They conducted an experiment on 22 patients with type-II hyperlipoproteinemia admitted to a hospital following a three-month trial period at home during which time they consumed a low-lipid, low-cholesterol diet. In the hospital, they received either (1) a low-lipid diet consisting of 21% (percent of calories) protein (62% animal and 38% vegetable), 21% lipids (46% polyunsaturated, 33% monounsaturated and 21% saturated fats), and 58% carbohydrates, or (2) a soybean textured protein diet, which was 21% protein (63% soybean protein, 30% other vegetable protein and 7% animal protein), 26% lipids (44% polyun-

saturated, 40% monounsaturated, and 16% saturated) and 53% carbohydrates. After three weeks, each patient switched to the alternate diet. Analysis of the data confirmed a strong hypocholesterolemic effect for those on the soy-protein regimen, whereas the standard low-lipid diet, which the patients had been following at home prior to the onset of the study, had no effect. Critics claim that interpretation of these data is difficult, due to changes in fat, fiber and carbohydrate content, which necessarily accompanied the soy-protein diet and are unaccounted for. To complicate matters, another investigator had failed to note a change in serum cholesterol when soybean protein replaced meat protein in the diet (Holmes, 1980). Despite the contradictory evidence, Sirtori et al. (1981) defend their findings and intimate that Holmes did not employ severely hypercholesterolemic individuals, in which case one would not necessarily expect plasma cholesterol to fall in normolipidemic patients after only a few weeks on a modified diet. In addition, Sirtori claims that the soy-protein diet must be completely animal protein-free to induce the effect, which Holmes' diet was not. Of further interest in the Holmes study was the significant contribution of the LDL and VLDL fractions in lowering serum cholesterol when patients were on a low-lipid diet. Additional research is needed to elucidate the effect of protein on serum cholesterol levels, particularly its influence on individual lipoprotein fractions.

The HDL fraction was the focus in one animal study (Srinivasan, 1978). Exogenous cholesterol given to spider monkeys on a very low protein diet (6% of calories) induced a marked increase in HDLC, whereas lowering the protein from 25% to 8% resulted in only a slight reduction. It appeared that the HDL increased markedly at some critical point below 8%.

Fat

Since the mid-1950's, research efforts have time and again supported the hypothesis that substituting unsaturated for saturated fats in the diet can elicit a lowering of serum cholesterol levels (Ahrens et al., 1957; Bronte-Stewart et al., 1956; Turpeinen et al., 1968; Shekelle et al., 1981). Ahrens et al. (1957), found that cholesterol levels dipped to their lowest when the fats were comprised of corn oil, safflower oil or cottonseed oil. The same investigators also submitted evidence that those subjects consuming these same oils, only hydrogenated, had higher total serum cholesterol than when ingesting the unhydrogenated form. Keys et al. (1957, 1959) have gone so far as to propose a formula by which they claim to predict changes in total serum cholesterol, depending on the relative intake of saturated, monoun-saturated and polyunsaturated fatty acids. Although not all investigators have found the formula to be accurate in predicting cholesterol changes (Turpeinin et al., 1968), most support the premise that diet can affect serum cholesterol levels, and in turn, aid in the prevention

of coronary heart disease (Stamler, 1978; Heyden and Williams, 1982). As is the case with dietary cholesterol, the evidence suggesting that saturated fats increase the risk for CHD is based largely on epidemiological studies, and therefore readies the battleground for critics who question the value of these efforts (Werkö, 1976; Mann, 1977). Along these same lines, the P/S ratio (polyunsaturated fatty acids/saturated fatty acids ratio) has gained fairly wide acceptance as an indication of the relative saturation of fats in the diet. Erickson et al. (1964) deny any relationship of the P/S ratio to serum cholesterol levels, although many researchers defend their opposing position on this issue (Keys et al., 1957). The extent to which hydrogenation of fatty acids influences serum cholesterol is still open to debate. In the first place, the problem is compounded by the fact that polyunsaturated fats are usually hydrogenated to a limited degree, thus increasing the saturation of natural fats high in polyunsaturated or monounsaturated fatty acids. In addition, there is a conversion of cis to trans configuration, which may result in fats nutritionally different from the naturally occurring ones (Erickson et al., 1964). It therefore becomes a difficult, if not insurmountable, task to discern which factor is contributing to or causing a given change in cholesterol levels. Erickson et al. (1964) found no change in serum cholesterol following partial hydrogenation of fat. However, others would argue

his findings. For example, Ahrens et al. (1957) found that hydrogenated corn and cottonseed oils consumed by human subjects resulted in higher serum-lipid levels than the unhydrogenated forms of the same fat.

Carbohydrates

Portman et al. (1956) introduced one of the first strong pieces of evidence that carbohydrates may affect serum cholesterol levels. Rats were fed various carbohydrates in combination with unchanging amounts of cholesterol and in some cases, cholic acid, as part of their basal diet. Sucrose, fructose and glucose independently effected higher total serum cholesterol and LDLC concentrations than cornstarch. He surmised that gastrointestinal microorganisms might offer an explanation, because, upon addition of sulfasuxidine to the sucrose diet, no serum cholesterol change occurred; yet, when this agent was added to the starch diet, cholesterol values rose to the level noted in the rats receiving sucrose. Hence, the rate of cholesterol metabolism in the intestines and, subsequently, the extent of reabsorption may well be influenced by bacteria which are, in turn, affected by the presence of carbohydrates.

Three years later, Grant and Fahrenbach (1959) published data confirming the contribution of sucrose in elevating total serum cholesterol, but only when cholesterol was fed. In this case, sucrose appeared to have a greater effect than glucose.

Hodges and Krehl (1965) proposed another feasible mechanism. They, too, found that sucrose resulted in higher cholesterol values than starch. But rather than focusing on the intestinal flora for an explanation, these researchers suspect that sucrose bombards the normal metabolic pathway for carbohydrates. Thus, the body resorts to the hexose monophosphate shunt, thereby enhancing fatty acid synthesis and conceivably that of cholesterol as well.

Especially pertinent here is a study Macdonald conducted in 1965 suggesting that estrogens play a critical role in cholesterol metabolism. More specifically, they may enhance carbohydrate metabolism in young women, which might shed some light on the observation that sucrose failed to elevate lipids of women of child-bearing age.

More recently, Srinivasan et al. (1978) presented the results of an experiment on non-human primates. This study was one of the first to examine the effect of carbohydrates on lipoprotein fractions. Squirrel and spider monkeys on high-sucrose, low-saturated fat diets exhibited increases in serum cholesterol with or without dietary intake of cholesterol. Likewise, LDLC increased in both species. HDLC showed a marked increase in squirrel monkeys, but was less notable in spider monkeys. No VLDLC differences were reported for either species.

The apparent inter-species variations in cholesterol metabolism make extrapolation to man impossible, yet human studies in this area are few. Reiser et al. (1979) studied

a group of ten men and nine women in a cross-over experiment. Thirty percent of the total calories was given as either sucrose or wheat starch. These researchers found that VLDLC values were 32% higher for sucrose than starch. No significant changes were identified for the other lipoprotein fractions. Serum cholesterol was significantly higher for those on the sucrose diet. The investigators further intimate that men may be more sensitive to sucrose as a serum lipid-elevating factor than women, based on the fact that VLDLC were significantly higher for men than women when on the sucrose diet.

On a larger scale, Garcia-Palmieri et al. (1980) reported findings for 8218 Puerto Rican men. No association between CHD and sucrose intake was uncovered. However, an inverse relationship was reported for CHD and carbohydrates consumed in the form of legumes.

Results from the adolescent studies were surprising. Morrison et al. (1980) maintain that plasma cholesterol and sugar intake are negatively correlated. Similarly, the Bogalusa researchers (1978) found that children above the 25th percentile for serum cholesterol had lower carbohydrate and sucrose intakes than those below the 25th percentile. For lipoproteins, the Princeton group (1980) established that high carbohydrate and sucrose intakes were associated with decreased HDLC and LDLC values. Frank et al. (1978) noted marginally significant inverse associations for sucrose and HDLC, but a positive

association for starch and HDLC. No relationship, however, was found for total carbohydrates and HDLC. In contrast to the above, Weidman et al. (1978) reported no associations between dietary variables and serum cholesterol.

Exercise

Ever since exercise was shown to increase HDLC (Krauss et al., 1977; Miller et al., 1979; Huttunen et al., 1979; Wood and Haskell, 1979), this lipoprotein has taken on an added dimension. Along with the elevated HDLC, a concomitant drop in total serum cholesterol has at times been demonstrated (Wood et al., 1976, 1977; Melish et al., 1978), but, this has not been a clear-cut association. Many researchers have failed to discern any relationship between exercise and serum cholesterol levels (Hurter et al., 1972; Epstein et al., 1976) or HDLC (Frey et al., 1982; Shepherd et al., 1980). As with most variables associated in one way or another with total serum cholesterol or lipoproteins, the evidence is inconclusive and contradictory.

The effect of physical activity on HDLC may be different for women than for men. Frey et al. (1982) observed that most studies citing higher HDLC values in active individuals involve men only. They found no differences in HDLC for sixteen women following a ten-week exercise regimen. Others draw similar conclusions. Moll et al. (1979) propound that HDL is at a normally higher level in women than men, possibly due to hormonal differences, and that this may offset

any changes one might expect to see in women from exercise. Lipson et al. (1979) observed a significant decrease in serum cholesterol ($P < .005$) and a tendency for lower HDL in eleven men and women subjected to an exercise program.

Hormones and Oral Contraceptives

> Virtually every hormone and/or endocrine gland has been examined for its effect on plasma cholesterol levels -- and some effect has been shown for all of them. But winnowing the grain from the chaff in this work is not easy, particularly as there seems to be proportionately little grain for an awful lot of chaff (Sabine, 1977, p. 227).

The already-complex interactions of hormones and their proposed associations with cholesterol and HDLC become even more intricate when evaluating the adolescent. In a population study, one must consider both pre-menarcheal and post-menarcheal subjects, due to hormonal changes that occur throughout adolescence and after menarche (Lee et al., 1975). To complicate the situation further, the lipoprotein fractions have been shown to fluctuate depending on the stage of the menstrual cycle. In one study, levels of total serum cholesterol and LDLC were lowest during the luteal phase in fourteen women over a three-month time interval (Kim and Kalkhoff, 1979). According to these researchers, this dip occurs shortly after 17-β-estradiol reaches its highest concentration in the plasma, just before ovulation. This is in agreement with results from studies in which estrogen induced lower plasma cholesterol in men who had

previously suffered a myocardial infarction (Oliver et al., 1953). Furthermore, Bradley et al. (1978) discovered that estrogens resulted in higher HDLC; those taking progestin had lower HDLC. Women taking hormones in the Lipid Research Clinics study (Heiss et al., 1980) had elevated total cholesterol until age 50. At this point, the opposite held true. In addition, women over 30 on hormones have higher HDLC, and the gap widens with age.

The correlation between myocardial infarction and oral contraceptives has been demonstrated (Mann et al., 1975), as has the influence of oral contraceptives on total serum cholesterol and lipoproteins. Bierenbaum et al. (1979) noted that total cholesterol did not vary, but HDLC was reduced. In another study on girls under 20 years of age (Wallace et al., 1979), the opposite was true; HDLC was higher, as was total serum cholesterol. Bradley et al. (1978) maintain that the effect of oral contraceptives depends on the relative amount and kind of progestin that is combined with the estrogen. Clearly, one cannot predict the effect that oral contraceptives will have on the user, but results do merit a cautious approach to the administration of these drugs.

Of the adolescent studies reviewed earlier, only the Princeton study addressed the topic of oral contraceptives. Morrison et al. (1979) observed a tendency for higher HDLC and LDLC in users than in matched controls.

CHAPTER III

METHODS AND PROCEDURES

This study was part of USDA's Southern Regional Research Project S-150, a more expansive endeavor involving eight states in the southeastern United States. One purpose was to evaluate the nutrient intake of teenage girls in order to relate diet to nutritional health. A variety of biochemical procedures on blood and urine samples was employed to establish vitamin, mineral and lipid status of these subjects. The Regional Technical Committee specified the questionnaires and biochemical methods to be used. Deviations from prescribed methodology were not permitted.

Population and Sample

The population for the regional study in Oklahoma included girls twelve, fourteen and sixteen years old in North Central Oklahoma in the spring of 1981. To qualify as members of these age-groups, the participants had to be born within designated age boundaries. In addition, a small number of subjects of intermediate ages were accepted as part of the local study (see Table II).

TABLE II

QUALIFYING BIRTH DATES TO PARTICIPATE IN REGIONAL STUDY

Age-group	Qualifying Birth Dates
12	September 1, 1968 to August 30, 1969
14	September 1, 1966 to August 30, 1967
16	September 1, 1964 to August 30, 1965

Not only age, but race differences between teenage girls' cholesterol levels were targeted for investigation. Hence, black and white subjects were recruited from major urban (population > 100,000), other urban (population > 2500) and rural (population < 2500) communities in North Central Oklahoma.

Efforts to enlist subjects were initiated through school administrators and teachers. One-hundred fifty teenagers volunteered, including 35 black and 115 white girls. Willing participants and parents were required to sign consent forms. The purpose of the study and the girls' responsibilities were briefly outlined therein (see Appendix A).

At this time, subjects were screened and declared ineligible if they knew they had sickle cell trait, diabetes or any other metabolic disease, or if they were pregnant.

Data Collection

One visit to the Oklahoma State University campus, the high school in a nearby town, or a health clinic in Tulsa made available by Tulsa Medical College was mandatory. Saturday mornings were designated as collection days. Anywhere from fifteen to thirty girls were scheduled per Saturday. Subjects were instructed to eat nothing after 10:00 p.m. the preceding Friday evening. Fasting blood samples were drawn from the arm in heparin-treated vacutainers soon after the girls arrived. The samples were kept on ice until the analyses for total cholesterol and HDLC were run later the same day.

During the visit information about the participant's diet was collected by the 24-hour dietary recall method. The interviewer stated, "Tell me, beginning with the time you got up yesterday morning, everything you ate or drank until you went to bed last night." The subject was then led through her day's activities to aid in prompting her recall of each food or beverage consumed without projecting any notion of what she "should" or "should not" have eaten. Along the way, the nutritionist secured a description of the foods or beverages ingested, the amount consumed, and as precisely as possible, the time of day each item was eaten. To diminish preconceived ideas of serving sizes and amounts, a simple set of three-dimensional food models devoid of demarcations was utilized, similar to those described by Moore et al. (1967). The subject was then

told to indicate the one food model or portion thereof that corresponded to the amount eaten or drunk. When possible, brand names of food items and recipes were obtained. In addition, a parent was interviewed in the home to obtain information regarding types of foods normally used in the household, to assist when the participant could not respond to questions, such as the type of fat in which foods were fried, whether or not the meat was trimmed before cooking, etc. (see Appendix B).

In addition to dietary information, an account of the subject's physical activity was obtained. Exercise levels were estimated by questioning the respondent as to the frequency, duration per day (or session in some cases) and intensity of a given activity (see Appendix C). These questions were asked repeatedly for twenty different types of activity (Table III).

TABLE III

TYPES OF PHYSICAL ACTIVITY MEASURED

Baseball	Bowling
Basketball	Calisthenics
Bicycling	Farming or Gardening
Dancing	Golfing
Racquetball	Swimming
Ice Skating	Soccer
Mountain Climbing or Hiking	Walking
Running	Water Skiing
Tennis	Roller Skating
Volleyball	Snow Skiing

Since the girls were instructed to refrain from food or beverage intake (other than water) after 10:00 p.m. the night before the blood sample was drawn, data concerning eating span and frequency could not be gathered. These variables were determined from a second 24-hour recall conducted when a nutritionist visited each participant's home at another date.

After collecting the dietary information, each food and beverage and the amount consumed were coded from the Nutritional Analysis System assembled by Louisiana State University, Department of Experimental Statistics. Each girl's diet was analyzed for nutrient content by the computer and an average value was obtained for the two dietary interviews for each dietary component.

Eating span and frequency were determined from the times of food or drink consumption quoted by the subject during the interview which did not follow an overnight fast.

Biochemical Analyses

Preparation of Plasma

Blood samples were removed from the ice in which they were stored or transported and promptly centrifuged at 4^{o} C at 1500 x g for 30 minutes. The plasma was immediately transferred to screw-top vials for storage. Aliquots needed for total cholesterol and HDLC determination were

removed and the remaining plasma frozen.

Total Cholesterol Determination

This procedure is based on the Liebermann-Burchard reaction in which blue color develops upon reaction of a reagent[1] containing acetic acid, acetic anhydride, sulfuric acid and phosphoric acid with cholesterol at the C^5-C^6 double bond. Instructions as described in Stanbio literature were followed. Standards provided included 200 mg/dl and 400 mg/dl cholesterol in glacial acetic acid. These were diluted to produce standards of 40, 100 and 200 mg/dl concentration, which were used to prepare a standard curve each time the experiment was run. Only one modification of the procedure as outlined was made; rather than using 0.10 ml of plasma, standards or water (blank), only 0.04 ml was used with a corresponding reduction in volume of the reagent. Because so many biochemical assays were being conducted on a limited amount of plasma, every effort was made to conserve it without sacrificing accuracy. With 0.04 ml plasma, standard curves were linear and values close to those obtained with 0.10 ml plasma were attained. Thus, 0.04 ml of cholesterol standards, plasma and water were carefully transferred to the bottom of clean test tubes, followed by 2.4 ml of cholesterol reagent. The tubes were vortexed until thoroughly mixed and placed in

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[1]Stanbio Laboratory, Inc., San Antonio, Tx.

a 37° C water bath for twenty minutes. The tubes were vortexed again and a spectrophotometer was used to measure color development at 625 nm within 30 minutes after removal from the water bath.

To calculate total cholesterol concentrations, the absorbance of the known standards was plotted against the concentration (mg/dl). From the derived curve, cholesterol concentrations of the unknown samples were determined. All samples were run in duplicate and analyses were repeated if satisfactory agreement of replicates was not obtained.

HDL Separation from Plasma and HDLC Determination

The procedure developed by Albers et al. (1978) was followed for separation of HDL. The method involves the precipitation of the apo-B associated lipoproteins from the plasma using heparin and a divalent cation (Mn^{2+}). The HDL remain in suspension and can be measured by the same methods used for total cholesterol determination.

Plasma samples were permitted to reach room temperature. Two ml of plasma were transferred to centrifuge tubes. Next, 0.2 ml of a combined heparin-manganese reagent was added. The combined reagent was prepared fresh for each run by combining 0.6 ml 40,000 units heparin/ml and 10 ml of 1.06 M manganese chloride. Upon addition of the reagent, a precipitate appeared. The tubes were vortexed and allowed to sit at room temperature

for ten minutes, at which time they were centrifuged in a refrigerated centrifuge for 30 minutes. Samples were analyzed in duplicate. The resulting supernatant solution was decanted and used for HDLC determination by the same procedure previously described for total plasma cholesterol.

Statistical Methods

Simple correlations were determined by the Spearman or Pearson correlation coefficients (Snedecor and Cochran, 1967). Frequencies were evaluated by using the chi-square test (Brown and Hollander, 1977). Duncan's multiple range test aided in establishing significant differences between means. Finally, analysis of variance was employed to investigate effects of race, age, maturity, exercise and diet on cholesterol concentrations and to obtain least squares (adjusted) means (SAS User's Guide, 1979). Unless otherwise noted, alpha equals .05 for all statistical analyses.

CHAPTER IV

RESULTS AND DISCUSSION

In an effort to reduce the high incidence of atherosclerosis and coronary heart disease in man, researchers have strived to identify risk factors associated with these disorders. One of them is high total cholesterol. Thus, it was surprising to find that high HDLC levels were associated with a decreased risk of developing coronary heart disease. To determine and understand the interrelationships of these and other risk factors, investigators have launched expansive studies. Among them have been surveys of pediatric and adolescent populations aimed at investigating whether diet, associated with plasma cholesterol levels in adults, has a similar effect on younger populations. The goal of this study was to examine a female adolescent population for association between cholesterol levels (total plasma cholesterol, HDLC and OPC), age, race and other factors. Factors selected were related by others to coronary heart disease in adult populations and in experimental animals: They included eating patterns, diet, exercise and oral contraceptives. Strong associations would pave the way for investigating the value of initiating preventive measures in childhood to reduce the chances of

developing atherosclerosis and coronary heart disease later in life.

Cholesterol Concentrations by Age, Race and Menarcheal Status

Total plasma cholesterol and HDLC were measured directly for 145 girls. By difference, the combined LDLC and VLDLC (LDLC+VLDLC) value was obtained. Means for the group as a whole are seen in Table IV.

TABLE IV

MEAN TOTAL CHOLESTEROL, HDLC AND LDLC+VLDLC (MG/DL, MEAN±SD) IN 145 FEMALE ADOLESCENTS

Plasma Fraction	Plasma Cholesterol mg/dl ± SD
Total Cholesterol	160 ± 27
HDLC	61 ± 14
LDLC+VLDLC	99 ± 29

Race, menarcheal status and age are variables identified in this study. Table V displays the mean plasma cholesterol values for the subjects by category. Girls

TABLE V

TOTAL CHOLESTEROL, HDLC AND LDLC+VLDLC (MG/DL, MEAN ± SD) IN FEMALE ADOLESCENTS BY RACE, AGE-GROUP, AND MENARCHEAL STATUS

Age-group (yr)	White Adolescents				Black Adolescents			
	n	Total Cholesterol (mg/dl±SD)	HDLC (mg/dl±SD)	LDLC + VLDLC (mg/dl±SD)	n	Total Cholesterol (mg/dl±SD)	HDLC (mg/dl±SD)	LDLC + VLDLC (mg/dl±SD)
12								
Pre-menarche	34	153 ± 23	58 ± 13	95 ± 28	3	168 ± 26	65 ± 9	102 ± 34
Post-menarche	17	155 ± 29	50 ± 14	106 ± 34	7	150 ± 26	65 ± 14	85 ± 36
All	45	154 ± 24	56 ± 13	98 ± 29	10	155 ± 26	65 ± 12	90 ± 34
14								
Pre-menarche	7	168 ± 25	69 ± 14	99 ± 27	0	-	-	-
Post-menarche	33	162 ± 31	60 ± 13	101 ± 32	18	154 ± 20	64 ± 13	90 ± 20
All	40	163 ± 30	62 ± 14	101 ± 31	18	154 ± 20	64 ± 13	90 ± 20
16								
Pre-menarche	0	-	-	-	0	-	-	-
Post-menarche	27	167 ± 28	61 ± 14	106 ± 27	5	171 ± 34	65 ± 19	106 ± 17
All	27	167 ± 28	61 ± 14	106 ± 27	5	171 ± 34	65 ± 19	106 ± 17

were not evenly distributed among them. The population was 75% white and 25% black. This unbalanced distribution is due partially to the lower percentage of blacks in this state than in other states in the study, where equal numbers of both races could readily be obtained as originally proposed for the regional study.

Only three pre-menarcheal black females took part in the study. Therefore, comparison with the 41 white, pre-menarcheal participants is impossible. Also, while 27 white sixteen year-olds participated, only five blacks of that age took part.

As mentioned in the methods section, the procedure used to quantitate cholesterol is based on the Liebermann-Burchard color reaction. Some of the problems inherent to this method are (1) Substances besides cholesterol can produce color, (2) The reaction is light-dependent, and (3) The reaction is time-dependent (Abell et al., 1951). All of these factors enhance the possibility of variation between sets of analyses. Table VI shows the mean total cholesterol and HDLC for the eight collection days. By Duncan's multiple range test, the probability that days one and eight are different from most of the rest is significant for total cholesterol. As Table VI illustrates, HDLC also showed significant variability between sets of analyses. This may be partially explained by the fact that Day 1 was the only day that the samples were frozen for later determination of total cholesterol. In every other

TABLE VI

TOTAL CHOLESTEROL AND HDLC MEANS (MG/DL, MEAN ± SD)
FOR BLOOD COLLECTION DAYS

Day	n	Total Cholesterol (mg/dl ± SD)	HDLC (mg/dl ± SD)
1	26	$181 \pm 23^{a*}$	68 ± 12^{a}
2	13	154 ± 21^{c}	$52 \pm 13^{c,d}$
3	13	141 ± 38^{c}	$67 \pm 9^{a,b}$
4	18	145 ± 25^{c}	71 ± 8^{a}
5	18	156 ± 21^{c}	$64 \pm 7^{a,b}$
6	20	$159 \pm 21^{b,c}$	$59 \pm 15^{b,c}$
7	18	150 ± 21^{c}	50 ± 10^{d}
8	19	$173 \pm 19^{a,b}$	49 ± 13^{d}

*Means in a column with a common superscript do not differ from each other ($P > .05$).

case, the samples were analyzed the day of collection. Furthermore, the first day's samples were analyzed in bright light. Thereafter, all were handled in dim light to minimize fluctuations caused by varying light conditions. Finally, since some collection days included a preponderance of subjects from a particular age or racial group, variation may in some cases reflect age and race differences. To correct for this incongruity, regression models will generally include day of analysis.

Age Differences

Total Cholesterol

Total cholesterol and age were shown to be interdependent by both the Pearson correlation coefficient and chi-square test (see Table VII). The chi-square contingency table was set up to test whether or not the proportion of girls exhibiting total cholesterol at high levels was dependent on age-group. In Table VII, the distribution of subjects among high and low cholesterol categories depended on age.

Since menarche is age-dependent, it was necessary to ascertain whether the effect of age on total cholesterol was independent of stage of maturity. This was accomplished by examining a linear regression on age in an analysis of variance. After eliminating the effect of menarche, age

TABLE VII

CHI-SQUARE TEST* FOR AGE-GROUP AND TOTAL CHOLESTEROL

Total Plasma Cholesterol (mg/dl)	Frequency	Age-group (yr)			Total Frequency
		12	14	16	
> 170	n	12	25	12	49
	n_e**	19.6	18.9	10.5	
	Row %	24.49	51.02	24.49	
≤ 170	n	48	33	20	101
	n_e**	40.4	39.1	21.5	
	Row %	47.52	32.67	19.80	
	Total Frequency	60	58	32	150

*Chi-square = 7.589, DF = 2, P = 0.0225.

**Expected frequency.

still significantly elevated total cholesterol levels (see Table VIII). This substantiates the findings of the Evans County researchers (Hames and Greenberg, 1961), who noted a tendency for total cholesterol to increase from ages six to twenty, although the reported increase was not a significant one. Muscatine investigators (Lauer et al., 1975), however, found no changes in total cholesterol between the ages of six and eighteen. In contrast, the Princeton (Morrison et al., 1978) and Bogalusa (Frerichs et al., 1976) researchers reported a tendency (not significant) for plasma cholesterol levels to fall during adolescence.

TABLE VIII

ANALYSIS OF VARIANCE WITH LINEAR REGRESSION FOR TOTAL CHOLESTEROL ON THE COVARIABLE AGE, ADJUSTED FOR MENARCHEAL STATUS IN FEMALE ADOLESCENTS

Variable	DF	Sum of Squares	F Value	P > F
Menarche	1	576	0.84	0.3605
Age	1	4193	6.13	0.0145

HDLC

The Pearson correlation coefficient showed a tendency for HDLC to increase with age-group (P = .05). As for total cholesterol, the effect of age on HDLC was examined after eliminating the effect of menarche in analysis of variance (see Table IX); HDLC increased with age. Neither the Princeton (Morrison et al., 1977) nor Bogalusa (Frerichs et al., 1976) group reported any change in HDLC values with age. However, the Bogalusa researchers determined HDLC by difference from serum cholesterol and LDLC+VLDLC, and age effects may have been obscured by variability of values estimated indirectly. Of the other adolescent studies described previously, only the Princeton group measured lipoproteins. Further investigation by direct measure of HDLC is warranted to resolve this discrepancy.

TABLE IX

ANALYSIS OF VARIANCE WITH LINEAR REGRESSION OF HDLC ON THE COVARIABLE AGE, ADJUSTED FOR MENARCHEAL STATUS IN FEMALE ADOLESCENTS

Variable	DF	Sum of Squares	F Values	P > F
Menarche	1	300	1.60	0.2077
Age	1	911	4.85	0.0292

LDLC and VLDLC

In this study, LDLC+VLDLC was calculated by difference (see Table V). No effects were noted on LDLC+VLDLC due to age. The Bogalusa study (Srinivasan et al., 1976), on the other hand, reported lower LDLC levels for adolescent females than for girls younger than any reported here. As for VLDLC, it increased with age from five through fourteen in the Bogalusa subjects. Princeton workers (Morrison et al., 1978) also determined lower LDLC for ages twelve to seventeen than for ages six to eleven for white subjects only. Both the Bogalusa and Princeton groups determined LDLC directly.

Racial Differences

Total Cholesterol

The findings of this project corroborate those published by Hames and Greenberg of the Evans County study (1961). In neither case were differences in total cholesterol discovered between participants of different races (see Table V). Even when race was examined independent of day of analysis, age and other variables, no race effect was discerned. This contrasts with the results of the Princeton study (Morrison et al., 1977), which showed a higher total cholesterol for black than for white females for all age-groups evaluated. Bogalusa workers could not find a race difference in total cholesterol among eleven

through thirteen year-olds, but did note a significantly higher total serum cholesterol for black girls in the fourteen year-old age-group. The large number of subjects participating in these two studies should make the detection of differences more sensitive than in the present study.

HDLC

Racial differences were noted for HDLC, but only among the post-menarcheal subjects, for which whites were lower than blacks. Analysis of variance was used to correct for effects established previously, "day" and "age." In addition, menarche and race were included in the model. The least squares mean for the white participants was 57 mg/dl ±2 (SE) and 64 mg/dl±3 for black subjects. The difference is significant. These findings are in agreement with those of both Bogalusa (Frerichs et al., 1976) and Princeton (Morrison et al., 1978) researchers who reported significantly higher HDLC levels for their black adolescent females than for whites of the same age.

LDLC and VLDLC

No racial differences were attributable to the LDLC +VLDLC fraction. Srinivasan et al. (1976) reported higher VLDLC for adolescent girls than for those under twelve. The methodology of the present study precluded the isolation of VLDLC from the LDLC. The only available value is

that from the combined LDL and VLDL fractions, which did not reflect a difference between races.

Age Effects Within Maturity Groups

It is almost certain that hormones play a role in regulating total cholesterol levels, but the nature of the influence is unknown. Thus, the sample was divided into pre-menarcheal and post-menarcheal categories to investigate each group separately.

It is very difficult to draw conclusions about the pre-menarcheal subjects from this study, since they numbered only 44. Furthermore, 41 of the 44 were white, and 34 of the 41 white, pre-menarcheal girls were members of the 12 year-old age-group (Table V). Hence, attempting to establish race or age differences between these very small groups is difficult. Perhaps due to the small size of the sample, or maybe because hormonal activity is erratic during adolescence, no clear-cut associations could be made of total cholesterol, HDLC or the remaining LDLC +VLDLC fraction within the pre-menarcheal girls. However, general trends were apparent for both groups. Figures 1 and 2 present the raw means for pre-menarcheal and post-menarcheal girls by age and race. Although differences are not significant, post-menarcheal white girls had higher total plasma cholesterol and LDLC+VLDLC than blacks at ages twelve and fourteen. The 16 year-old black girls, on the other hand, revealed higher total cholesterol than whites,

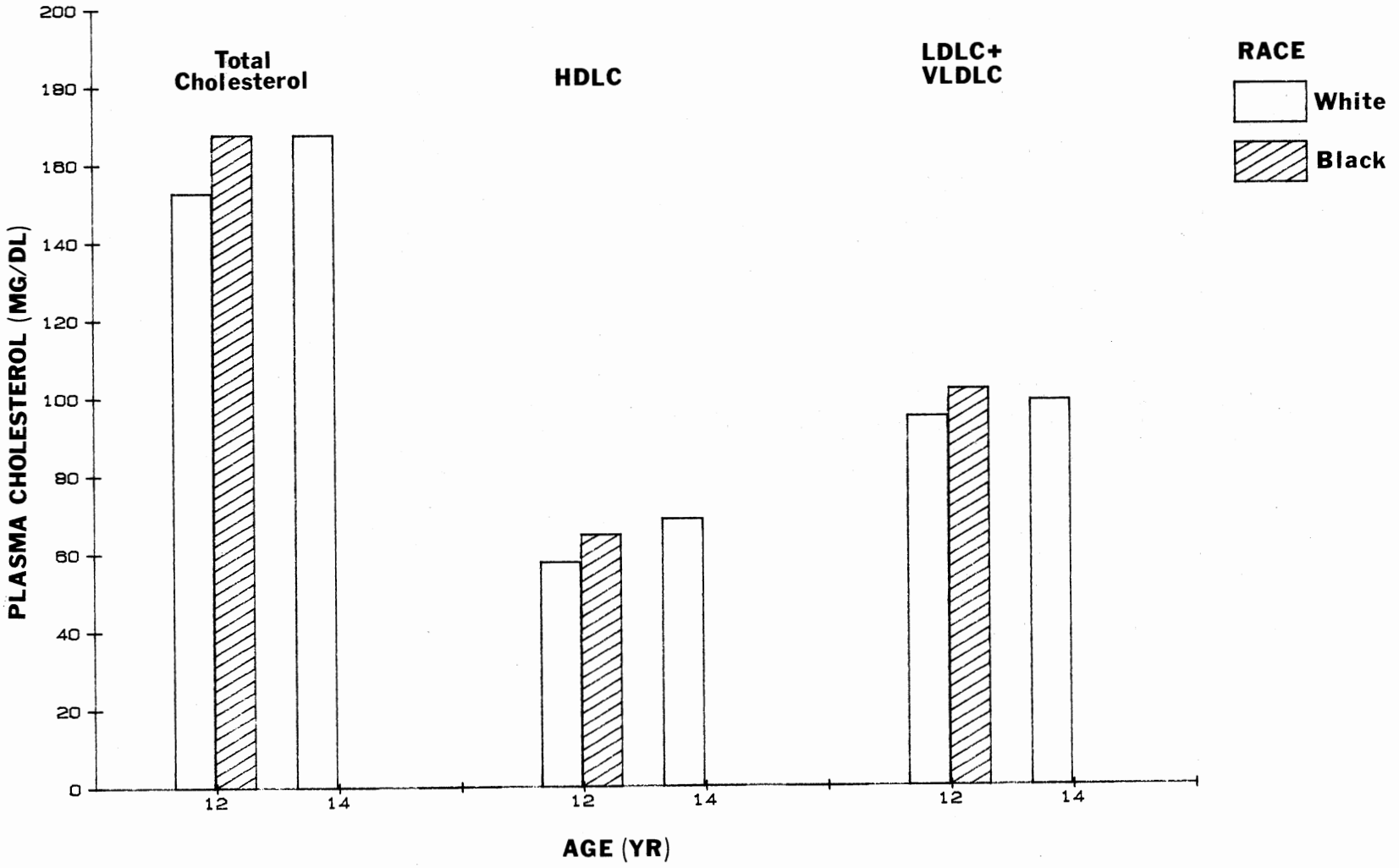

Figure 1. Pre-menarcheal Cholesterol Levels (mg/dl) by Race and Age-Group

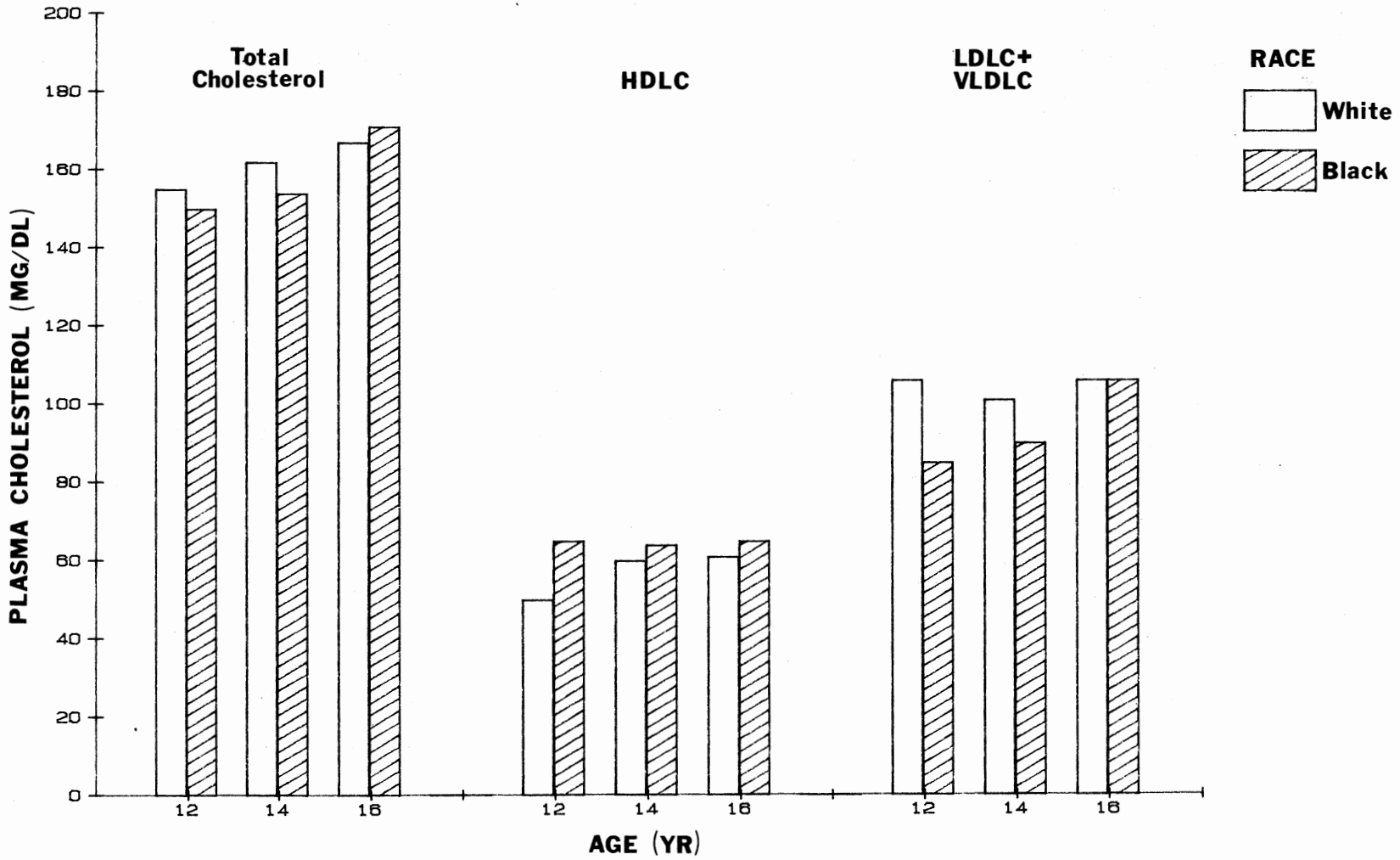

Figure 2. Post-menarcheal Cholesterol Levels (mg/dl) by Race and Age-Group

and equal LDLC+VLDLC concentrations. The HDL fraction is significantly higher in blacks than whites for all three post-menarcheal age-groups, while the 12 year-old black girls displayed higher concentrations of total cholesterol, HDLC and LDLC+VLDLC than white girls of that age.

Of the hormones, estrogen and progesterone levels change most dramatically during puberty. The progesterone is thought to increase after menarche (Lee et al., 1975). This and the fact that alterations have been shown to follow a cyclic pattern (Kim and Kalhkhoff, 1979) make associations between hormonal status and cholesterol levels difficult to demonstrate in survey data from fewer than several hundred individuals. Although one study (Kim and Kalkhoff, 1979) presented evidence that total cholesterol and LDLC fall to their lowest levels during the luteal phase, this study did not. After grouping post-menarcheal girls according to day of menstrual cycle reported on the morning of blood collection, cholesterol values of girls between days 1 and 14 were compared with those later in the menstrual cycle. The chi-square test revealed no difference.

HDLC data painted a more lucid picture in post-menarcheal girls. An analysis of variance linear regression model was tested for this group. Because exercise has been shown to exert an effect on HDLC (Miller et al., 1979; Krauss et al., 1977; Huttunen et al., 1979), time reported spent in vigorous exercise was introduced into the model

(Exercise will be discussed in detail elsewhere). Vigorous exercise was defined as activity inducing profuse sweating and/or very tiring. With this and a correction for day of analysis, post-menarcheal subjects showed age and race differences in HDLC (see Table X). Black participants had significantly higher HDLC levels than whites. Also, HDLC increased with age for post-menarcheal girls as in the entire sample. Since estrogen levels increased after the onset of menstruation, and because estrogens have been shown to elevate HDLC (Bradley et al., 1978), this finding is a logical one. No such association was found for pre-menarcheal girls alone.

TABLE X

ANALYSIS OF VARIANCE WITH LINEAR REGRESSION OF HDLC IN POST-MENARCHEAL FEMALE ADOLESCENTS BY DAY OF ANALYSIS, RACE, AGE AND TIME SPENT IN VIGOROUS EXERCISE

Variable	DF	Sum of Squares	F Values	P > F
Day of Analysis	7	5700	5.86	0.0001
Race	1	645	4.64	0.0339
Time Spent in Vigorous Exercise	1	201	1.45	0.2323
Age	1	730	5.25	0.0243

This study was to include information about subjects taking oral contraceptives to further explore the hormonal connection to cholesterol levels, especially since this medication has been associated with increased risk of myocardial infarction (Mann et al., 1975). But, only two subjects of the 150 stated that they were currently taking oral contraceptives, so that aspect of the study was omitted.

Eating Frequency and Eating Span

The means and range for eating frequency and span are outlined in Table XI.

TABLE XI

MEAN (MG/DL±SD) AND RANGE BY RACE OF EATING FREQUENCY AND EATING SPAN

Race	n	Mean±SD	Minimum	Maximum
White				
Frequency	112	4.4±1.4	2.0	10.0
Span	112	11.6±2.8	3.0	17.5
Black				
Frequency	35	4.3±1.5	2.0	9.0
Span	35	11.0±2.8	5.8	16.3

By the Spearman correlation method, eating span was positively correlated with energy, starch, free glucose, total carbohydrate and cholesterol intakes (see Table XII), and there was a tendency for greater total protein and saturated fatty acid consumptions ($P < .1$). Eating frequency was positively correlated with energy, animal protein, vegetable protein, total protein, saturated fatty acids, unsaturated fatty acids, total fat, sodium, cholesterol, free glucose, sucrose, starch and total carbohydrate intake. These findings suggest that eating frequency, more than eating span, was associated with increased consumption of foodstuffs most often related to high total plasma cholesterol. One might expect, then, that those eating with the greatest frequency would exhibit a greater tendency for higher total cholesterol than those with a long eating span. This was not the case. To the contrary, no effect of frequency or span on total cholesterol was found by simple correlation, analysis of variance or chi-square test.

By comparison, Bogalusa investigators (Frank et al., 1978) also found that longest eating span and greatest eating frequency meant significantly higher consumption of carbohydrate, protein, fat, energy and sodium. In addition, those with the greatest eating frequency ingested more sucrose, starch, saturated and polyunsaturated fatty acids, iron, calcium and cholesterol than those eating less frequently (see Table XII). Here again, eating frequency

TABLE XII

A COMPARISON OF FINDINGS BETWEEN THE BOGALUSA HEART STUDY* AND OKLAHOMA STATE UNIVERSITY ADOLESCENT FEMALE STUDY RELATING EATING FREQUENCY AND EATING SPAN TO DIETARY VARIABLES

	Energy	Cholesterol	Vegetable Protein	Animal Protein	Total Protein	Total Carbohydrates	Free Glucose	Starch	Sucrose	Total Fat	SFA	PUFA	Sodium	Calcium	Iron
Eating Frequency															
O.S.U.	+	+	+	+	+	+	+	+	+	+	+	+	+	NI	NI
Bogalusa	+	+	NI	NI	+	+	NI	+	+	+	+	+	+	+	+
Eating Span															
O.S.U.	+	+	•	•	X	+	+	+	•	•	X	•	•	NI	NI
Bogalusa	+	•	NI	NI	+	+	NI	•	•	+	•	•	+	•	•

*Frank et al., 1978.

\+ = Significant Positive Correlation ($P < .05$).
• = No Correlation Noted.
X = Tendency for Positive Correlation ($P < .1$).
NI = Not Identified for Study.

more than span influenced intake of nutrients associated with high total cholesterol. Although those with the longest eating span in the Bogalusa study had higher serum cholesterol levels than subjects in the low and moderate categories, frequency and total cholesterol levels were not related. This was surprising, because saturated fat and sucrose intakes were significantly associated with eating frequency, but not eating span. Bogalusa workers also noted that those who ate the least frequently had the highest HDLC. This study was unable to corroborate that claim.

Although one would expect to relate these parameters (span and frequency) to plasma cholesterol levels, the problem must be a more complex one, at least for adolescent populations, than this study could clarify. One must also point out the limitations of using one 24-hour recall as a reflection of a person's general eating pattern.

Dietary Factors

It is an arduous task to sort the effect of dietary factors from the many other conditions that have been shown to affect total and HDL cholesterol. The dietary variables included in the analyses of this study were energy, total protein, vegetable protein, animal protein, total fat, saturated fat, polyunsaturated fat, hydrogenated fat, sodium, cholesterol, free glucose, sucrose, starch and total carbohydrate. Table XIII presents the mean intakes

TABLE XIII

MEAN±SD AND RANGE OF DIETARY COMPONENTS ANALYZED FOR STUDY

Nutrient	White Adolescents n=115			Black Adolescents n=33		
	Mean±SD	Minimum	Maximum	Mean±SD	Minimum	Maximum
Sodium (mg)	2907±1488	744	10,683	3051±1102	1071	5984
Cholesterol (g)	258±137	50	722	242±105	79	525
Sucrose (g)	67±34	3	175	75±38	13	161
Starch (g)	57±28	8	160	52±31	4	150
Free Glucose (g)	12±93	0.2	44	11±9.2	0.9	42
Total Carbohydrate (g)	213±76	48	423	223±79	95	438
Animal Protein (g)	45±20	10	126	41±15	12	80
Vegetable Protein (g)	15±7.4	3	53	13±6.0	2	30
SFA (g)	29±13	8	89	30±11	7	52
PUFA (g)	14±8.1	1	65	17±11	4	55
Hydrogenated Fat (g)	12±9.8	0	52	13±11	0.7	38
Total Fat (g)	80±33	25	246	89±33	31	187

and ranges for these components by race. The only one effecting a simple correlation with plasma cholesterol in this study was sodium with HDLC. The reason for this unexpected relationship is unclear. Sodium is, however, one of the most difficult nutrients to quantitate from a dietary interview. Subjects often do not know how much salt was used in cooking the food they consumed. Also, it is difficult to get an accurate measure of how much salt one used at the table. Finally, since teenagers are notorious for their consumption of convenience foods, they may be expected to ingest large quantities of sodium (Table XIII). All of this makes an accurate record of sodium intake extremely difficult to obtain, and enhances the possibility of introducing error, making interpretation of a relationship of sodium and HDLC difficult.

The lack of correlation between plasma cholesterol and diet has become a hallmark of adolescent studies. Morrison et al. (1980), for instance, had to

> exclude children having plasma cholesterol, triglycerides, and calories less than or equal to the first or greater than or equal to the 99th percentiles for the random recall children (p. 727).

in order to determine that plasma cholesterol was inversely correlated with dietary sucrose and that LDLC was negatively correlated with the polyunsaturated/saturated fat ratio, total carbohydrate and sugar. In the same vein, Frank et al. (1978, p. 328) stated that "a lack of correlation was noted in large matrices of dietary components and risk

factor variables" in the Bogalusa study. He subsequently grouped children according to risk factor variables and found that "the children with the middle and high serum cholesterol values showed significantly greater fat intakes than those with the lowest serum cholesterol levels" (p. 336). Clearly, the associations are tenuous at best.

It was not altogether unforeseen, then, that this study would fail to find simple correlations between diet and cholesterol levels. More sophisticated statistical techniques, however, did show a significant and independent effect of exogenous cholesterol on total plasma cholesterol and on HDLC. Included in an analysis of variance linear regression model were race, day of analysis, age, time spent in vigorous exercise and exogenous cholesterol. The results are outlined in Table XIV.

The same model was used to test the other dietary variables for an independent effect, but none was found. Much of the evidence supporting a role for diet in affecting cholesterol levels has involved changing a dietary regimen in already hyperlipidemic individuals. For example, changing from a diet high in animal protein to a vegetable protein diet has been shown to lower total serum cholesterol (Olson et al., 1958); or substituting unsaturated for saturated fats can cause a reduction of total serum cholesterol (Ahrens et al., 1957; Turpeinen et al., 1968). In these and other cases, a change over time is noted due to an extreme change in a subject's diet. In this study, cholesterol

TABLE XIV

ANALYSIS OF VARIANCE WITH LINEAR REGRESSION OF TOTAL CHOLESTEROL AND HDLC ON THE COVARIABLE CHOLESTEROL (EXOGENOUS), ADJUSTED FOR RACE, DAY OF ANALYSIS, AGE AND TIME SPENT IN VIGOROUS EXERCISE

TOTAL PLASMA CHOLESTEROL

Variable	DF	Sum of Squares	F Values	P > F
Day of Analysis	7	21940	5.72	0.0001
Race	1	619	1.13	0.2897
Age in Months	1	452	0.83	0.3652
Time Spent in Vigorous Exercise	1	866	1.58	0.2107
Exogenous Cholesterol	1	2652	4.84	0.0295

HDLC

Variable	DF	Sum of Squares	F Values	P > F
Day of Analysis	7	8685	10.09	0.0001
Race	1	413	3.36	0.0690
Age in Months	1	576	4.68	0.0322
Time Spent in Vigorous Exercise	1	240	1.95	0.1649
Exogenous Cholesterol	1	620	5.04	0.0264

levels were examined in healthy individuals consuming self-selected diets, which make variables in plasma cholesterol induced by diet difficult to detect.

Exercise

Due to the controversy and contradictions in published findings surrounding the effect of exercise on cholesterol levels, and, in particular, on the HDLC fraction, this topic has spurred numerous studies aimed at delineating the mechanism by which physical activity may elevate HDLC, and in so doing, exert a protective effect against CHD. Most of the research on this topic has been published in the last five or six years, and deals primarily with the adult male. Since the effect is thought to be hormonally regulated, the findings that exercise may increase HDLC in men may not apply to women. In fact, this is the position taken by Moll et al. (1979) and Frey et al. (1982). In neither case were differences in HDLC attributable to exercise in adult women. None of the adolescent studies outlined herein have pursued an association between plasma cholesterol and exercise.

The results from this study tend to support the premise that women will not necessarily respond in the manner that men do to exercise. For analysis, subjects reported the minutes per day, week or month that they engaged in each of three categories of exercise: light exercise, defined as causing no sweating, not tiring;

moderate exercise, defined as causing sweating, somewhat tiring; and vigorous exercise, causing profuse sweating, tiring to exhausting. The means and range for these categories are outlined in Table XV. Surprisingly, those engaging in vigorous exercise showed a negative correlation with HDLC by the Pearson correlation method. This finding is in opposition to studies on adult populations that present evidence for a positive correlation between exercise and HDLC. However, girls are generally thought to become less active with age. Therefore, by analysis of variance linear regression, when the effect of age and day of analysis were eliminated, the time spent in vigorous exercise no longer had an independent effect (P = .2875).

TABLE XV

MEAN (HOURS/WEEK)±SD AND RANGE BY RACE FOR TIME SPENT IN VIGOROUS, MODERATE AND LIGHT EXERCISE

Race	Exercise Category	n	Mean±SD (hrs/wk)	Minimum	Maximum
White	Vigorous	111	2.94±4.06	0	18.0
	Moderate	111	8.45±8.96	0	49.3
	Light	111	10.2±10.1	0	44.5
Black	Vigorous	34	3.62±6.05	0	26.5
	Moderate	34	11.8±10.8	0.7	36.9
	Light	34	11.0±12.0	0	49.5

If women do respond differently than men to exercise because of hormonal differences between sexes, then girls in the midst of puberty may respond differently still. Additional study is needed in this area to clarify the effect of physical activity on adolescent populations.

General Discussion

The results of this study led to the conclusion that total plasma cholesterol and HDLC increase with age between twelve and sixteen. But the relative effects of hormones, eating patterns, nutrient intake and exercise are very difficult to interpret from available information.

The stage of maturation certainly affects hormonal status in adolescent girls. However, the time of transition from pre-puberty to post-puberty is not uniform in all subjects. This makes analysis of data from a cross-sectional study of teenagers extremely complex. Even in a longitudinal study, determining the stage of maturation is a challenging task. At best, one hopes that in numbers most of the variation between age and level of maturation can be evaluated.

Despite the fact that those with the longest eating frequency and span consumed more of those foods known to elevate plasma serum lipids in adults, no associations were found between span or frequency and plasma lipids. Hence, based on these results, there is no reason to attempt to influence the eating frequency or span of

teenagers, except to prevent other sequelae of poor eating habits such as obesity due to greater food intake with longer span or greater frequency.

Diet, too, poses a problem when contemplating whether or not to recommend a reduction of intake of those foods associated with increased risk of coronary heart disease later in life. Evidence from this study suggests that even teenagers would benefit by monitoring their cholesterol intake, since plasma cholesterol depended on the amount of this sterol in the diet. As for other foodstuffs known to affect cholesterol levels in adults, this study failed to establish a relationship.

Finally, exercise did not elevate HDLC. Further research is needed to unravel the mystery surrounding exercise and its effect on HDLC, especially in women. Also, most research to date has involved sedentary individuals suddenly placed in rigorous exercise programs for a few weeks, after which time the change in their cholesterol is determined. Studies are needed to establish the persistence and benefits of a regular, prolonged exercise program, if it can elevate the HDL fraction in women.

In conclusion, exploring the association of various risk factors and plasma cholesterol levels in adolescents is a complex issue. But the effort is well worth it, considering the extremely high mortality rate from coronary heart disease in this country. To achieve this end, the

puzzle must be taken apart, and then put back together, piece by piece.

CHAPTER V

SUMMARY AND CONCLUSIONS

Summary

The purpose of this study was to establish associations between plasma cholesterol values and age, race, dietary practices and exercise in 150 adolescent girls in North Central Oklahoma. Total cholesterol and HDLC were measured directly in plasma, while the combined LDLC and VLDLC value was calculated by difference. Dietary information was obtained by the 24-hour recall method on two different occasions. From this, nutrient content of the diet was determined. Eating frequency and eating span were based on one 24-hour dietary recall. Girls were questioned in detail about the extent to which they engaged in twenty different types of exercise. Activity in minutes per day was assigned to each of three categories: vigorous activity, moderate activity and light activity. Based on these data, simple correlation, analysis of variance, chi-square and Duncan's multiple range test were employed for statistical analysis.

Conclusions

Age Differences

Total plasma cholesterol and age were positively correlated. The effect of age on total cholesterol was independent of menarcheal status. After dividing the girls into pre-menarcheal and post-menarcheal categories, no associations between total plasma cholesterol and age were discovered. HDLC showed a tendency to increase with age by simple correlation. After eliminating the effect of menarche, the increase was significant. Age did not influence the LDLC+VLDLC fraction calculated by difference.

Racial Differences

No differences in total plasma cholesterol were attributable to race, neither for the group as a whole nor for pre-menarcheal nor post-menarcheal girls. Among post-menarcheal subjects, blacks had higher HDLC than whites, after adjusting for the effect of day of analysis and age in an analysis of variance. No racial differences were noted for LDLC+VLDLC.

Dietary Factors

The dietary variables examined in this study were energy, total protein, vegetable protein, animal protein, total fat, saturated fat, polyunsaturated fat, hydrogenated fat, sodium, cholesterol, free glucose, sucrose, starch and

total carbohydrate. Of all these dietary components, only sodium was found to be positively correlated with HDLC. After eliminating the effects of day of analysis, race, age and time spent in vigorous exercise in an analysis of variance, dietary cholesterol significantly increased both total plasma cholesterol and HDLC.

Eating Span and Eating Frequency

Eating span was positively correlated with the consumption of energy, starch, free glucose, total carbohydrate and cholesterol. Eating frequency was positively correlated with energy, animal protein, vegetable protein, total protein, saturated fatty acids, unsaturated fatty acids, total fat, sodium, cholesterol, free glucose, sucrose, starch and total carbohydrate. No associations between either span or frequency and total cholesterol or the lipoprotein fractions could be uncovered.

Exercise

Simple correlation showed exercise to be negatively correlated with HDLC. After adjusting for day of analysis and age, the association was no longer apparent.

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APPENDIX A

INFORMED CONSENT

Oklahoma State University

Department of Food, Nutrition and Institution Administration

STILLWATER, OKLAHOMA 74078
(405) 624-5039

CONSENT TO PARTICIPATE

I have been told about the nutrition study at Oklahoma State University to find out how teenage girls eat, how this affects their health, and why they choose the foods they eat. I understand that being in this study means that I agree to:

1) Go to a designated place for a free physical examination by a doctor or nurse and/or a dental examination by a dentist each year I participate.
2) Allow a medically qualified person to take about 30 milliliters (about 2 tablespoonfuls) of blood from a vein in my arm and not eat that day until after the blood is drawn.
3) Bring a urine sample in the container provided.
4) Tell about what I eat, physical activities I take part in, personal health habits, and menstrual cycle.
5) Take tests to describe my personality and behavior.
6) Be studied again in 2 years, if I am now 12 or 14 years old.

I understand that I will be well cared for, can ask questions at any time, and can drop out of the study any time I am sure I want to. Any unusual findings from the tests will be reported to me, and no information about me will be given to other people.

I voluntarily agree to be in this study.

_____ _____
Daughter Date

PARENT'S CONSENT FOR CHILD'S PARTICIPATION

Having legal custody of and responsiblity for_____,
I authorize Oklahoma State University, the Oklahoma Agricultural Experiment Station, Dr. Mary Alice Kenney and whomever she may designate, to carry out the procedures my daughter has agreed to and which have been explained to me. We assume whatever risk is involved, but my consent to be in this study does not mean we give up any legal rights or release the persons in charge from liability due to negligence. I understand that all information about our family will be strictly confidential.

I (or the adult female manager in the household, if it is not I) will give information each year about the family, our food buying habits, income and my daughter's past health as well as my opinions about how people in general get along. I can ask questions or withdraw from the study at any time.

_____ _____
Parent or guardian Date

APPENDIX B

DIETARY INFORMATION SUPPLEMENT

Form D4 Supplemental data on food use and purchase for home

Subject number_ _ _ _Subject name_____Date_____Interviewer_____

1. What type of vegetables do you usually use?
   __canned
   __frozen
   __fresh

2. What spread is used at the table? __butter __stick margarine __soft margarine

3. What kind of fat is used to fry? __solid shortening (brand = _____)
   __oil (brand = _____)

4. What kind of fat do you usually use in baking? __butter __shortening
   __margarine __oil
   (brand, if different:_____)

5. What kind of milk is usually bought for your daughter to drink?
   __Skim __½% __1-1½% __2% __whole __chocolate skim __chocolate-whole

6. Does your child usually take vitamins? __Yes __No
   If Yes, what brand?_____ Does it contain iron?_____

7. What ground meat is usually purchased?
   __regular ground beef (60-70%lean) __extra lean (80-90% lean)
   __ground chuck __ ground round

8. Do you trim fat from meat before cooking it? __Yes __No __Part of it

9. Does your daughter like to eat the fat that is cooked on meat? __Yes __No

10. Do you skin chicken before cooking? __Yes __No

11. What kind of salt to you buy? __Plain __Iodized.

12. What size egg do you buy? Small_____ Medium____ Large____ X Large____.

APPENDIX C

PHYSICAL ACTIVITY QUESTIONNAIRE

(FIRST PAGE)

S-150 REGIONAL PROJECT — SUBJECT ___ ___ ___ (1 2 3)

FORM A7 — STATE ___ (4)

SUBJECT NO. _____ — STATION ___ (5)

DATE _____ — YEAR 1 OR 3 ___ (6)

EXERCISE/ACTIVITY LEVELS

THE FOLLOWING SET OF INFORMATION NEEDS TO BE ASKED FOR EACH ACTIVITY WHICH THE SUBJECT HAS PARTICIPATED IN DURING THE PAST YEAR.

FREQUENCY (YEARLY BASIS)

1. ONCE OR TWICE/YEAR
2. MONTHLY
3. WEEKLY

SEASONALLY

1. YES - LESS THAN OR EQUAL TO 6 MONTHS
2. NO - MORE THAN 6 MONTHS

OF DAYS/WEEK

ANSWER ONLY IF YEARLY BASIS IS WEEKLY, BY PUTTING THE APPROPRIATE NUMBER OF DAYS.

DURATION/DAY

_____ MINUTES

INTENSITY SCALE

1. LIGHT (NOT TIRING, OR NO SWEATING)
2. MODERATE (SOMEWHAT TIRING, OR SWEATING)
3. VIGOROUS (TIRING TO EXHAUSTING, OR PROFUSE SWEATING)

****

1. BASEBALL OR SOFTBALL	-	FREQUENCY (YEARLY BASIS)	(7)
		SEASONALLY	(8)
		# OF DAYS/WEEK	(9)
		MINUTES	(10 11 12)
		INTENSITY	(13)
2. BASKETBALL	-	FREQUENCY (YEARLY BASIS)	(14)
		SEASONALLY	(15)
		# OF DAYS/WEEK	(16)
		MINUTES	(17 18 19)
		INTENSITY	(20)
3. BICYCLING	-	FREQUENCY (YEARLY BASIS)	(21)
		SEASONALLY	(22)
		# OF DAYS/WEEK	(23)
		MINUTES	(24 25 26)
		INTENSITY	(27)

APPENDIX D

INDIVIDUAL DATA

INDIVIDUAL DATA

SUBJ	AGE	RACE	MENS	DCYC	DAY	SCHOL	HDLC	LDLC	CHOL	SFA	PUFA	FAT	N31	N6	N7	PRO	N50	SUCR	STAR	N45	NA	LEX	MEX	VEX	SPAN	FREQ
1	146	1	2	.	2	169.86	84.87	84.99	444	25.0	13.5	74	17.4	52.4	29.7	84	15.1	37	103	264.8	3269	24.4	5.5	0.0	12.50	6
2	189	1	1	3	1	142.50	54.00	88.50	135	38.6	26.5	111	12.0	35.1	20.2	61	4.6	43	118	241.1	4165	0.0	26.6	0.0	9.75	4
3	168	1	2	.	1	190.38	63.00	127.38	265	33.1	14.6	98	10.7	57.2	17.0	90	13.6	74	69	265.0	6081	5.5	4.6	8.5	10.50	4
4	187	1	1	24	1	205.20	92.00	113.20	449	38.3	20.6	108	8.3	32.0	26.9	65	19.7	105	84	346.3	3958	6.2	1.0	0.0	13.50	5
5	162	1	1	4	1	177.84	52.00	125.84	242	20.0	7.5	53	8.8	35.1	11.1	47	4.3	41	46	127.6	2002	2.5	12.1	0.0	11.50	4
6	189	1	1	3	1	204.06	56.00	148.06	175	28.6	17.8	76	0.0	44.8	8.0	60	3.1	37	37	178.3	2723	15.3	32.0	0.1	11.50	5
7	166	1	2	.	1	176.70	71.00	105.70	310	48.0	16.0	119	8.3	84.3	24.2	109	18.3	78	91	256.9	2591	0.0	6.6	4.5	11.75	6
8	193	1	1	26	1	182.40	83.00	99.40	96	17.6	7.4	46	8.1	22.4	10.3	33	10.5	48	46	134.6	876	6.8	2.5	0.0	12.50	5
9	174	1	2	.	1	201.78	73.00	128.78	452	34.6	10.2	86	10.3	55.1	11.7	67	5.0	69	31	161.2	2834	0.2	3.6	1.2	10.83	5
10	163	1	1	32	1	136.80	90.00	46.80	289	29.0	6.9	62	0.0	20.2	11.6	32	4.0	70	38	132.8	785	16.2	6.7	8.1	13.25	3
11	189	1	1	11	1	197.22	67.00	130.22	219	17.3	24.6	68	3.3	19.9	5.6	32	5.1	30	22	94.6	1953	17.1	4.1	0.0	13.50	3
12	193	1	1	.	1	172.14	74.00	98.14	215	29.7	11.5	82	8.4	51.8	7.4	62	3.7	3	36	71.2	2323	1.3	0.0	0.0	8.00	2
13	172	1	1	25	1	157.32	71.00	86.32	170	27.7	5.5	70	7.5	31.7	10.3	53	6.6	41	53	184.6	2526	3.6	1.4	0.0	10.58	4
14	165	1	1	34	1	150.48	86.00	64.48	336	34.0	10.8	87	4.0	64.7	11.2	94	21.0	84	44	310.2	3768	2.5	13.4	6.2	12.25	5
15	164	1	1	4	1	212.04	64.00	148.04	257	45.6	9.5	106	19.5	69.6	18.3	90	4.5	53	75	206.2	4424	0.3	5.0	1.0	16.75	5
16	162	1	1	18	1	159.60	72.00	87.60	272	12.6	6.2	42	5.8	53.8	5.0	69	4.5	26	22	123.0	1542	0.6	4.8	0.0	11.00	4
17	175	1	1	12	1	186.96	67.00	119.96	262	38.2	9.4	91	9.7	78.3	15.7	105	10.3	72	68	234.3	5006	4.1	49.3	0.0	11.50	4
18	162	1	1	26	1	157.32	56.00	101.32	96	14.3	8.5	44	18.6	25.6	10.0	36	5.9	45	51	122.2	1504	1.2	1.5	0.8	13.50	4
19	149	1	2	.	3	134.52	54.50	80.02	279	24.8	13.4	77	16.3	40.2	11.4	59	9.3	36	51	180.9	2068	10.6	4.8	3.5	11.50	6
20	165	1	2	.	2	157.32	47.97	109.35	483	55.5	36.1	149	10.6	87.2	15.2	102	12.3	107	61	301.9	4458	3.4	19.0	2.0	13.75	6
21	193	1	1	.	1	210.90	61.00	149.90	127	18.8	14.1	60	10.7	29.1	9.0	39	12.1	55	36	153.0	1208	2.2	0.3	0.0	9.25	5
22	193	1	1	20	1	159.60	81.00	78.60	225	33.0	13.8	85	8.3	58.0	21.0	80	20.4	53	95	243.2	4348	0.1	5.3	0.1	13.25	6
23	179	1	1	.	1	161.88	61.00	100.88	102	13.0	1.4	31	0.0	20.4	3.2	34	3.0	49	8	135.2	2487	.	.	.	9.25	3
24	174	1	1	26	1	218.88	68.00	150.88	137	21.8	11.6	61	3.1	26.9	4.8	34	38.0	58	8	131.1	1724	10.5	1.4	0.0	9.00	4
25	173	1	1	4	1	175.56	56.00	119.56	110	20.1	7.0	50	6.8	23.2	9.5	36	8.6	34	46	122.1	1833	0.1	3.6	0.0	12.00	3
26	198	1	1	22	2	152.76	66.42	86.34	405	29.1	14.2	105	16.2	51.3	11.2	85	11.6	90	50	301.5	3306	3.8	9.4	9.0	11.50	4
27	173	1	1	3	1	213.18	56.00	157.18	96	14.7	4.9	33	0.9	29.3	5.6	42	1.0	15	17	86.6	3447	0.9	15.8	0.2	14.00	6
28	195	1	1	6	1	186.96	68.00	118.96	348	29.7	16.4	92	28.5	39.9	27.3	70	44.4	137	99	361.0	3045	0.0	0.0	0.1	10.50	5
29	164	1	1	25	1	191.52	54.00	137.52	129	9.6	5.1	40	2.2	28.0	3.8	49	3.8	68	16	146.9	2137	2.8	6.5	0.0	5.92	2
30	173	1	1	23	1	173.28	77.00	96.28	223	17.3	11.2	59	18.7	41.7	8.8	56	13.1	42	64	180.5	4740	6.7	3.3	0.0	10.00	3
31	143	1	2	.	2	111.72	41.82	69.90	317	45.0	13.3	104	5.3	83.2	13.6	97	5.3	37	46	174.0	2865	21.0	1.9	3.1	13.50	4
32	188	1	1	8	3	152.76	58.86	93.90	224	31.0	15.5	96	12.2	52.3	16.1	82	6.5	41	68	219.7	4636	7.4	5.9	18.0	6.25	3
33	148	1	2	.	7	164.16	55.59	108.57	498	40.3	11.7	97	9.6	78.4	16.2	96	15.1	54	68	201.5	3178	1.8	2.0	0.8	8.50	3
34	188	1	1	.	8	153.90	49.05	104.85	223	34.9	14.2	100	24.5	41.4	19.8	64	29.2	125	97	331.7	2979	6.1	3.4	7.5	12.50	6
35	172	1	1	10	3	98.04	59.95	38.09	166	27.0	14.8	75	6.2	39.3	7.5	59	1.6	31	45	189.3	2234	11.2	5.7	1.0	11.75	5
36	144	1	2	.	3	164.16	64.31	99.85	554	46.3	20.2	115	17.2	51.2	27.1	83	15.3	112	53	307.3	3641	30.1	17.5	2.7	12.00	4
37	189	1	1	14	3	168.72	53.41	115.31	722	49.4	16.8	125	14.9	46.6	9.3	61	8.1	16	56	152.0	3044	10.1	8.1	0.6	.	.
38	146	3	2	.	2	196.08	57.81	138.27	281	42.7	26.9	124	35.1	49.9	23.0	74	7.1	59	97	245.7	3790	14.2	18.2	0.0	.	.
39	168	3	1	.	2	142.50	35.67	106.83	415	35.0	16.9	98	33.3	56.8	40.3	104	14.8	65	132	312.5	4418	19.0	10.8	26.5	14.75	4
40	140	1	2	.	8	141.36	39.24	102.12	128	24.7	8.9	63	6.0	39.5	10.8	50	18.7	38	37	160.1	1972	26.4	2.8	0.0	12.50	4
41	147	1	1	23	2	163.02	41.82	121.20	116	20.4	7.5	56	13.2	50.5	14.4	72	5.9	22	38	174.9	2492	15.3	5.3	0.0	12.50	4
42	169	1	1	10	2	131.10	47.97	83.13	333	29.2	18.1	88	13.1	54.3	17.1	75	6.7	14	60	142.3	3020	0.1	29.7	0.0	11.00	3
43	140	1	2	.	2	156.18	51.66	104.52	326	47.2	20.3	123	33.2	75.7	19.9	98	18.2	55	73	247.3	3385	3.5	5.9	0.0	8.50	3

SUBJ	AGE	RACE	MENS	DCYC	DAY	SCHOL	HDLC	LDLC	CHOL	SFA	PUFA	FAT	N31	N6	N7	PRO	N50	SUCR	STAR	N45	NA	LEX	MEX	VEX	SPAN	FREQ
44	150	1	2	.	8	191.52	41.42	150.10	312	40.5	19.2	105	26.2	76.3	16.6	98	27.9	99	76	363.2	3731	15.3	7.5	9.2	12.00	5
45	188	1	1	30	2	142.50	51.66	90.84	135	21.4	9.4	62	14.1	33.8	27.2	53	15.3	97	65	241.6	2261	11.5	25.2	4.0	12.25	5
46	171	1	1	29	2	177.84	51.66	126.18	397	28.2	10.4	67	6.5	48.6	10.0	66	8.1	69	45	196.2	2110	4.8	9.5	10.1	14.00	4
47	191	1	1	19	2	145.92	57.81	88.11	308	32.4	9.2	70	3.7	51.6	12.3	68	15.8	55	53	208.7	2000	1.2	11.1	3.3	13.92	4
48	190	1	1	5	2	157.32	34.44	122.88	75	23.9	13.7	74	0.6	17.1	12.0	32	1.0	53	72	153.5	1240	2.4	40.8	0.4	3.00	2
49	196	1	1	21	3	228.00	73.03	154.97	215	13.3	9.2	36	4.8	15.7	4.7	20	7.1	61	16	111.8	744	25.1	28.9	2.8	6.00	3
50	174	1	1	20	3	94.62	73.03	21.59	59	12.1	6.5	36	0.8	17.0	5.3	27	4.5	42	10	130.3	3165	10.1	9.8	1.2	10.50	3
51	150	1	2	.	3	177.84	74.12	103.72	191	34.2	23.7	96	13.4	39.4	18.0	60	5.5	64	84	247.7	2971	21.0	7.4	1.9	12.25	4
52	148	1	2	.	3	115.14	70.85	44.29	679	88.6	65.4	246	31.8	124.9	25.3	163	36.8	175	102	423.5	10683	17.1	14.0	2.0	.	.
53	190	1	1	22	3	157.32	78.48	78.84	290	27.5	5.5	57	3.3	45.1	13.8	59	3.4	93	56	202.9	2816	3.6	0.6	4.6	11.50	5
54	186	1	1	3	3	116.28	59.95	56.33	156	13.9	9.0	43	0.2	19.1	12.6	38	6.0	26	55	139.8	1622	1.1	7.5	2.6	12.50	4
55	150	1	2	.	3	110.58	74.12	36.46	223	19.8	11.7	55	6.0	63.4	13.5	82	7.3	38	66	223.8	3184	0.0	3.7	10.8	14.00	4
56	187	1	1	22	3	126.54	78.48	48.06	376	33.0	10.5	78	13.2	41.8	31.9	74	11.3	55	93	193.7	5786	24.2	1.5	5.2	12.50	4
57	149	1	2	.	7	175.56	37.06	138.50	163	22.2	7.2	56	5.2	44.4	12.4	62	16.2	55	31	171.5	2310	39.8	17.5	11.0	10.50	5
58	141	1	2	.	7	140.22	40.33	99.89	289	27.9	13.3	80	6.3	40.6	28.6	70	16.1	111	83	297.7	3838	1.4	1.7	5.8	12.00	6
59	144	1	2	.	8	161.88	34.88	127.00	127	18.6	6.6	45	5.2	36.9	11.5	50	4.1	81	50	175.5	1480	1.0	24.1	7.3	11.50	4
60	168	1	1	6	8	172.14	56.68	115.46	586	45.8	15.2	111	15.7	64.8	14.3	80	18.4	77	58	253.8	3542	7.1	1.0	4.1	12.25	5
63	141	1	2	.	7	165.30	55.59	109.71	260	23.0	18.4	73	3.4	30.2	8.8	42	12.5	30	32	112.5	1686	34.3	20.1	12.8	12.25	4
64	168	1	1	13	7	103.74	52.32	51.42	131	19.2	19.6	75	12.6	28.3	8.6	49	3.7	91	30	201.7	2268	1.0	3.1	0.0	11.83	3
65	142	1	2	.	7	148.20	55.59	92.61	220	48.4	18.1	130	35.3	60.8	26.7	104	15.8	139	87	373.6	6078	7.6	3.9	0.0	11.75	3
66	150	1	1	27	7	153.90	39.24	114.66	115	17.7	6.9	44	6.7	30.8	10.7	44	9.4	70	34	224.8	1540	1.7	4.2	0.0	13.50	3
67	173	1	1	.	7	135.66	41.42	94.24	172	24.9	7.3	65	11.5	27.1	15.1	45	16.1	80	77	220.6	1384	3.6	4.9	1.0	14.25	6
68	174	1	1	25	7	142.50	55.59	86.91	174	22.8	11.9	69	18.2	34.8	16.9	56	5.9	50	76	228.4	2938	1.1	0.0	0.0	12.75	3
69	148	1	1	18	7	133.38	35.97	97.41	170	27.2	6.8	56	6.0	39.9	8.2	50	2.1	65	25	152.1	1194	0.8	3.8	1.0	11.50	4
70	149	1	.	.	.	.	.	.	129	21.8	8.1	69	8.8	32.8	11.2	46	4.3	37	62	124.5	1343	.	.	.	9.50	4
71	143	1	2	.	7	165.30	54.50	110.80	323	42.3	31.3	135	14.0	48.4	20.4	71	17.2	170	86	335.9	3861	21.2	8.4	5.0	14.00	5
72	198	1	1	.	7	129.96	56.68	73.28	167	8.1	4.2	25	7.3	10.3	3.5	17	4.6	8	15	48.2	1317	15.2	1.0	14.0	12.00	3
73	142	2	.	.	8	.	.	.	183	32.2	8.3	65	2.6	42.5	9.5	54	8.7	98	28	180.6	2806	.	.	.	6.67	2
74	141	2	1	25	7	161.88	74.12	87.76	250	34.0	39.8	127	30.9	34.9	12.8	49	12.7	33	45	146.7	4724	14.5	2.2	0.0	11.50	4
75	197	1	1	17	8	166.44	56.68	109.76	151	20.4	9.7	53	1.1	39.0	11.0	60	10.8	68	20	196.2	1960	16.2	12.3	7.0	11.25	3
76	147	1	2	.	7	171.00	56.68	114.32	247	37.6	14.2	96	17.0	44.6	18.9	71	12.1	129	65	334.7	3013	3.2	0.6	0.0	12.25	4
77	166	1	1	10	7	132.24	42.51	89.73	97	12.1	4.4	32	1.9	26.6	2.9	42	0.2	42	9	135.4	1592	15.0	3.6	0.1	14.00	4
78	192	1	1	19	8	184.68	54.50	130.18	372	19.8	14.8	64	13.8	46.2	9.6	60	2.7	32	39	95.4	1859	12.4	2.6	1.6	10.75	3
79	144	1	2	.	8	184.68	51.23	133.45	131	21.6	24.7	76	15.3	24.9	19.5	45	4.1	90	99	256.0	2923	2.6	9.6	1.2	10.50	4
80	142	1	2	.	7	189.24	47.96	141.28	310	25.7	13.0	67	19.0	38.8	12.3	57	28.3	71	52	230.5	2001	0.3	2.4	0.0	12.00	4
81	163	2	1	6	4	133.38	65.40	67.98	266	35.1	17.4	97	10.7	54.1	12.8	72	1.1	42	21	158.4	3474	3.2	3.0	0.0	8.33	4
82	170	2	1	26	4	143.64	70.85	72.79	199	30.4	8.8	98	18.6	28.5	6.9	54	5.0	73	42	219.0	1920	3.3	0.7	0.0	0.03	6
83	171	1	1	20	4	143.64	66.49	77.15	166	36.9	10.8	77	0.1	45.3	21.8	72	4.1	90	79	278.4	2404	21.8	2.5	0.7	14.00	4
84	145	1	2	.	4	121.98	82.84	39.14	671	74.3	37.2	225	47.4	126.2	52.5	184	13.8	77	160	364.5	9706	0.5	4.0	0.6	3.50	0
85	157	2	1	3	4	142.50	67.58	74.92	254	30.8	25.8	97	16.0	52.1	11.3	69	29.9	161	54	310.0	3771	29.0	18.9	0.4	8.83	5
86	170	2	1	8	4	144.78	78.48	66.30	239	32.3	16.1	90	12.9	48.6	11.3	67	41.9	120	54	318.2	3533	27.7	28.2	0.5	14.58	5
87	168	1	1	14	4	157.32	65.40	91.92	165	25.5	11.0	67	1.6	34.2	9.2	48	7.9	64	47	205.8	1763	19.6	3.7	0.2	14.25	4
88	171	1	2	.	4	172.14	78.48	93.66	418	35.3	16.3	93	1.9	29.8	10.2	45	21.7	75	48	242.9	1508	15.0	2.8	0.0	16.00	7
89	171	1	2	.	4	145.92	91.56	54.36	270	25.9	11.6	81	11.1	49.5	11.8	68	25.4	110	42	257.2	4053	19.6	2.3	0.0	11.25	10

SUBJ	AGE	RACE	MENS	DCYC	DAY	SCHOL	HDLC	LDLC	CHOL	SFA	PUFA	FAT	N31	N6	N7	PRO	N50	SUCR	STAR	N45	NA	LEX	MEX	VEX	SPAN	FREQ
90	140	2	2	.	4	.	.	.	215	18.8	10.3	48	1.1	31.9	6.3	43	1.6	136	37	111.1	3079	7.3	2.3	1.0	11.00	3
91	144	2	2	.	4	144.78	75.21	69.57	327	39.2	17.0	109	27.5	64.5	29.8	99	10.1	58	150	307.7	5984	3.8	36.9	17.3	12.50	4
92	168	2	1	9	4	175.56	79.57	95.99	356	52.2	55.2	187	32.1	37.1	20.4	66	12.3	79	117	438.3	3062	4.4	10.8	2.5	7.00	6
93	142	1	1	.	4	92.34	69.76	22.58	197	19.3	11.9	78	13.8	37.4	18.3	78	39.5	110	35	289.3	2672	22.2	6.7	0.0	9.33	4
94	140	1	1	6	4	204.06	59.95	144.11	504	27.0	13.5	74	22.5	36.2	18.2	58	4.3	58	86	235.1	2821	19.9	4.3	3.2	14.00	4
95	146	1	2	.	4	157.32	68.67	88.65	177	18.6	13.2	65	23.0	32.9	15.4	53	3.1	40	53	162.0	1764	44.5	14.4	4.1	4.83	3
96	149	2	1	4	4	156.18	64.31	91.87	161	35.5	14.4	112	16.9	37.7	22.3	68	24.0	45	114	307.3	4086	7.2	1.0	3.5	13.58	6
97	151	2	1	3	4	110.58	73.03	37.55	196	13.5	7.9	40	5.5	33.5	13.1	47	12.2	13	50	103.6	1966	26.1	28.6	0.0	12.25	3
98	146	1	2	.	4	125.40	57.77	67.63	324	50.2	18.5	122	25.8	73.6	19.1	96	2.3	71	109	333.0	3464	9.7	1.6	0.0	12.00	5
99	170	2	1	23	4	141.36	68.67	72.69	182	24.6	18.3	74	14.0	32.5	15.0	51	4.0	73	59	200.4	2064	21.0	22.3	5.1	11.17	3
100	140	1	2	.	5	142.50	57.77	84.73	466	43.8	23.7	125	52.1	62.7	30.0	93	37.3	118	124	403.0	4987	8.1	6.8	11.6	12.33	5
101	170	1	1	5	5	189.24	66.49	122.75	373	29.8	23.5	87	16.6	42.2	19.8	62	21.3	50	82	201.0	4094	15.8	6.8	7.7	12.33	5
102	151	1	2	.	5	191.52	68.67	122.85	227	39.8	11.3	86	6.0	65.4	16.1	85	20.3	59	46	214.0	2503	25.1	3.2	2.2	12.25	4
103	144	1	2	.	5	166.44	55.59	110.85	504	38.3	18.0	101	15.8	52.2	11.5	70	4.1	74	44	206.2	4013	20.8	6.2	0.1	7.92	4
104	168	2	1	.	5	183.54	67.58	115.96	240	34.9	12.9	96	7.4	60.2	13.3	80	20.4	77	41	244.8	3954	22.9	31.3	0.0	11.25	5
105	150	1	2	.	5	149.34	58.86	90.48	289	54.4	22.7	137	10.3	80.4	27.3	113	8.4	68	67	219.5	5028	24.5	0.1	1.4	12.25	3
106	165	1	1	18	5	177.84	77.39	100.45	159	15.1	8.6	45	5.2	35.0	11.1	50	3.0	41	25	122.4	2325	5.8	24.1	0.0	7.50	5
107	150	2	2	.	5	161.88	63.22	98.66	222	38.9	16.8	105	7.1	34.1	23.4	66	14.9	138	58	320.8	4239	11.7	4.3	7.5	14.00	5
108	144	1	2	.	5	157.32	68.67	88.65	50	11.4	5.3	33	19.9	12.8	5.9	19	3.4	54	30	120.6	4187	33.6	14.8	11.6	.	.
109	149	1	2	.	5	156.18	52.32	103.86	214	29.5	17.9	82	10.3	37.2	19.2	61	3.7	109	95	277.1	2982	23.2	15.3	0.3	4.00	8
110	168	1	1	26	5	135.66	56.68	78.98	287	27.1	14.0	78	16.4	58.2	19.7	79	24.6	122	84	313.2	2859	3.5	11.0	9.8	14.67	6
111	164	2	1	11	5	160.74	63.22	97.52	197	35.2	26.2	101	38.3	41.6	15.5	62	9.8	131	88	336.1	2934	35.2	18.2	8.0	8.83	4
112	171	1	1	21	5	175.56	76.30	99.26	212	26.2	14.2	89	13.1	29.8	12.3	66	5.5	105	52	242.5	3371	37.4	15.1	2.4	8.00	4
113	168	2	1	5	5	131.10	56.68	74.42	257	31.5	13.3	107	8.5	31.3	14.1	71	8.7	112	47	246.7	3151	15.3	16.2	5.3	9.50	4
114	151	1	2	.	5	127.68	62.13	65.55	253	30.3	22.6	88	7.7	25.6	11.3	44	30.6	105	34	252.7	1746	13.3	1.0	0.0	10.50	3
115	149	1	2	.	5	136.80	70.85	65.95	169	23.0	9.9	58	5.8	40.1	11.7	61	33.0	123	61	309.9	2911	10.6	1.6	3.5	14.25	6
117	146	1	2	.	5	139.08	67.58	71.50	151	22.8	21.5	88	32.9	44.8	15.6	66	11.6	75	56	242.0	3021	16.0	0.7	0.0	11.00	4
118	153	2	1	7	5	126.54	61.04	65.50	133	20.0	10.4	54	11.5	43.6	10.0	57	2.7	42	49	151.0	2628	0.5	13.4	0.0	.	.
119	144	1	2	.	6	141.36	51.23	90.13	316	22.1	17.1	72	10.7	69.9	18.7	91	6.9	62	72	218.5	2208	0.8	4.7	0.2	9.75	5
120	193	2	1	18	6	139.08	42.51	96.57	177	27.5	18.1	85	11.1	31.0	18.3	58	16.1	83	94	276.8	2933	0.0	1.0	17.5	16.25	6
121	190	2	1	.	8	216.60	83.93	132.67	525	37.6	9.4	83	4.3	48.5	4.8	60	3.6	73	19	164.6	2603	10.6	3.2	3.9	12.00	5
142	196	2	1	29	6	137.94	47.96	89.98	275	28.0	14.6	76	13.7	46.9	13.8	69	21.3	50	50	190.1	4174	0.8	8.3	0.0	9.25	4
143	162	2	1	14	6	160.74	78.48	82.26	82	15.8	4.4	51	0.7	27.6	1.6	39	1.1	74	4	172.0	1560	12.6	6.8	2.7	10.50	6
144	180	2	1	10	6	129.96	62.13	67.83	264	25.1	13.8	84	22.5	35.4	10.9	55	12.8	148	43	314.6	3444	49.5	33.9	0.0	12.50	6
145	158	2	1	.	6	190.38	35.97	154.41	347	30.2	17.2	104	9.2	41.3	5.1	74	10.8	55	20	242.0	2838	0.3	26.3	1.9	12.50	5
146	166	2	1	7	6	155.04	38.15	116.89	450	24.9	19.6	84	31.9	51.9	13.2	70	21.8	66	60	249.9	2599	0.0	1.4	9.1	11.50	3
147	169	1	2	.	6	129.96	56.68	73.28	172	26.4	10.4	65	6.1	42.0	9.3	56	9.3	44	34	154.0	1934	0.0	3.0	16.1	11.25	3
148	175	2	1	5	6	124.26	57.77	66.49	174	11.2	4.6	35	11.4	11.5	7.9	30	12.7	40	26	160.0	1445	3.8	1.2	0.0	5.75	2
149	167	2	1	20	6	155.04	73.03	82.01	219	44.6	22.6	123	6.1	46.1	14.8	63	0.9	34	60	193.0	1945	5.1	10.1	0.0	10.00	4
150	169	1	1	16	6	160.74	37.06	123.68	306	28.0	11.3	78	7.9	35.5	10.2	54	11.3	134	39	293.8	2411	0.1	10.2	0.0	13.00	6
151	170	1	1	11	6	159.60	57.77	101.83	494	27.3	12.5	76	20.6	56.2	15.0	71	23.3	65	53	202.1	2837	2.1	1.9	0.0	17.50	7
152	172	2	1	19	6	181.26	66.49	114.77	79	7.1	9.4	31	2.9	11.8	6.4	25	12.4	21	25	95.0	1071	16.9	7.1	0.0	11.33	3
153	177	2	1	8	6	180.12	78.48	101.64	369	49.4	37.5	151	36.1	58.7	20.9	84	4.4	105	51	244.5	3568	1.0	5.3	1.9	13.00	5

SUBJ	AGE	RACE	MENS	DCYC	DAY	SCHOL	HDLC	LDLC	CHOL	SFA	PUFA	FAT	N31	N6	N7	PRO	N50	SUCR	STAR	N45	NA	LEX	MEX	VEX	SPAN	FREQ
154	173	2	1	25	6	173.28	46.87	126.41	80	17.2	10.7	51	8.0	15.9	12.4	31	4.8	42	55	163.3	1667	2.5	6.6	0.0	11.00	4
155	189	2	1	9	6	169.86	71.94	97.92	318	43.5	18.3	119	12.2	79.9	13.8	107	5.3	74	55	249.5	5291	1.4	3.3	6.0	11.50	5
156	173	2	1	.	6	143.64	64.31	79.33	436	36.7	8.5	90	4.3	59.6	7.0	72	2.5	84	16	204.5	3081	1.9	1.5	0.0	14.50	9
157	175	2	1	.	6	176.70	83.93	92.77	151	21.5	19.4	71	1.7	31.0	8.1	41	11.3	45	33	134.6	2626	0.0	9.2	0.6	6.00	2
158	171	1	1	10	6	173.28	56.68	116.60	144	26.3	12.9	71	13.3	35.5	20.6	62	9.2	39	102	250.8	3292	0.4	1.7	0.6	14.58	5
159	192	2	1	.	6	192.66	80.66	112.00	174	25.5	25.3	80	5.7	42.2	11.7	60	4.4	49	45	200.9	2466	0.1	8.6	1.8	14.00	5
161	143	1	2	.	7	167.58	64.31	103.27	254	12.2	6.2	36	6.2	37.1	10.7	50	9.9	33	39	152.7	1713	6.6	5.9	5.2	12.50	4
162	191	1	1	9	8	164.16	43.60	120.56	155	24.6	7.0	60	7.4	33.5	16.0	50	6.8	83	60	207.0	2508	21.0	17.6	7.8	15.25	5
163	146	1	1	8	7	127.68	42.51	85.17	198	32.2	14.4	86	12.1	47.5	16.5	66	7.9	77	56	197.5	2005	2.7	8.9	9.9	12.50	4
164	150	1	.	.	8	.	.	.	118	18.0	5.8	51	6.5	21.6	6.3	32	15.9	78	26	173.7	1718	.	.	.	15.00	7
165	.	1	.	.	.	.	.	.	265	32.6	13.3	86	16.9	51.4	11.2	67	4.5	65	60	266.2	2254	.	.	.	13.25	5
166	156	1	1	.	8	175.56	52.32	123.24	237	41.0	18.0	117	47.7	48.2	17.5	72	2.9	102	100	266.4	3968	5.4	29.0	0.4	11.50	4
167	166	1	1	19	8	165.30	53.41	111.89	254	35.4	26.6	114	16.8	51.1	18.4	70	9.9	60	57	182.6	3113	10.5	19.2	7.0	10.50	7
168	140	1	1	14	8	164.16	26.16	138.00	489	30.4	19.3	92	6.2	59.4	14.1	80	14.1	105	60	239.0	2520	6.1	2.0	0.0	13.25	5
169	172	1	1	21	8	171.00	51.23	119.77	233	35.1	25.4	100	13.3	42.3	20.2	64	11.3	105	94	307.7	4783	0.6	16.1	3.8	9.00	6
170	168	1	1	35	8	200.64	65.40	135.24	268	32.3	11.4	88	20.4	66.4	13.1	83	6.1	13	62	128.7	3118	6.0	8.9	0.0	5.00	2
171	194	1	1	20	8	149.34	51.23	98.11	319	25.8	11.1	87	6.8	35.2	6.0	63	8.3	72	22	206.4	2332	14.7	6.0	3.8	15.50	6
172	178	1	1	13	8	161.88	30.52	131.36	88	23.4	10.2	68	6.7	22.2	11.9	41	6.3	55	41	186.5	1391	2.0	14.3	1.2	11.50	5
173	190	1	1	34	8	165.30	35.97	129.33	127	22.2	7.1	51	5.2	36.8	14.6	53	2.9	59	73	164.4	3273	21.5	9.4	1.4	11.50	2
174	191	1	1	14	8	199.50	61.04	138.46	290	18.0	8.1	58	12.8	25.0	10.3	38	8.5	42	45	135.8	1711	14.4	1.8	0.1	8.50	4

Subj = Subject
Race: 1 = White 2 = Black 3 = Other
Mens: 1 = Post-menarche 2 = Pre-menarche
DCYC = Day of menstrual cycle
SCHOL = Total Plasma Cholesterol (mg/dl)
HDLC = High-Density Lipoprotein Cholesterol (mg/dl)
LDLC = Low-Density Lipoprotein Cholesterol + Very-Low-Density Lipoprotein Cholesterol (mg/dl)
CHOL = Dietary Cholesterol (g)
SFA = Saturated Fat (g)
PUFA = Polyunsaturated Fat (g)
FAT = Total Fat (g)
N31 = Hydrogenated Fat (g)

N6 = Animal Protein (g)
N7 = Vegetable Protein (g)
Pro = Total Protein (g)
N50 = Free Glucose (g)
Sucr = Sucrose (g)
Star = Starch (g)
N45 = Total Carbohydrate (g)
Na = Sodium (mg)
Lex = Light Physical Activity (hr/wk)
Mex = Moderate Physical Activity (hr/wk)
Vex = Vigorous Activity (hr/wk)
Span = Eating Span
Freq = Eating Frequency

VITA 2

Cynthia Shaw Pitts

Candidate for the Degree of

Master of Science

Thesis: LIPOPROTEIN AND TOTAL PLASMA CHOLESTEROL: INFLUENCE OF AGE, RACE, DIET AND EXERCISE IN 150 ADOLESCENT FEMALES

Major Field: Food, Nutrition and Institution Administration

Biographical:

Personal Data: Born in Salt Lake City, Utah, March 4, 1954, the daughter of Dr. and Mrs. Manford A. Shaw, the wife of Dr. Randolph J. Pitts, the mother of Nicholas J. Pitts.

Education: Graduated from Highland High School, Salt Lake City, Utah, 1972; received Bachelor of Arts degree in German from Willamette University, Salem, Oregon, in May, 1976; completed requirements for the Master of Science degree at Oklahoma State University in May, 1983; concurrently enrolled in Master of Science degree program in Biochemistry.

Honors: Phi Kappa Phi, Phi Lambda Upsilon.