COMPARISONS OF SERUM LIPID LEVELS AND DIETARY LIPID INTAKES OF PARENTS AND CHILDREN

by

Ai-Leng Ng

Thesis submitted to the Faculty of the
Virginia Polytechnic Institute and State University
in partial fulfilment of the requirements for the degree of

MASTER OF SCIENCE

in

Human Nutrition and Foods

APPROVED:

Mary Ann Novascone
M. A. Novascone, Chairman

Mary K. Korslund
M. K. Korslund

G. E. Bunce
G. E. Bunce

May, 1988

Blacksburg, Virginia

COMPARISONS OF SERUM LIPID LEVELS AND DIETARY LIPID INTAKES OF PARENTS AND CHILDREN

By

Ai-Leng Ng

Committee Chairperson: Mary Ann Novascone
Human Nutrition and Foods

(ABSTRACT)

Fifty-seven subjects from 14 families participated in a study designed to investigate similarities and differences between parents and children residing with them relative to their serum lipid levels and dietary lipid intakes. To participate, at least one of the parents needed to have a serum total cholesterol of at least 240 mg/dL.

Fasting blood samples obtained from the participants were analysed for serum total cholesterol, HDL-C, LDL-C, and VLDL-C levels. Anthropometric measurements and blood pressure readings also were taken. Dietary records, questionaires on lifestyle, health habits, health history, and nutrition knowledge were completed by the participants.

Correlation coefficients between serum total cholesterol and dietary cholesterol intakes of the fathers were 0.66 ($p = 0.01$) in all 14 families and 0.64 ($p = 0.05$) in the 11 families in which at least one parent had a family history of CHD. The values of the correlation coefficients of HDL-C and the intake of dietary cholesterol of the children for the 14 families

and the 11 families were -0.36 ($p = 0.07$) and -0.55 ($p = 0.01$) respectively. A significant correlation was found between the dietary pattern of the parents and that of their children. The following correlation coefficients were found for the five families in which both parents had a fmaily history of CHD: 0.65 ($p = 0.02$) for total fat, 0.79 ($p = 0.002$) for saturated fat , and 0.59 ($p = 0.04$) for cholesterol.

ACKNOWLEDGEMENTS

The author wishes to express her deepest gratitude and most sincere appreciation to the following individuals:

Dr. Mary Ann Novascone for her expertise and experienced guidance and helpfulness as my graduate advisor. Her influence has been invaluable throughout the entire course of my graduate career, and, particularly, the completion of my research thesis. Her understanding and sensitivity also are deeply appreciated and remembered.

Dr. G. Edwin Bunce and Dr. Mary K. Korslund, for sharing their expertise and wisdom throughout the preparation of this thesis.

Dr. William Herbert, who permitted me to recruit subjects from the Cardiac Therapy and Intervention Program and who was himself a volunteer in the study. His cooperation and immense assistance throughout the recruiting process is truly appreciated.

Ellen Coale, for her guidance and tremendous help in the recruiting process, and her generosity in sharing her expertise.

Barbara Chrisley, for her assistance in the collection of blood samples; and Lisa Haedrich for all her help, moral support, and fun at lunch hours.

The parents and children who volunteered to participate in this study. Without their cooperation and dedication, this thesis would never have materialized. I am deeply indebted to them.

My family, for their love, understanding, and support.

Finally, my most caring and understanding husband, Chu-Ngi Ho, who provided me with support and confidence at times when I needed them most. He has always been there for me.

TABLE OF CONTENTS

TABLE OF CONTENTS
(continue)

TABLE OF CONTENTS
(continue)

LIST OF TABLES

LIST OF TABLES
(continue)

LIST OF FIGURES

CHAPTER I

INTRODUCTION

Although the rate of cardiovascular mortality has been on the decline for the past 15 years, coronary heart disease (CHD) remains responsible for about one in every three deaths in the United states (1). Nationally, CHD costs about 60 billion dollars a year in health care, lost wages, and productivity (2). This enormous toll has focused attention on identifying causal factors of CHD as a possible approach to retarding the epidemic of this public health problem.

Numerous experimental, clinical, and epidemiological studies have related genetic, environmental, and nutritional factors to the genesis of CHD. These observations have resulted in the identification of three primary risk factors most intimately associated with CHD: elevated serum cholesterol, hypertension, and smoking. Modifications of these risk factors in adults have been shown to play a very important role in the alteration and retardation of the atherosclerotic process which is the major etiologic cause of CHD (3-5). This process is thought to originate in childhood, progress through young adulthood, and manifest itself at middle age or later (6-9). Population studies have indicated that children in countries with a high incidence of CHD generally exhibit higher plasma total cholesterol levels than their

counterparts from countries with a low incidence of CHD (10-12). Other researchers (13,14) found that both abnormalities in plasma lipids and lipoproteins, and the clinical consequences of these abnormalities also tend to cluster in families.

To reduce familial related CHD, greater efforts are now being made to identify risk factors for CHD among these high risk children (15,16). Although the effect of dietary modifications in children relative to the incidence of CHD in later life has not been demonstrated definitively, evidence supporting the merit of lowering plasma cholesterol levels is strong (2-4). Introduction of preventive measures at this stage in life seems appropriate since lifestyle and health habits associated with these risk factors begin in childhood and may persist into adulthood. Thus the intervention steps taken at this stage may help to deter the underlying etiologic process of CHD and help reduce the risk of developing athrosclerosis in adulthood. According to the American Heart Association (AHA), modifiable risk factors include smoking, sedentary lifestyle, and those risk factors that have strong dietary determinants such as elevated plasma total cholesterol, obesity, and high blood pressure (17).

Although studies have shown that children's plasma lipid profiles tend to resemble those of their parents

(13,14), no study has been conducted to determine whether these children who are residing with their parents exhibit eating habits similar to those of their parents. Thus the role of these acquired dietary habits relative to the similarity between blood lipid profiles of parents and children is unclear. Hence additional information regarding family dietary habits and their influence on the children would provide a useful basis in preventive measures, especially in a high risk pediatric population with family history of CHD. This research project was undertaken in light of this perceived need.

The specific objectives of this study include the following:

1) to assess whether there is an association between intakes of dietary lipids and the serum lipid profiles in the sample studies;
2) to identify if intrafamilial correlations exist betweeen the serum lipid profiles of the parents and the children in families in which either one or both of the parents has a family history of CHD:
3) to compare the dietary pattern of the parents with those of their children residing with them.

From these analyses, one should be able to devise strategies to halt the progress of CHD in the offspring of families which are considered at risk. Early inter-

vention should be aimed at promoting dietary changes that favorably influence serum cholesterol and blood pressure levels, discourage smoking, and encourage physical activity to reduce or prevent overweight. Understanding the impact of dietary habits on the clinical status of children would be of great interest in the early prevention of CHD because dietary habits can play an important role in risk factor prevention, particularly among high-risk children.

CHAPTER II

REVIEW OF LITERATURE

Familial Aggregation of Risk Factors for CHD

The familial occurence of CHD has been well documented in several studies (18-24). This phenomenon is attributed, in part, to familial resemblance relative to major risk factors such as hyperlipidemia, hypertension, and obesity. Rissanen and Nikkila (21) investigated the aggregation of risk factors in families of men with fatal and non-fatal CHD. Their results indicated that familial hyperlipidemia was twice as prevalent and familial hypertension was three times as prevalent in case families than in control families. Also, the clustering of these risk factors was most impressive in families whose mother had died of CHD before age 70. Their rationale for this observation was based upon the maternal tendency to pass to her offspring a set of potent determinants of early-onset hypertension and hyperlipidemia. This phenomenon observed in the study is also in accord with another finding by Forde and Thelle (25) who reported similar results. Rissanen and Nikkila (21) also showed that the major risk factors for those who had CHD and whose mothers had died of CHD by age 70 were hyperlipidemia and hypertension. However, for individuals with CHD whose fathers died of CHD by age 70, they found that the above mentioned major risk factors

alone could not account for all the risks relating to this disease. These risks, it appears, also depended on other factors yet to be identified. Possible predisposing factors might include familial resemblance in the structure of coronary arteries, body build, and smoking habits or other shared environmental influences.

To identify the high-risk groups relative to familial CHD, Rissanen and Nikkila (26) later conducted another study targeting families with CHD. They reported that the risk for early onset of CHD was about three times greater for the sibs of patients who developed the disease before age 46 as opposed to the sibs of patients who developed the disease at age 46-55. The risk was even greater, up to ten-fold, for these sibs when either one of their grandparents also had died of CHD before age 70. On the contrary, the risk was much less for the sibs of middle-aged patients who did not have a family history of CHD. Thus, it appeared to these investigators that familial history of CHD was the best predictive feature in identifying individuals at high risk for premature CHD. The observation of this strong familial trait in the development of premature CHD was also reported in several other studies (27,28). This view, however, is not fully supported by other researchers whose studies have shown a weak association of family history to the risk of CHD (29,30).

The procedure in obtaining the information concerning familial history remains, perhaps, an open question. Any study of this nature has inherent difficulties in isolating definite causal factors for the genesis of CHD. Hence, it is difficult to differentiate genetic effects from shared environmental influences. Furthermore, response to environmental factors may be determined genetically. That is to say, a weak environmental effect may easily become a strong predisposing risk factor to a genetically susceptible phenotype (21,24).

Besides utilization of familial incidence of CHD as a means of identifying high risk individuals, considerable interest also has been directed toward examing the possible clustering of other risk factors within the families. Well documented risk factors associated with CHD which appear to cluster within families include serum total cholesterol, high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) (13,14,31-33).

Morrison and co-workers (34) focused their study on the nature and extent of intrafamilial lipoprotein ratio, HDL-C/LDL-C ratio. They reported a consistent, positive, and significant correlation ($r = 0.48$) in HDL-C between parents and children. However, mother-offspring correlation ($r = 0.58$) of this lipoprotein was higher than father-offspring correlation ($r = 0.46$). For LDL-C,

significant and positive correlation was seen between mothers and daughters ($r = 0.60$). When the data were categorized into parental and pediatric lipoprotein quartiles, 46% of the children of parents in the highest LDL-C quartiles were themselves in the highest LDL-C quartile when compared to the pediatric population. A similar observation also was seen for the lowest HDL-C quartile. Based on these observations, Morrison and co-workers (34) concluded that having a knowledge of parental LDL-C and HDL-C status could help identify children at risk by virtue of their levels of lipoproteins. Other researchers also had drawn similar conclusions when parent-child cholesterol levels, triglycerides, and ponderosity (obesity) were taken into consideration (19,35-37). Furthermore, researches (34,36,38) had found that the interaction between parental and progeny cholesterol levels might be age dependent. That is, parental cholesterol levels have greater effects on their younger progeny than the older sibs. This interaction may reflect the younger progeny's closer shared environment with their parents than their older sibs (34,36,38). These consistent findings have led to a general consensus that knowledge of parental risk factor status will improve sampling efficiency and permit closer supervision of children more likely to develop premature CHD.

Growth curve data released from the National Center for Health Statistics (39) indicated that children tend to maintain their height and weight percentile rank within their age-sex group with increasing age. With reference to this characteristic, researchers have raised questions as to whether or not risk factors observed in early childhood also continue into adulthood. Considerable evidence has indicated that serum total cholesterol, LDL-C, triglyceride, and blood pressure do indeed track (40-43).

The Effect of Physical Activity and Weight Loss On Lipoprotein Cholesterol

Evidence from epidemiological studies has indicated that a low level of plasma HDL-C is strongly associated with the risk of atherosclerotic heart disease (44-47). However, various, conditions such as decreased activity level and obesity, have been found to cause a decrease in the concentration of plasma HDL-C. Therefore, attempts have been made to evaluate the reversibility of the low HDL-C status. Observations by numerous researchers (48-50) have shown an increase in HDL-C in physically active individuals as compared to their sedentary counterparts. This has stimulated intensive research into the identification of the mechanism responsible for producing this increase in HDL-C during exercise. However, results from studies have been

conflicting. Researchers whose findings are similar may also come to different conclusions about the relationship between exercise and HDL-C (51-56). Argument has been centered on determining the confounding factor(s) responsible for raising the level of serum HDL-C.

William et al. (51) reported that the increase in HDL-C among the subjects during exercise treatment was the consequence of physical conditioning. This change did not occur until a threshold level of ten miles of jogging per week was maintained for at least nine months. In a later study, this group (52) further demonstrated that such an increase in HDL-C from weight loss during exercise was strongly associated with the increase in HDL-C. However, this study showed that the weight loss experienced by the control group did not result in a corresponding rise in HDL-C levels. William et al. (52) thus concluded that there was a difference in the physiological process as a result of exercise-induced weight loss and dieting weight loss. However, other workers (53-56) have reported significant increases in HDL-C from substantial weight loss through dieting. In contrast, Thompson et al. (57) found a decrease in HDL-C in weight loss observed in dieting subjects and Widhalm et al. indicated no change in HDL-C levels of dieting subjects (58). More studies are needed to better define the mechanism(s) responsible for causing such a change in

this lipoprotein metabolism.

Other investigators (59,60) whose findings are consistent with those of William et al. offer different rationales for the increase in serum HDL-C concentration. Their opinion is that the increase observed in HDL-C may not be the direct effect of the exercise itself. Rather, other health habits such as abstinence from smoking, consuming a carbohydrates-rich diet, and weight reduction that an individuals may have, also can cause an increase in the serum HDL-C.

Results from other findings, however, seem to indicate that weight loss and exercise were confounding yet independent factors which contributed to the increase in serum HDL-C (61,62). Studies conducted to observe the pattern of weight loss in relation to HDL-C levels among obese subjects indicated that a favorable increase in a plasma lipoprotein pattern appeared to take place only after remarkable and stable weight loss have been achieved in obese individuals (55,63).

The fact that HDL-C can be produced experimentally in vitro from very low density lipoprotein cholesterol (VLDL-C) and triglyceride (TG) in the presence of lipoprotein lipase (LPL) (64,65) has stimulated researchers to explore the relationship of this enzyme activity, exercise, and weight loss. Findings from studies (66-69) indicated that increased activity of LPL

was positively correlated with the elevation of HDL-C and negatively correlated to the level of TG. The elevation of HDL-C was thought to be the consequence of increased catabolism of TG by LPL in which part of the cholesterol, phospholipids, and apoproteins were transferred to HDL-C. Since LPL was the major factor in determining the rate of the catabolism of the circulating TG, the increased LPL activity could account for the lower serum TG levels seen in well-trained individuals (68). Such response in increased LPL activity was thought to be an adaptive phenomenon aimed at increasing the capacity of the body to efficiently mobilize and utilize fat needed during endurance exercise. These studies found that the increased LPL activity often was seen in skeletal muscle tissue and adipocytes (67,68). This metabolic pathway seemed to be operating during weight reduction via diet-restriction.

However, this pattern of HDL-C increase during exercise is not compatible with other studies which found that intense exercise did not seem to cause any change in the level of HDL-C(70-72). It is possible that the amount and the duration of exercise may be insufficient to stimulate efficiently the metabolic pathway (LPL activity) responsible for enhancing HDL-C production.

The Association of Smoking to Selected Risk Factors for CHD

Although the association between cigarette smoking and cardiovascular disease generally has been accepted, the underlying mechanism responsible for this relationship is poorly understood. Stamford et al. postulated that cigarette smoking may influence risk factors that are closely related to the disease (73). Risk factors that have been observed to be most often related to cigarette smoking are lower HDL-C and higher serum total cholesterol (73-81). The majority of this information has been obtained through cross-sectional epidemiological studies. Attempts have been made to rationalize the disparity in the levels of serum total cholesterol and HDL-C between smokers and non-smokers . However, mechanisms that are responsible for the differences exhibited in smokers and non-smokers remain speculative.

Stamford et al. (73) had shown that highly active non-smokers had an increased HDL-C, whereas the highly active smokers did not. In fact, they found that the levels of HDL-C in highly active smokers were no different from inactive non-smokers. Hence, cigarette smoking appeared to reduce the effect of chronic exercise on HDL-C levels. It was possible that cigarette smoking had induced an alteration of lipoprotein metabolism. On the other hand, Rabkin (74) observed a significant

increase in HDL-C during cigarette smoking cessation despite the weight gain often observed.

Hjermann et al. (76) reported a significant positive correlation between serum total cholesterol and the amount of cigarette smoking exposure. Their results revealed individuals with an increasing daily exposure to cigarette smoke in the following order: never-cigarette smokers, ex-smokers, present-non-inhaling smokers, present-inhaling smokers and present-non-filter smokers. This order was paralleled with an increasing level of serum total cholesterol. Hjermann and co-workers did not offer an explanation for the above observation, but did recommend that dietary intervention would be a more effective approach than smoking reduction to lower serum total cholesterol.

The percent of weight gain often associated with smoking reduction is supported by considerable, consistent data (74-78). One report showed that obese quitters tended to gain more weight than non-obese quitters (80). Also, the weight gain associated with cessation of smoking appeared to be permanent. That is, the "quit" cohorts remained heavier than cohorts who continued to smoke for at least 25 years after cessation (78). Explanations for this weight gain are conflicting. The fact that smokers were lighter in body weight and leaner than non-smokers even though smokers consumed

12.6% more calories than non-smokers could be due to the effect of nicotine to reduce the efficiency of the body to store excess calories and to increase metabolic rate in smokers, thus accounting for lower body weight among smokers and weight gain after cessation (73). Others suggested that the lower body weight was due to the effect of cigarette smoking on decreased palatability; hence smokers tended to consume fewer calories than non-smokers (79).

Findings regarding the effect of smoking cessation on blood pressure are rather inconsistent. Gordon et al. (80) reported an increase in blood pressure among quitters, whereas Greene (81) found no change in blood pressure whether an individual continue to smoke or quit smoking. In general, smokers appeared to have lower blood pressure than non-smokers (75,82,83).

The Effect of Diet on Serum Lipids and Lipoproteins

A highly significant association between elevated serum total cholesterol and incidences of CHD has served as a stimulus for several metabolic studies in an attempt to examine a possible relationship between dietary habits with this risk factor. Investigations to link this relationship range from absorption capacity of cholesterol to manipulation of the different levels of cholesterol intake, ingestion of dietary fats, or a

combination of both. These dietary components received the greatest attention by investigators (84-101) in an effort to lower serum total cholesterol.

The extent to which cholesterol can be absorbed by humans, however, has been the subject of debate. Kaplan and Wilson (85) suggested that the limitation in absorptive capacity in human would help maintain the body cholesterol within a narrow range despite the ingestion of a cholesterol-rich diet. Apparently, factors such as micellar solubilization of cholesterol and the degree of saturation of the mucosa also restrict the amount of cholesterol absorbed (85,86). Others found that large quantities of dietary cholesterol can be absorbed into the human system (87-90). Connor and Mattson (90,91) observed that, in general, an increase of 200 to 1000 mg of cholesterol per day would produce a 15% to 30% rise in plasma cholesterol. However, Quintao et al. (92) noted that the increment in the concentration of plasma cholesterol rarely exceeded 20% despite the quantity of intake and absorption. According to these authors, this small variation in cholesterol pools could be explained by the operation of two compensatory mechanisms: 1) increased re-excretion of cholesterol through the biliary tract, as reflected by an enhanced fecal excretion of neutral steroids which was endogenous in origin, and 2) decreased synthesis of cholesterol.

Furthermore, these mechanisms might not operate at the same degree in some individuals, thus explaining the different amounts of cholesterol accumulation in different individuals. Also, the accumulation did not necessarily reflect in the increased plasma cholesterol level. On the other hand, other workers had reported that cholesterol loading could lead to an increase in LDL-C (93). It appears that the expansion of the cholesterol pools as a consequence of increased absorption of dietary cholesterol is yet to be determined.

Brown and Goldstein (94) stated that most of the atherosclerosis seen in the general population is attributed to alarmingly high level of LDL-C resulted from a failure to produce enough LDL receptors. This deficiency may be due to subtle genetic and environmental factors that limit the manufacturing of recptors even in people without familial hypercholesterolemia. Environmental factor that has been identified to play this role is the high intake of dietary cholesterol and saturated fats derived from animal sources. To understand the role that LDL receptors play in clearing the level of circulating LDL-C in the blood stream, Brown and Goldstein (94) indicated that the receptors bind LDL particles and extract them from the surrounding fluid of the cells. The LDL is then taken into cells and degraded to yield the cholesterol for the needs of the cells.

These receptors thus help to remove the circulating LDL-C from the blood. The number of receptors produced varies with the cell's need for cholesterol. This protects the cells from cholesterol loading. However, this regulatory mechanism initiates the process of atherosclerosis because it results in the elevation of circulating LDL-C. This process seems to be accentuated by high intake of dietary cholesterol.

Besides cholesterol, different types of fat in the diet are thought to have a considerable influence on the plasma lipids. Packard et al. (93) reported an elevation of plasma lipids in normolipidemic individuals as a consequence of an increased dietary cholesterol content and decreased dietary polyunsaturated/saturated fat (P/S) ratio. This elevation in plasma lipids was attributed to an increase in cholesterol in VLDL-C (59.7%), LDL-C (15.0%) and HDL-C (29.9%). The increase in the level of HDL-C was thought to be associated with elevated apoprotein A-1. Conversely, diets with low cholesterol content and high P/S ratios would lead to the reduction in the levels of serum total cholesterol, LDL-C, and HDL-C. However, no significant change was seen in the LDL-C:HDL-C ratio (95). Study by Thuesen et al. also showed reduction in serum total cholesterol and LDL-C, but HDL-C did not change significantly (96).

When comparing polyunsaturated fatty acids (PUFA)

from different dietary sources, it was shown that PUFA from marine sources, especially omega-3 fatty acids had greater hypocholesterolemic effects than those from vegetable sources (97-99). Furthermore, there were indications that PUFA could also exhibit hypotensive effects in individuals (100-102). Other studies (103-104) indicated that monounsaturated fatty acid (MUFA) appeared to be as effective in lowering plasma cholesterol as the diet that was low in fat and high in carbohydrates.

Relationship of Obesity and CHD

It had been observed that mortality and morbidity associated with CHD and stroke was relatively high among obese individuals (105-107). This observation had led researchers to study the association of CHD and obesity and to determine if obesity is an independent risk factor of CHD, or that it may be just a manifestation of underlying metabolic derangements which also caused an elevation of known risk factors such as high blood pressure and hyperlipidemia, for CHD (105-112).

Some researchers (108,109) attributed the cardiovascular mortality in the obese individuals to the elevation of common risk factors of CHD. This causal role of obesity was supported in parts by obseving that a reduction in weight led to a decrease in the level of

these risk factors (110). Thus these studies (105-110) suggested that obesity itself, was not a risk factor for CHD, rather it was associated with the metabolic derangements which predispose an individual to elevation of these risk factors and manifestation of obesity. However, Keys et al. (111) contended that such may not be entirely the case because they could not find an increased risk for mortality associated with CHD with increased body weight in the large and diverse sample that they studied for 15 years. Hubert et al. (112) in a 26-year follow up of participants in the Framingham Heart Study of 5209 adults supported the contention of Keys and his co-workers and came to the conclusion that obesity was an independent risk factor for cardiovascular death. By using an obesity index of the Metropolitan Relative Weight (MRW) calculated from the 1959 Metropolitan Life Insurance Company's desirable weight table, and applying regression analysis, they came to such a conclusion.

Burack et al. (113) attempted to determine if morbidity experienced by obese individuals may be partially due to the elevation of associated risk factors. In their study, they attempted to see if the baselines of blood pressure, glucose, and cholesterol were elevated prior to weight gain in 4015 participants. The reason for such an attempt being that if the baselines did increase, it would indicate that metabolic

predisposition was the cause of the elevation of these risk factors in obese individuals. They were not able to demonstrate an elevation in the baseline of these risk factors in association with increased level of fatness during a 15 year follow up of these individuals. They thus concluded that elevation of risk factors were the result of weight gain, rather than the manifestation of underlying metabolic derangement that caused the elevation and weight gain.

With these many studies (105-114) obesity had been shown to play an important role in development of CHD despite the debate over its precise mechanism and association. Better means of quantitative measurement of obesity need to be explored and more studies done to fully elucidate the complexity and heterogeneity of obesity associating to CHD.

CHAPTER III

METHODOLOGY

Recruitment of Subjects

Recruitment of the subjects was initiated after the study had been approved by the Institutional Review Board for Research Involving Human Subjects at Virginia Polytechnic Institute and State University. Subjects were recruited through the Cardiac Therapy and Intervention Center at Virginia Tech and advertisements in the campus and local newspapers. To be eligible for the study, either one of the parents in the family needed to fulfill all of the following requirements:

1) a serum total cholesterol level of greater than 240 mg/dl;
2) at least one progeny and spouse are residing in the same household and willing to participate in the study.

Individuals who met the above stated requirements were contacted either in person or by the telephone and a summary of the objectives and the experimental procedures were explained to them.

Data Collection Strategies

All participants were scheduled to attend a mandatory orientation meeting on a weekday during which written and verbal explanations of the study (Appendix A)

were given. The responsibilities of the participants were outlined clearly (Appendix B). Detailed instructions on keeping dietary records (Appendices C and D) also were presented in this meeting.

Toward the end of the meeting, all participants were given diet record sheets (Appendix E), general questionaires concerning family health history, smoking and drinking status (Appendix F), diet history (Appendix G), and level of physical activity (Appendix H). Finally consent forms (Appendix I) were signed by all the participants. After the distribution of these packets, all participants were scheduled for an appointment to return for blood sampling. Participants were contacted by telephone 24 hours prior to this appointment. Every family was instructed to fast for at least 12 hours. Light apparel also were recommended for ease and comfort during the sampling and measurements.

Upon arrival at the experimenter's laboratory, each participant was requested to sit quietly for 15 minutes before a measurement of their blood pressures was done. This was then followed by the determination of anthropometric measurements and the collection of fasting blood samples. About five milliliters of blood free from hemolysis was drawn via venipucture from each participant by a licensed medical technician. The blood samples were centrifuged and the separation of serum from cells was

completed within 45 minutes of venipuncture. The serum samples were then carefully transfered into clean transport tubes using disposable pipettes, properly packaged, and sent to Roche Biomedical Laboratory for analysis of serum total cholesterol, HDL-C, LDL-C, and VLDL-C (lipid profile analysis #033886, Roche Biomedical Lab, Inc.). At this appointment, the participants also returned all the questionaires and diet records they had completed. These were checked for completeness and clarity to ensure they were valid for the study. Testing of knowledge of the AHA dietary guidelines (Appendix J) also was administrated at this time. The test focused on 1) the recommended intakes of total fat, saturated fat, PUFA, and MUFA relative to the total kilocalories, and 2) cholesterol and fat content in different foods. The experimenter chose to focus only on these few aspects of the AHA dietary guidelines because these dietay components are most often associated with risk of CHD (93-99).

Data Analysis

The data obtained from the participants were carefully screened and tabulated. The diet records were processed by a computer program developed by Wentworth and Choquette (115) to analyse dietary intake information. The serum data and these dietary data were all

later analysed using statistical programs on Statistical Analysis System (SAS Inc., Cary North Carolina) to calculate the means and standard deviations for age, test scores, quantities of lipids, and also Pearson correlation coefficients between serum lipids and intakes of dietary lipids, dietary patterns between parents and children, and serum lipid profiles between parents and children in the sample.

CHAPTER IV

RESULTS AND DISCUSSIONS

The purpose of this study was to determine, broadly, if a correlation exists between 1) parents' serum lipid profiles and those of their children, 2) parents' intakes of dietary lipid and those of their children, and 3) serum lipid profiles, intakes of dietary lipids, and body mass index (BMI), among the parents and children.

Characteristics of the Participants

A total of 57 participants from 14 families were recruited (Appendix K). These included 28 parents and 29 children, 15 males and 14 females. All participants completed their project except for two drop-outs, both children. One child elected not to participate while another child was afraid of having the blood drawn although she had completed everything else. Also one family did not return their four-day diet records although they had their serum lipid profile done. The number of participants used in calculations of Pearson correlation coefficients involving these dropouts were accounted for by the computer programs.

The average age of parents and children is shown in Table 1. The participants ranged in age from 10 to 55 years old, with a mean age of 29.2 $\pm$ 15.0 years. The

TABLE 1

Age of the parents and children (means and standard deviations) by gender.

		Age (years)	
		Mean	Standard deviation
Parents	n		
All	28	43.8	± 4.9
Father	14	44.7	± 5.0
Mother	14	42.9	± 5.0
Children			
All	29	15.2	± 3.4
Male	15	16.1	± 3.4
Female	16	14.2	± 3.3

parents were very similar in age. The oldest child was 21 years old while the youngest was 10 years old. The children thus showed a greater distribution in age. These children were all residing with their parents.

Nutritional Knowledge. Table 2 is a summary of the overall performance of the participants on the test of their knowledge on the three categories of the dietary guidelines established by the American Heart Association (AHA). The categories were the proper level of intake of dietary fats, food selection, and food composition. This test (Appendix J) was used to assess the participants' knowledge of these guidelines about intake of fats and cholesterol. Analysis of the test scores shows that the male children had the lowest scores with a mean score of 9.6 $\pm$ 2.7 out of 21 points. This may indicate that the children were not aware of the importance of nutrition and their diet. It may be because they were still too young to be concerned with this aspect of preventive health . The parents also did not perform well themselves since the mean test scores were 13.6 $\pm$ 3.7 and 13.3 $\pm$ 3.2 for fathers and mothers, respectively. The parents themselves were in need of better nutrional education. From the test results, it appeared that both the parents and children were somewhat more aware of what the food composition in their diet was. However, they were not as aware of food selection and what the percent

TABLE 2

Summary of the nutritional knowledge test scores of the participants based on selected dietary guidelines of the American Heart Association.

Test Category	Total Possible points	Points attained (mean, S.D.) Parents Father (n=14)	Parents Mother (n=14)	Children Male (n=15)	Children Female (n=14)
Dietary Fat	3	2.2±0.8	2.3±0.6	1.5±0.6	1.5±0.8
Food Selection	3	2.6±0.5	2.3±0.5	1.9±0.6	2.1±0.5
Food Composition	15	10.0±3.1	10.2±2.5	7.5±2.2	8.6±2.8
Overall	21	13.6±3.7	13.3±3.2	9.6±2.7	11.0±2.6

of total fat, saturated fat, and polyunsaturated fat should be in the total diet recommended by AHA and the food sources for these lipids. In general though, their nutritional knowledge was much less than adequate. This overall deficiency in nutritional knowledge may lead to persistent poor dietary habits which can aggravate their diet related health problems.

Physical Activity. Table 3 shows that the majority of the participants were moderately active. This was judged to be so by their responses in the questionaire (Appendix H) when they indicated that their routine activities were mostly of either category C only, or category A or category B in addition to the regular activities they engaged in. These regular activities included jogging, biking, swimming, and playing other sports three or four times per week. The parents in general reported moderate to active physical activity levels. Generally, the children were slightly more active perhaps because of the routine structured activity they had in schools. These activities included physical education and sports which were parts of the extracurricular activities required by the schools. The correlation coefficient between physical activity and the serum lipid profile was calculated since these variables had been demonstrated to be intimately correlated (48-54), however, no statistically significant correlation was

TABLE 3

Physical activity levels of participants

Physical Activity Level	Parents (n = 28)		Children (n = 29)	
	n	%	n	%
sedentary	4	15	3	10
light	6	22	5	17
moderate	13	48	18	62
active	4	15	3	10

observed in this study.

Drinking and Smoking Status. Table 4 is a tabulation of the drinking status of the participants. Most parents considered themselves as social drinkers who drank about 6-10 oz of wine or cocktails during social functions only. Only 29% replied that they did not drink at all. Most children did not drink. Those who reported drinking were those who were in college. Even so, they also considered themselves to be social drinkers. Table 5 is a summary of the smoking status of the participants. The majority of the participants (64%) were non-smokers. The parents who were smokers were regular smokers (14%) who smoked about 24 cigarettes on a typical day. Only 7% of the children said that they occassionally smoked.

Familial History of CHD. Since familial history of CHD had been shown by many workers to be an important risk factor for the offspring (18-24), this information was solicited using a questionaire (Appendix G). From the questionaire, it was found that four adult participants had a personal history of CHD, two had siblings with history of CHD, 12 had fathers, and eight had mothers, and five had both parents with histroy of CHD. These results are tabulated in Table 6. In this study, a family was classified as having familial history of CHD if at least one of the parents in the family had indicated that either he or she personally had, or that

TABLE 4

Drinking status of participants

Drinking status	Parents (n = 28)		Children (n = 29)	
	n	%	n	%
drinker	20	71	7	24
non-drinker	8	29	22	76

TABLE 5

Smoking status of participants.

Smoking status	Parents (n = 28)		Children (n = 29)	
	n	%	n	%
smoker	4	14	2	7
ex-smoker	6	21	0	0
non-smoker	18	64	27	93

TABLE 6

Summary of the number of adult participants with different categories of family history of CHD.

History Type	Number of Participants
Personal	4
Father only	12
Mother only	8
Both Parents	5
Sibling	2

either his or her parents or siblings had CHD. According to this criterion, 11 of the participating families were classified as having familial CHD. However, only five families had both parents with familial CHD, and only three families had no known familial CHD. Furthermore, when considerations were made on an individual basis, 16 of the 28 adults had a family history of CHD.

Clinical Characteristics of Participants

Blood Pressure. An analysis of the blood pressure status of the parents indicated that 21 (75%) of them were classified as normotensive (less than 140/90 systolic/diastolic blood pressure). An additional 7% of the adults (only two, both males) also were normotnesive with the aid of prescribed antihypertensive agents. The rest of the audlt participants (17%) were considered hypertensive. Table 7 is a summary of the means and standard deviations of the blood pressure of the participants. The male adults and male children had higher blood pressure measurements than their female counterparts. On the average, it can be seen that the participants seemed to have normal blood pressure.

Serum Lipid Profile. Table 8 is a tabulation of the means and standard deviations of the serum lipid components of the parents and children separated by gender. The male adults had a much higher level of serum total

TABLE 7

Blood pressures for parents and children (means and standard deviations).

	Blood Pressure (mmHg)	
	Systolic	Diastolic
Parents		
All	123.2 ± 12.3	81.3 ± 11.3
Father	130.6 ± 10.8	87.9 ± 9.7
Mother	115.8 ± 8.7	74.7 ± 8.8
Children		
All	115.7 ± 13.8	66.1 ± 13.5
Male	122.8 ± 14.5	70.0 ± 11.8
Female	108.1 ± 8.1	62.1 ± 8.3

TABLE 8

Serum lipid profiles (means and standard deviations) of parents and children.

	TC	HDL-C	LDL-C	VLDL-C
Parents				
All	222 ± 39	46 ± 12	139 ± 35	37 ± 28
Father	237 ± 39	39 ± 11	149 ± 37	48 ± 33
Mother	208 ± 34	52 ± 9	128 ± 31	27 ± 17
Children				
All	185 ± 34	47 ± 11	116 ± 30	21 ± 6
Male	177 ± 33	43 ± 9	112 ± 28	22 ± 7
Female	195 ± 34	51 ± 10	121 ± 32	21 ± 6

cholesterol, LDL-C, and VLDL-C, while at the same time had a considerably lower level of HDL-C compared to the female adults. The children did not show such a great disparity although the level of HDL-C for the male children was also lower than that of the females. Tables 9 and 10 are summaries of the serum lipid profile characteristic of the adults and children participants, respectively, in terms of the percentile rank based upon the Lipid Research Clinics Population Studies Data from the Lipid Metabolism Branch of the National Heart, Lung, and Blood Institute (116). Since one of the parents in each family must have had a serum total cholesterol of at least 240 mg/dL without regard to familial history of CHD to participate, it is not surprising that 78.6% of the participants had above average (more than 50 percentile) serum total cholesterol. But only 14.3% of the adult participants had HDL-C level above the 50th percentile. The percent distribution of participants with the corresponding percentile rank LDL-C level almost parallels that of the serum total cholesterol. Similar observation is seen in the pediatric participants.

Correlation of Serum Lipids With Dietary Lipids

As mentioned in the section on familial history of CHD, a participating family was considered to have a family history of CHD if at least one of the parents had

TABLE 9

Distribution of serum lipids of parents, based on The Lipid Research Clinics Population Studies Data (116).

Serum Lipid Profile	Percentile Rank below 50	50-75	75-90	90-95
Total cholesterol (mg/dl)	21.4%	35.7%	14.3%	28.6%
HDL-cholesterol (mg/dl)	85.7%	7.1%	3.6%	3.6%
LDL-cholesterol (mg/dl)	35.7%	32.1%	14.3%	17.9%
VLDL-cholesterol (mg/dl)	67.9%	--	--	32.1%

TABLE 10

Distribution of serum lipids of children, based on The Lipid Research Clinics Population Studies Data (116).

Serum Lipid Profile	Percentile Rank below 50	50-75	75-90	90-95
Total cholesterol (mg/dl)	18.0%	25.0%	25.0%	32.0%
HDL-cholesterol (mg/dl)	68.0%	14.3%	7.0%	10.7%
LDL-cholesterol (mg/dl)	25.0%	25.0%	25.0%	25.0%
VLDL-cholesterol (mg/dl)	14.3%	--	--	32.1%

had a family history of CHD. One point needed to be mentioned again, however, is that all participating families had at least one of the parents with hypercholesterolemia. Also because only three families are without any known family history of CHD, meaningful and interpretable correlations are difficult to obtain for this subsample. Henceforth, no discussion on this small subset of the participants will be attempted.

Several of the risk factors associated with CHD were identified as having strong dietary determinants (90-93,97-99), for instance, hypercholesterolemia, which had been found to be associated with CHD was associated with diet, particularly high caloric intake from dietary fats and cholesterol. Some studies had demonstrated that changes in dietary habits, particularly the reduction of saturated fats and cholesterol consumption had led to a decrease in serum lipids in hypercholesterolemic individuals (95,96). However, results of other studies conducted to confirm the association of hypercholesterolemia and dietary habits have been inconsistent and interpretations by different investigators vary (84,86).

In this study, an investigation was carried out to determine whether or not an association between serum lipids and dietary lipids can be established among the participants. The significance of this association is that it determines the validity and usefulness of dietary

modification in ameliorating hypercholesterolemia.

The means and standard deviations of the intakes of dietary lipids (Appendix M) of the participants are summarized in Table 11. In general the male participants consumed greater quantities of the food. The male children, particularly, consumed the most food, understandably because of their greater activity and growth. Table 12 shows a summary of the dietary intakes of these participants, compared with the AHA dietary guidelines. It can be seen that the majority of the subjects were taking more than the recommended levels of total and saturated fat.

Pearson Correlation coefficients were calculated for the lipid profiles and intakes of dietary lipids for the different subsamples and for different subgroups. The subsamples refer to the three categories of participants: 1) all 14 families, 2) 11 families in which at least one of the parents had a family history of CHD, and 3) 5 families in which both parents had a family history of CHD. The subgroups include parents, fathers, mothers, children, father-children, mother-children, and parents-children. Representative values of Pearson correlation coefficients and their probability values are tablulated in Tables 13 and 14. Although they were calculated, many of the insignificant correlation coefficients are not tabulated. These tables only tabulate the values

TABLE 11

Dietary intakes of parents and children (means and standard deviations).

	Parents		Children	
	Father	Mother	Male	Female
Total Kcal	2191 ± 575	1603 ± 344	2433 ± 763	1646 ± 520
Total Fat (gm)	93 ± 33	69 ± 23	100 ± 31	63 ± 27
Saturated Fat (gm)	30 ± 10	22 ± 9	40 ± 15	24 ± 13
PUFA (gm)	29 ± 9	21 ± 7	32 ± 9	20 ± 7
MUFA (gm)	19 ± 10	14 ± 5	13 ± 5	8 ± 4
Cholesterol (mg)	240 ± 107	173 ± 50	307 ± 167	190 ± 111

TABLE 12

Number of participants with dietary intakes of fats and cholesterol greater than the levels recommended by the American Heart Association.

Dietary Lipids	Parents				Children			
	Father (n=13)		Mother (n=13)		Male (n=14)		Female (n=13)	
	n	%	n	%	n	%	n	%
Total fat (>30%)	12	92	12	92	14	100	7	54
Saturated fat (>10%)	8	62	6	46	14	100	10	77
PUFA (>10%)	1	8	1	8	0	0	0	0
MUFA (>10%)	9	69	7	54	9	64	7	54
Cholesterol (>300mg)	3	23	0	0	7	50	3	23

TABLE 13

Pearson correlation coefficients and probability values (in parenthesis) for correlation between serum lipids and intakes of dietary lipids in fathers.

Dietary Lipids	Number of Families	Serum lipids: Total Cholesterol	HDL-C	LDL-C	VLDL-C
Total fat	14[1]	-0.32 (0.29)	0.18 (0.56)	-0.29 (0.34)	-0.11 (0.73)
	11[2]	-0.34 (0.34)	0.17 (0.64)	-0.34 (0.33)	-0.08 (0.84)
	5[3]	0.45 (0.44)	-0.51 (0.38)	-0.28 (0.64)	0.38 (0.53)
Saturated fat	14	-0.12 (0.70)	0.33 (0.27)	-0.28 (0.35)	0.08 (0.80)
	11	-0.17 (0.63)	0.37 (0.30)	-0.43 (0.22)	0.13 (0.72)
	5	0.68 (0.20)	-0.74 (0.15)	-0.52 (0.37)	0.63 (0.36)
Cholesterol	14	0.66 (0.01)	-0.27 (0.36)	0.53 (0.06)	0.28 (0.28)
	11	0.64 (0.05)	-0.19 (0.50)	0.35 (0.32)	0.41 (0.24)
	5	0.55 (0.33)	-0.47 (0.43)	-0.09 (0.89)	0.27 (0.66)

1 = all 14 families
2 = 11 families in which at least one of the parents had a family history of CHD
3 = 5 families in which both families had a family history of CHD

TABLE 14

Pearson correlation coefficients and probability values (in parenthesis) for correlation between serum lipids and intakes of dietary lipids in children.

Dietary Lipid	Number of Families	Serum Lipids			
		Total Cholesterol	HDL-C	LDL-C	VLDL-C
Total Fat	14[1]	-0.10 (0.62)	-0.05 (0.82)	-0.05 (0.82)	-0.01 (0.96)
	11[2]	-0.19 (0.42)	-0.42 (0.06)	-0.06 (0.81)	0.04 (0.87)
	5[3]	-0.17 (0.62)	-0.54 (0.09)	0.03 (0.93)	-0.04 (0.90)
Saturated Fat	14	-0.12 (0.56)	-0.25 (0.22)	-0.05 (0.82)	-0.01 (0.69)
	11	-0.20 (0.39)	-0.37 (0.10)	-0.07 (0.77)	-0.05 (0.84)
	5	-0.19 (0.58)	-0.46 (0.15)	0.002 (0.99)	-0.18 (0.60)
Cholesterol	14	-0.05 (0.80)	-0.36 (0.07)	0.04 (0.83)	0.14 (0.51)
	11	-0.15 (0.54)	-0.55 (0.01)	0.01 (0.95)	0.16 (0.49)
	5	-0.17 (0.61)	-0.63 (0.04)	0.06 (0.86)	-0.07 (0.84)

1 = all 14 families
2 = 11 families in whcih at least one of the parents had a family history of CHD
3 = 5 families in which both parents had a family history of CHD

obtained from the fathers and children subgroups in the three categories of sample, ie., that of all 14 families, 11 families in which at least one of the parents had a family history of CHD, and 5 families in which both parents had a family history of CHD. Even for those tabulated, one can see that a great majority of the values are of no significance. Looking at the overall correlation coefficients tabulated in Table 13, correlation between the serum lipid profiles and the intakes of dietary fats is not well established. For all the different subgroups, for instance, parents, fathers, mothers, and children by themselves, or father-children, mother-children, parents-children or even separation by gender, it is found that there is no significant correlation between serum total cholesterol, HDL-C, LDL-C, VLDL-C with dietary total fat, saturated fat, MUFA, PUFA, or total kilocaloric intake. This is the case regardless if the sample taken is all the 14 participating families or only 11 families in which at least one of the adults had familial CHD.

However, intake of dietary cholesterol, specifically, does show varying degree of significant correlation with the serum total cholesterol, HDL-C, and LDL-C for some subgroups of participants. For the subgroup fathers, as shown in Table 13, the Pearson correlation coefficients for serum total cholesterol and dietary

cholesterol for all the 14 families and for sample in which only the 11 families with familial CHD are considered, are 0.66 ($p = 0.01$) and 0.64 ($p = 0.05$), respectively. Figure 1 is a scatter plot for the correlation of serum total cholesterol and dietary cholesterol for the father subgroup in the case in which the 11 families with familial CHD are considered. Such significant correlation are not observed in all other subgroups. The other rather significant correlation of 0.53 ($p = 0.06$) was also found between LDL-C and intake of dietary cholesterol for the fathers subgroup in the case which had all 14 participating families included. Thus the results seem to indicate that for the adult males, the serum total cholesterol level correlates well with the amount of dietary cholesterol intake much more than other subgroups. The other rather significant correlation was found between HDL-C and intake of dietary cholesterol for the children subgroup shown in Table 14. In this instance it was a negative correlation. The values of the Pearson correlation coefficient were -0.55 ($p = 0.01$) and -0.36 ($p = 0.07$) for the sample of 11 families in which at least one of the parents had a family history of CHD, and all 14 families included, respectively.

Studies (16,21,29) had shown that children with family history of CHD were considered at high risk. The correlation between HDL-C and intake of dietary choles-

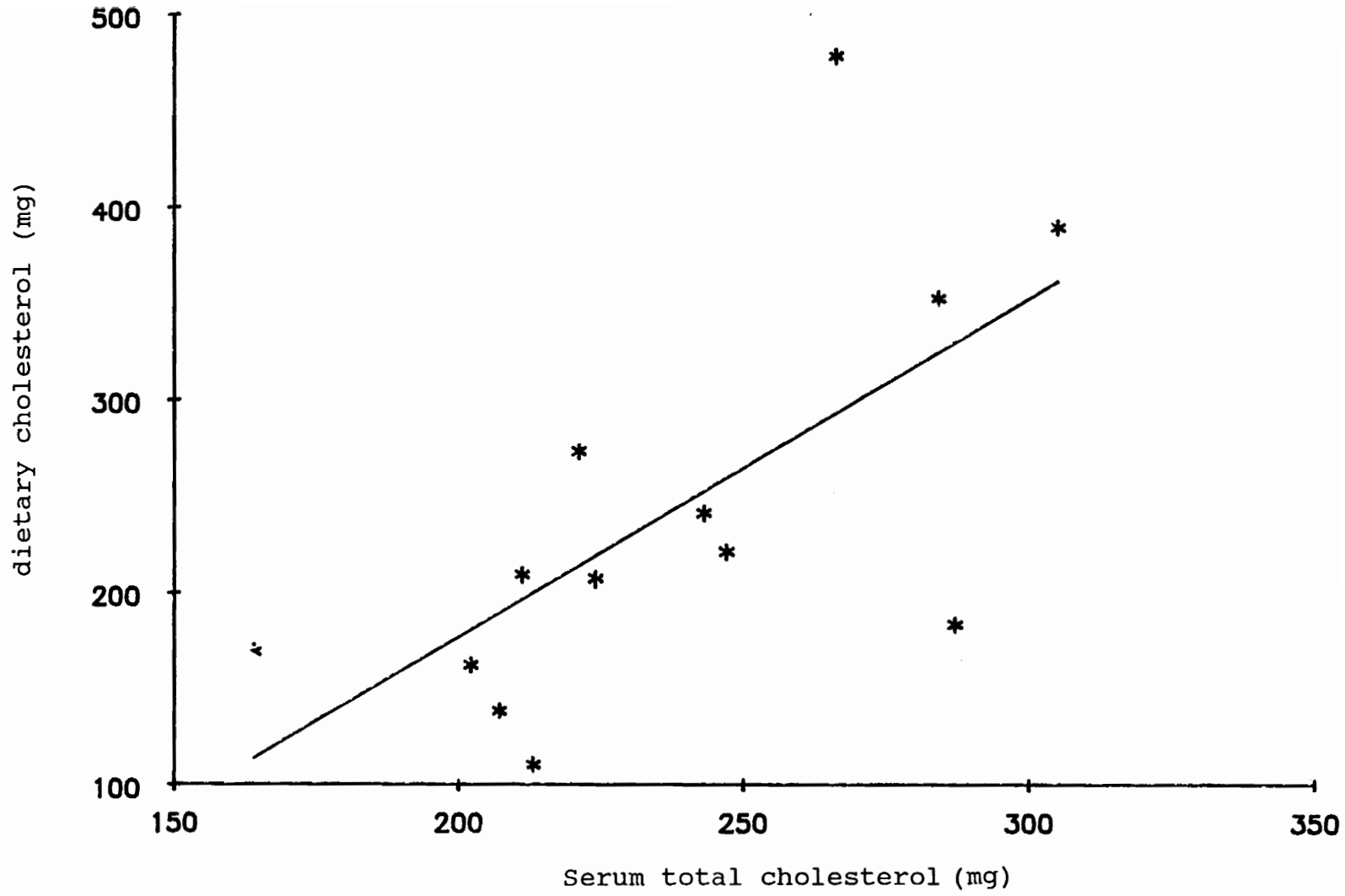


Figure 1. Scatter plot for dietary and serum total cholesterol of fathers

terol for the children shown here seems to agree with the above studies because low level of HDL-C was commonly found in individuals with such familial history. If a low level of HDL-L may be the result of high level of dietary cholesterol, this dietary habit of high intake of cholesterol may exacerbate the risk of CHD. It is also notable that the correlation coefficient is greater in magnitude for the children in the subpsample with familial history of CHD (0.55 vs 0.36). The results from the study by Pometta and co-workers (117) indicated that the level of HDL-C was more genetically regulated than by dietary factors. Thus this study seems to point in the same direction.

Similarity of Dietary Patterns Between Parents and Children

Familial aggregation of hyperlipidemia had been known to cluster in members of the family with history of CHD (13,14,16,18,20). Such phenomenon may be attributed, in part to the shared environments of the parents and children (34,36,38). This phenomenon, specifically, the dietary patterns of the families was studied further in this project. An attempt was made to observe if there were similarities in dietary patterns of the parents and those of their children. This similarity, if any, may result in a perpetuation or accentuation of familial CHD,

particularly in light of proven validity of deleterous effects of high intake of dietary lipid on serum lipid profiles.

Pearson correlation coefficients of intake of dietary lipids between parents and children were calculated for the subsamples based on these three categories of participants:

1) all 14 families,
2) 11 families in which at least one of the parents had a family history of CHD, and
3) five families in which both parents had a history of CHD.

Correlation for these three categories were calculated for all the dietary lipid components between the different members of the families, such as between father and children, mother and children, and parents and children. The discussion below will focus on dietary total fat, saturated fat, and dietary cholesterol because these are the ones which had significant correlation as shown in Table 15.

For the sample with all 14 families included, the Pearson correlation coefficients for the dietary fat and saturated fat between parents and their children was 0.43 ($p = 0.02$) and 0.50 ($p = 0.01$), respectively. For cholesterol, the value is only 0.27 ($p = 0.16$). However, such is not the case when only the 11 families in which

TABLE 15

Pearson correlation coefficients, and probability values (in parenthesis) for correlation of dietary lipids between children and parents, fathers, and mothers.

		Total fat	Saturated fat	Cholesterol
Parents and Children	14[1]	0.43 (0.02)	0.50 (0.01)	0.27 (0.16)
	11[2]	0.47 (0.02)	0.56 (0.01)	0.51 (0.01)
	5[3]	0.65 (0.02)	0.79 (0.002)	0.59 (0.04)
Fathers and Children	14	0.28 (0.16)	0.33 (0.10)	0.03 (0.90)
	11	0.34 (0.14)	0.41 (0.07)	0.38 (0.09)
	5	0.59 (0.06)	0.65 (0.03)	0.55 (0.08)
Mothers and Children	14	0.22 (0.26)	0.27 (0.17)	0.57 (0.002)
	11	0.11 (0.62)	0.19 (0.41)	0.64 (0.002)
	5	0.23 (0.50)	0.40 (0.22)	0.62 (0.04)

1 = all 14 families
2 = 11 families in which at least one of the parents had a family history of CHD.
3 = 5 families in which both parents had family history of CHD.

at least one parent had a family history of CHD were considered. In this case, the Pearson correlation coefficients for the dietary fat, saturated fat, and cholesterol are 0.47 ($p = 0.02$), 0.56 ($p = 0.01$), and 0.51 ($p = 0.01$), respectively. For the sample in which both the parents were with family history of CHD, the Pearson correlation coefficients obtained were respectively, 0.65 ($p = 0.02$), 0.79 ($p = 0.002$), and 0.57 ($p = 0.04$), for intake of dietary total fat, saturated fat, and cholesterol between parents and children. Figure 2 is a scatter plot for the correlation of dietary cholesterol between children and parents for the case in which the 11 families with familial CHD are considered. Somewhat lower correlation was seen between children with father and with mother separately. These results taken together, seemed to indicate that the dietary patterns of the children were associated with that of their parents. Also it seemed that the association between the parents and the children in their dietary habits is stronger in the case where there is a family history of CHD. This means that, if the association between dietary lipids and serum lipid profile is firmly established, which seems to be so for some populations (93-97), that familial CHD may be partially perpetuated and accentuated by familial dietary pattern. Thus dietary modification of children in families with a history of CHD can prove to be

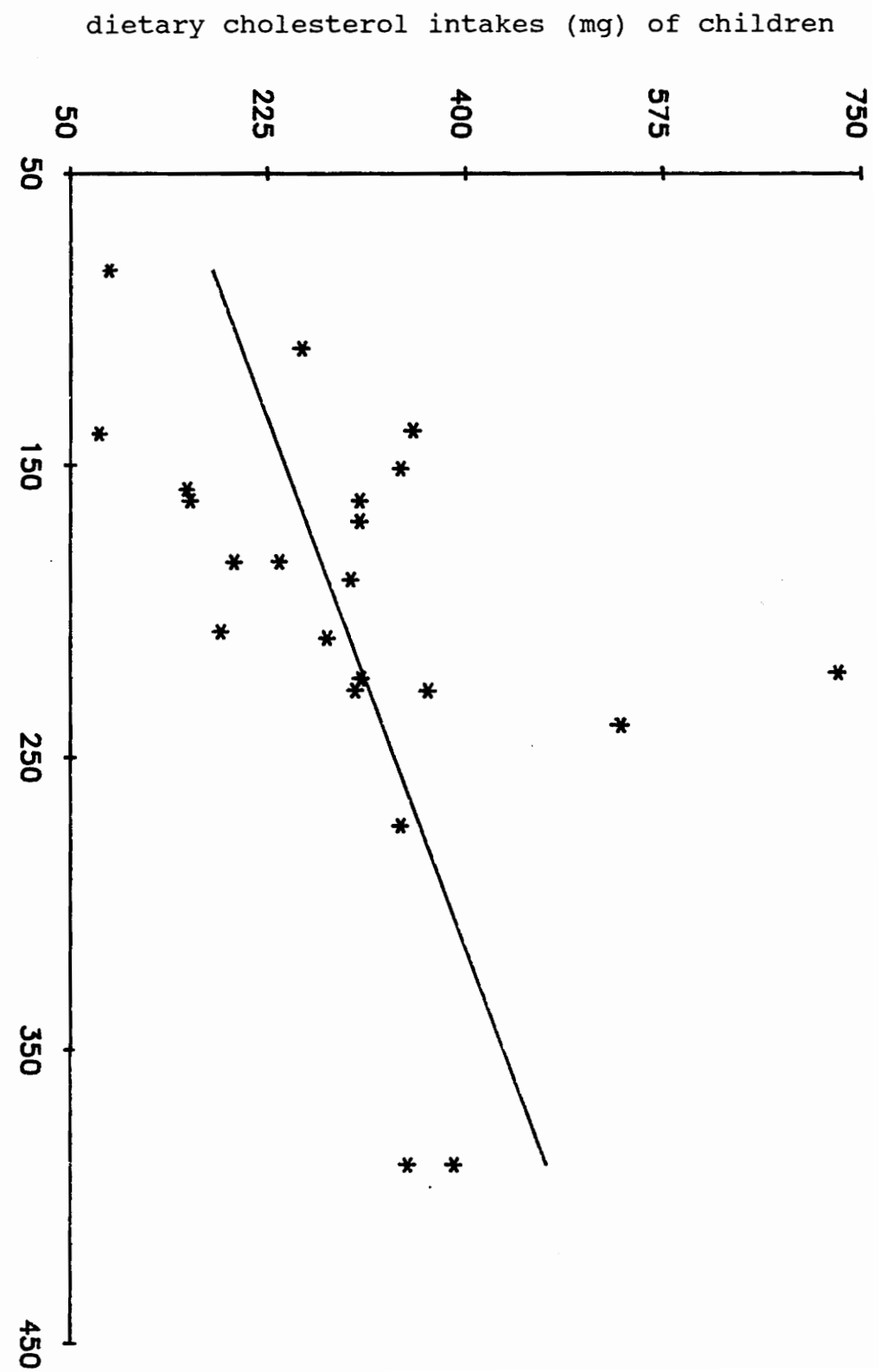


Figure 2. Scatter plot for dietary cholesterol of children and parents

beneficial in the long term picture. More studies are needed to further substantiate these findings.

Correlation of Serum Lipid Profiles Between Parents and Their Children

Observation of the clustering of elevated serum lipid profiles in children whose parents had family history of CHD had led researchers (30) to conclude that identification of family history was a valuable means to assess the relative risk of developing CHD in pediatric population. Studies by Morrison and co-workers (34) also found that there was a significant association in HDL-C between parents and children. In order to reaffirm the role and relative importance of familial dietary patterns in clustering of familial CHD in this study, one also needs to determine the role and relative importance of serum lipid profiles in the clustering of familial CHD in the sample, and then compare their association to familial aggregation of CHD with each other. If the serum lipid profile of children closely parallels that of their parents without similar association for the dietary lipids, this may suggest a relatively low involvement of dietary factor in familial CHD. If, however, the reverse is true, that is, a higher correlation is found betwen the dietary patterns of parents and children as compared to the serum lipid profiles between both, and that association between serum lipid profiles and intakes of

dietary lipids is established then dietary pattern is indeed having an important role in familial CHD. Obviously, results intermediate to both cases mentioned above can arise.

Pearson correlation coefficients were calculated for the serum lipid profiles between father and children, mother and children, and parents and children for the different categories of participants mentioned in the section on similarity of dietary pattern between parents and children. As shown in Table 16 no significant correlation was seen between the members of the families for serum total cholesterol, LDL-C, and VLDL-C. The only significant correlation was found for HDL-C between parents and their children in the case with 11 families in which at least one parent had a family history of CHD. The correlation coefficient for HDL-C between the parents and children for this sample is 0.58 ($p = 0.003$) while that for the five families only is 0.48 ($p = 0.01$). Similar trend is seen in father-children and mother-children subgroups. The correlation of HDL-C for children and father is greater than that between mother and children, and across the board for all the three subsamples of 14, 11, and 5 families taken together. Figure 3 is a scatter plot for the correlation of HDL-C between chiledren and fathers for the subsample of 11 families. These results thus seem to indicate that

TABLE 16

Pearson correlation coefficients and probability values (in parenthesis) for correlation of serum lipids between the children and parents, fathers, and mothers.

Members		Total Cholesterol	HDL-C	LDL-C	VLDL-C
Parent and Children	14[1]	0.01 (0.95)	0.48 (0.01)	0.13 (0.50)	0.25 (0.19)
	11[2]	-0.07 (0.73)	0.58 (0.003)	-0.29 (0.17)	0.36 (0.16)
	5[3]	0.44 (0.14)	0.08 (0.81)	-0.19 (0.54)	0.35 (0.26)
Father and Children	14	-0.29 (0.14)	0.52 (0.01)	-0.19 (0.32)	0.08 (0.68)
	11	-0.33 (0.14)	0.51 (0.02)	-0.20 (0.40)	0.08 (0.72)
	5	0.002 (0.996)	0.57 (0.07)	0.07 (0.85)	0.15 (0.65)
Mother and Children	14	0.22 (0.26)	0.44 (0.02)	0.06 (0.70)	0.29 (0.13)
	11	0.10 (0.66)	0.46 (0.03)	0.06 (0.79)	0.41 (0.06)
	5	0.43 (0.19)	0.25 (0.46)	0.34 (0.31)	0.56 (0.01)

1 = all 14 families
2 = 11 families in which at least one of the parents had a family history of CHD
3 = 5 families in which both parents had a family history of CHD

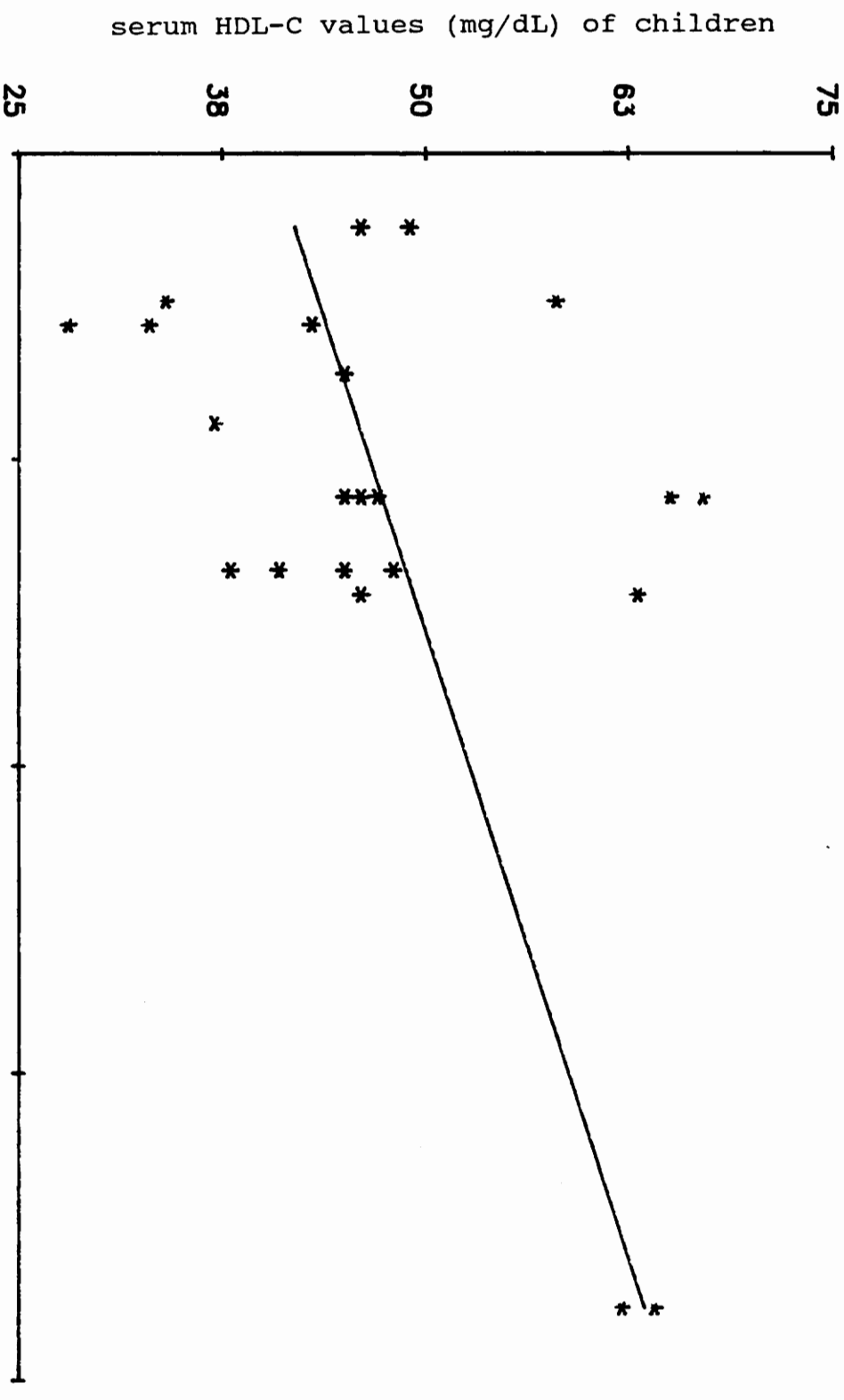


Figure 3. Scatter plot for HDL-C of children and fathers.

family history of CHD may play a role in familial aggregation of CHD especially considering the importance of HDL-C.

Correlation of Serum and Dietary Lipids With Body Mass Index

Because obesity has become an increasingly important risk factor for CHD (110), an analysis of the association between serum and dietary lipids and body mass index was done. Tables 17 and 18 are tabulations of the values of Pearson correlation coefficient and the probability values for these associations of the 14 families of participants. From Table 17, it can be noted that for all the participants, the serum lipid profiles correlated well with body mass index except for HDL-C. For the father subgroup, significant correlations were seen for serum total cholesterol (0.75, $p = 0.002$) and LDL-C (0.77, $p = 0.001$). For the children subgroup, there was correlation between body mass index with VLDL-C only. However, the mother subgroup showed very little significant correlation.

There was no significant correlation between body mass index with dietary lipids in all, mother and children subgroups. An exception to this was the very significant correlation (0.69, $p = 0.01$) between intake of cholesterol and body mass index for the father subgroup, the population which is generally known to be

TABLE 17

Pearson correlation coefficients and probability values (in parenthesis) for correlation between body mass index and serum lipids in all 14 families.

	TC	HDL-C	LDL-C	VLDL-C
All	0.48 (0.0002)	-0.29 (0.03)	0.45 (0.001)	0.36 (0.007)
Father	0.75 (0.002)	-0.33 (0.25)	0.77 (0.001)	0.12 (0.69)
Mother	-0.12 (0.68)	-0.45 (0.11)	0.07 (0.82)	-0.12 (0.69)
Children	0.09 (0.66)	-0.17 (0.40)	0.07 (0.74)	0.42 (0.03)

TABLE 18

Pearson correlation coefficients and probability values (in parenthesis) for correlation between body mass index and dietary lipids in all 14 families.

	Kcal	Total fat	Saturated fat	Cholesterol
All	-0.02 (0.90)	0.04 (0.77)	-0.12 (0.39)	0.01 (0.95)
Father	0.03 (0.91)	0.04 (0.90)	0.15 (0.63)	0.69 (0.01)
Mother	-0.37 (0.21)	-0.27 (0.37)	-0.31 (0.31)	0.26 (0.40)
Children	0.05 (0.81)	0.07 (0.72)	0.02 (0.91)	-0.14 (0.48)

at greater risk of CHD than the others. This set of results seem to be in agreement with generally held notion of association of a particular group of the general population and their overall response to intake of dietary lipids. More in-depth studies would be needed to confirm the observations obtained in this study.

CHAPTER V

SUMMARY AND CONCLUSIONS

The objectives of this research project were several: 1) to confirm the association between serum lipid profiles and intake of dietary lipids for the sample under study, 2) to ascertain whether or not there are associations between the serum lipid profiles of the parents and those of the children, and 3) to determine whether or not the children's dietary pattern is similar to that of their parents.

From the 14 families in which at least one of the parents had hypercholesterolemia with serum total cholesterol level of at least 240 mg/dL, lipid profiles, dietary records, information on family history of CHD, and other pertinent information are obtained to perform correlation studies. These 14 families comprised five families in which both parents had a family history of CHD, 11 families with at least one of the parents having a family history of CHD, and other three families with no known family history of CHD. The sample size and population categories were less than ideal, particularly the number of families with both parents without a known histroy of CHD. Thus some of the findings may be tentative. However, the results obtained seem to affirm some of those in the literature.

For the sample investigated, it was found that there was no significant correlation between serum total cholesterol, HDL-C, LDL-C, VLDL-C and dietary total fat, saturated fat, MUFA, PUFA, or total kilocalorie. However, significant correlations of 0.66 ($p = 0.01$) and 0.64 ($p = 0.05$) were noted for the fathers subgroup between serum total cholesterol and dietary cholesterol for all the 14 families and the 11 families in which at least one of the parents had a family history of CHD, respectively. The other rather significant inverse correlations of -0.36 ($p = 0.07$) and -0.55 ($p = 0.01$) were found between the HDL-C and dietary cholesterol for the children subgroup for the total sample and the 11 families subgroup, respectively.

Associations of serum lipid profiles between the parents and children were found to be insignificant. The exception to this was for HDL-C between the parents and children for the total sample with $r = 0.48$ ($p = 0.02$) and the subsample of 11 families, with $r = 0.57$ ($p = 0.03$). The small difference in the correlation seems to indicate some suggestion of a family history factor. However, the poor correlation among other subsamples make this observation tentative.

These results show that there are similarities in the dietary patterns between parents and their children. Rather low correlation coefficients of 0.42 ($p = 0.02$)

and 0.23 ($p = 0.24$) were obtained for dietary total fat and cholesterol, respectively, in the total population. However, the correlation coefficients increase to 0.46 ($p = 0.01$) and 0.48 ($p = 0.02$), respectively, for the subsample of 11 families with at least one parent having a family history of CHD. The correlations increase further, to 0.67 ($p = 0.01$) and 0.57 ($p = 0.03$), respectively, for dietary total fat and cholesterol, for the subsample with five families in which both parents had a family history of CHD. This is the most notable result of this study, one which seems not to have been reported before. This finding seems to suggest that familial aggrgation of CHD may have been perpetuated and accentuated by dietary pattern passed on between generations. It would mean that dietary modification may be an important means to break the cycle of family history of CHD. Further research involving a population in which family units tend to stay at one locale and are closely knit, might focus on such a process.

LITERATURE CITED

1. Levy, R. I. The decline in cardiovascular disease mortality. Ann. Rev. Pub. Health, 2:49, 1981.

2. Lipid Research Clinics Program. The lipid research clinics coronary primary prevention trial results. I. Reduction in incidence of coronary heart disease. J. Am. Med. A., 251:351-364, 1984.

3. Berenson, G. S. Causation of cardiovascular risk factors. Raven Press, New York, 1986.

4. Blumental S. Prevention of atherosclerosis. Am. J. Card., 31:591, 1973.

5. Strong, J., Eggen D. A., Oalman, M. C., Richard, M. L., and Tracy, R. E. Pathology and epidemiology of atherosclerosis. J. Am. Med. A., 62:262, 1973.

6. Laven, R. M., Shekelle, R. B. Childhood Prevention of Atherosclerosis and Hypertension. Raven Press, New York, 1980.

7. Holman, R. L., Mcgill, H. C., Strong, J. P. and Greer, J. C. The natural history of atherosclerosis: The early aortic leisions as seen in New Orleans in the middle of the 20th Century. Am. J. Pathol., 34:209, 1958.

8. Berenson, G. S. Cardiovascular risk factors in children: The Early Natural History of Atherosclerosis and Essential Hypertension. Oxford University Press, New York, 1980.

9. Multiple Risk Factor Intervention Trial Research Group: Multiple risk factor intervention trial-risk factor changes and mortality results. J. Am. Med. A., 248:1465, 1982.

10. Hass, J. Study of atherosclerosis precurssors in children. Report of W.H.O. Consultation, WHO CVD-74.4, Geneva, 1974.

11. Epstein, F. H., and Lloyd, J. Study of atherosclosis precussor in children. Report of W.H.O. Consultation, WHO CVD-74.4, Geneva, 1974.

12. Conference on the Health Effects of Blood Lipids: Optimal Distributions for Populations Workshop Report: Epidemiologic Section III. The epidemiologic evidence from comparative childhood lipid distribution. Prev. Med., 8:64-652, 1979.

13. Morrison, J. A., Khoury, P., Laskarzewski, P. M., Mellies M. J., Kelly, K., and Glueck, C. J. Intrafamilial associations of lipids and lipoproteins in families of hypercholesterolemic probands. Atherosclerosis, 2:151-159, 1982.

14. Morrison, J. A., Khoury, P., Laskarzewski, P. M., Mellies, M. J., Kelly, and K., Glueck, C. J. Intrafamilial associations of lipids and lipoproteins in kindreds with hypertriglycerides proband: The Princeton School Family Study. Circulation, 66:67-76, 1982.

15. Puska P. Possibilities of a preventive approach to CHD starting in children. Acta. Paed. Scand. Suppl., 318:229, 1985.

16. Sveger, T., Fex, G., and Borgfers, N. Hyperlipidemia in school children with family history of premature coronary heart disease. Acta Paediatr. Scand., 76: 311-315, 1987.

17. Steering Committee of the American Heart Association: Guidelines for Healthy American Adults. Circulation, 74:1465A, 1986.

18. Glueck, C. J., Fallat, R. W., Tsang, R., and Buncher, C. R. Hyperlipidemia in progeny of parents with myocardial infarction before age 50. Am. J. Dis. Child., 127:70, 1974.

19. Schrott, H. G., Clarke, W. R., Wiebe, D. A., Connor, W. E., and Lauer, R. M. Increased coronary mortality in relatives of hypercholesterolemic school-children-Muscatine Study. Circ., 59:320, 1979.

20. Deutscher, S., Epstein, F. H., Kjelberg, M. O. Familial aggregation of factors associated with CHD. Circ., 33:911, 1966.

21. Rissanen, A. M., Nikkila, E. A. Aggregation of coronary risk factors in families of men with fatal and non-fatal CHD. Br. Heart J., 42:373, 1979.

22. Berg, K., Dahlen, G., Borrensen, A. L. Lipoprotein lipase activity ,phenotypes, other lipoprotein parameters and a familial history of CHD in middle-aged males. Clin. Genet., 16:347, 1979.

23. Morrison, J. A., Horvitz, R., Khoury, P., Laskarzewski, P., Gazside, P. S., Kelly, K., Mellies, M., and Glueck, C. J. Parental history of CHD, hypertension, diabetes, and stroke: Relationship to CHD risk factor variables in their adult children. Prev. Med., 9:773, 1980.

24. Kate, L. P., Bowman, H., Daiger, S. P., and Motulsk, A. G. Familial aggregation of CHD and its relation to known genetic risk factors. Am. J. Card., 50:945, 1982.

25. Forde, O. H., and Thelle, D. S. The Thromso Heart Study: risk factors for CHD related to the occurrence of myocardial infarction in the first-degree relatives. Am. J. Epidemiol., 105:192, 1977.

26. Rissanen, A. M., and Nikkila, E. A. Identification of the high risk groups in familial CHD. Atherosclerosis, 53:37, 1984.

27. Rissanen, A. M. Familial occurence of CHD-Effect of age at diagnosis. Am. J. Cardiol., 44:60, 1979.

28. Phillip, R. L., Lilienfield, A. M., Diamond, E. L., and Kagan, A. Frequency of CHD and cerebrovascular accidents in parents and sons of CHD index cases and controls. Am. J. Epidemiol., 100:87, 1974.

29. Paffenbarger, R. S., and Wing, A. L. Chronic disease in former college students,part 10: The effects of single and multiple characteristics on risk of fatal CHD. Am. J. Epidemiol., 9:484, 1980.

30. Sholtz, R. I., Rosenman, R. H., and Brand, R. J. The relationship of reported parental history to the incidence of CHD in the Western Collaborative Group Study., Am. J. Epidemiol. 102:350, 1975.

31. Gordon, T., Castelli, W. P., Hjortland, M. C., Kannel, W. B. and Dauber, T. R. High density lipoprotein as a protective factor against CHD: the Framingham Study. Am. J. Med., 62:707, 1974.

32. Friedlander, Y., Kark, J. D., Fainaru, M., Gotsman, M., and Stein, Y. Aggregation of plasma lipids and lipoproteins in families with and without CHD. Atherosclerosis, 57:235, 1985.

33. Namboodird, K. K., Green, P. P., Kaplan, E. B., Morrison, J. A., Chase, G. A., Owen, R. C., Rifkind, B. M., Glueck, C. J., and Tyroler, H. A. The Collaborative Lipid Research Clinic Program Family Study IV-Familial association of plasma lipids and lipoproteins. Am. J. Epidemiol. 119:975, 1984.

34. Morrison, J. A., Khoury, P., Mellies, M. J., Kelly, K. A., and Glueck, C. J. Identifying CHD risk factors in children: Intrafamilial lipoprotein correlations., Prev. Med. 9:484, 1980.

35. Morrison, J. A., Kelly, K. A., Mellies, M. J., et al. Parent-child associations at upper and lower ranges of plasma cholesterol and triglycerides levels. Ped., 62:468, 1978.

36. Laskarzewski, P. M., Morrison, J. A., Kelly, K., Khoury, P., Mellies, M., and Glueck, C. J. Parent-child coronary heart disease risk factor associations. Am. J .Epidemiol., 114:827, 1981.

37. Moll, P. P., Sing, C. F., Weidman, W. H., Gorden, H., Ekefson, R. D., Hodgson, P. A., and Kottle, B. A. Total cholesterol and lipoproteins in school children: Prediction of CHD in adults relatives. Circ., 67:127, 1983.

38. Garrison, R. J., Castelli, W. P., Feinleib, M., Kannel, W. B., Havlik, R. J., Padgett, S. J., and McNamara, P. M. The association of total cholesterol, triglycerides and plasma lipoprotein cholesterol levels in the first degree relative and spouse pairs. Am. J. Epidemiol., 110:313, 1979.

39. National Center for Health Statistics: NCHS Growth Charts, Monthly Vital Statistics Report. Vol. #3 suppl. (HRA) p.76, Health Resources Administration, Rockville, Maryland, 1970.

40. Freedman, D. S., Shear, C. L., Srinivason, S. R., Webber, L. S., and Berenson, G. S. Tracking of serum lipids and lipoproteins in children over eight-year period: The Bogalusa Study. Prev. Med., 14:203, 1985.

41. Clarke, W. R., Schrott, H. G., Leaverton, E., Connor, W. E., and Lauer, R. M. Tracking of blood lipids and blood pressure in school age children: The Muscutine Study. Circ., 58:626, 1978.

42. Frerich, R. R., Webber, L. S., Voors, A. W., Srinivasan, S. R. and Berenson, B. S. Cardiovascular risk factor variables in children at two successive years-The Bogalusa Heart Study. J. Chron. Dis., 32:251, 1979.

43. Laskarzewski, P., Morrison, J. A., Degroot, I. Kelly, K. M., Mellies, M., Khoury, M. J., and Glueck, C. J. Lipid and lipoprotein tracking in 108 children over a 4-year period. Ped., 35:223, 1984.

44. Miller, G. J., Miller, N. E. PLasma-HDL-C concentration and development of Ischaemic heart. Lancet, 1;16, 1975.

45. Gerdon, T., Castelli, W. P., Hjertland, M. S., Kannel, W. B., and Dawber, T. R. High density lipoprotein as a protective factor against CHD. Am. J. Med., 62:707, 1977.

46. Miller G. J., Miller, N. E., Ashcroft, M. T,. Inverse relationship in Jamaica between plasma HDL-C concentration and CHD risk as predicted by multiple risk-factor status. Clin. Sci. Mod. Med., 51:475, 1976.

47. Rhoads, G. G., Kagan, A., Gulbrandsen, C. L,. Serum lipoproteins and CHD in a population study of Hawaii Japanese men. New Eng. J. Med., 294:293, 1976.

48. Lopez, S. A., Vial, R., Balart, L., Arroyave, G. Effects of exercise and physical fitness on serum lipids and lipoproteins. Atherosclerosis, 20:1, 1974.

49. Wood, P. D., Haskell, W., Klein, W., Lewis, S., Stern, M. P., and Farquhar, J. W. The distribution of plasma lipoproteins in middle aged male runners. Metabolism, 25:1249, 1976.

50. Lehtonen, A., Viikari, J., Serum triglycerides and cholesterol and serum high density lipoprotein cholesterol in highly physically active men. Acta. Med. Scand., 204:111, 1978.

51. Williams, P. T., Wood, P. D., Haskell, W. L., and Vranizan, K. The effects of running mileage and duration on plasma lipoprotein levels. J. Am. Med. A., 247:2674, 1982.

52. Williams, P. T., Wood, P. D., Krauss, R. M., Haskell, W. L., Vranizan, K. M., Blair, S. N., Terry, R., and Farquhar, J. W. Does weight loss cause the exercise-induced increase in plasma HDL-C? Atherosclerosis., 47:173, 1983.

53. Sopko, G., Leon, A. S., Jacobs, D. R., Foster, J. N., Moy, J., Kuba, K., Anderson, J. T., Casal, D., McNally, C., and Frantz, I. The effects of excercise and weight loss on plasma lipids in young obese men. Metabolism, 34:227, 1985.

54. Schwartz, R. S., and Brunzell, J. D. Increase of adipose tissues lipoprotein lipase activity with weight loss. J. Clin. Invest., 67:1425, 1981.

55. Contaldo, F., Strazzullo, P., Postiglione, A., Riccardi, G., Patti, L., Biase, G. D., and Mancini, M. Plasma HDL-C in severe obesity after stable weight loss. Atherosclerosis, 37:163, 1980.

56. Brownell, K. D., and Stunkard, A. J. Differential changes in plasma HDL-C in obese men and women during weight reduction. Arch. Inter. Med., 141:1142, 1981.

57. Thompson, P. D., Jeffrey, R. W., Wing, R. R., and Wood, P. D. Unexpected decrease in plasma HDL-C with weight loss. Am. J. Clin. Nut., 32:2016, 1979.

58. Widhalm, K., Maxa, E., and Ztman, H. Effects of diet and exercise upon the cholesterol and triglycerides content of plasma lipoproteins in overweight children. Europ. J. Ped., 127:121, 1978.

59. Hickey, K., Mulcahy, R., Bourke, G. J., Graham, I and Davis, K. W. Study of coronary risk factors related to physical activity in 1571 men. Br. Med. J., 3:507, 1975.

60. Huttunen, J. K., Lansimies, E., Voultilainen, E., Ehnholm, C., Hietanen, E., Denttila, I., Siitonen, O., and Rauramaa, R. Effect of moderate physical exercise on serum lipoprotein-A controll-

ed clinical trial with special reference to serum HDL-C. Circ., 60:1220, 1979.

61. Hagan, R. D., and Gettman, L. R. Maximal aerobic power, body fat and serum lipoprotein in male distance runners. J. Cardiac. Rehab., 3:331, 1983.

62. Wolf, R. N., and Grundy, S. M. Influence of weight reduction on plasma lipoproteins in obese patients. Arteriosclerosis, 3:160, 1983.

63. Chad, F. I., Falko, J. M., Patel, S. T., Kim, M. H., Newman, H. A., and Bamws, H. Serum lipid responses during active and stable weight reduction in reproductive obese females. J. Clin. Endocrinol. Metab., 55:258, 1982.

64. Altekruse, E. B., Wilmore, J. H. Changes in blood chemistries following a controlled exercise pro gram. J. Occup. Med., 15:110, 1973.

65. Brunzell, J. D., Magill, P., Rao, S. N., Miller, N., Hilaire, S., Nicoil, R. J., and Lewis, B. HDL-C kinetics and adipose tissue lipoprotein lipase. Circ., abstr.# 272 suppl II, 1980.

66. Kekki, M. Lipoprotein-lipase action determining plasma HDL-C level in adult normolipidemics. Atherosclerosis, 37:143, 1980.

67. Nikkila, E. A., Taskinen, M. R., Rehunen, S., Harkonen, M. Lipoprotein lipase activity in adipose tissues and skeletal muscle of runners: Relation to serum lipoprotein. Metab. Clin. and Expt., 27:166: 1978.

68. Kanto, M. A., Cullinane, E. M., Herdert, P. W.,and Thompson, P. P. Acute increase in lipoprotein lipase following prolonged exercise. Metabolism, 33:454, 1984.

69. Davis, T. A., Anderson, E. C., Girisburg, A. V., and Goldberg, A. P. Weight loss improves lipid profiles in patients with hyperlipidemia. J. Lab. Clin. Med., Oct:447, 1985.

70. Lipson, L. C., Bonow, R. O., Schaeffer, E. J., Brewer, H. B., and Lindgren, F. T. Effect of exercise conditioning on plasma HDL-C and other lipoprotein. Atherosclerosis, 37:529: 1980.

71. Allison, T. G., Iammarino, R. M., Metz ,K. F., Skrinar, G. S., Skrinar, G. S., Kuller, L. H., and Robertson, R. J. Failure of exercise to increase HDL-C. J. Cardiac. Rehab., 1-2:257, 1981.

72. Horby-Peterson, J., Grande, P., and Christiansen, C. Effect of physical training on serum lipids and serum HDL-C in young men. Scand. J. Clin. Lab. Invest., 42:387, 1982.

73. Stamford, B. A., Matter, S., Fell, R. D., Sady, S., Papanek, P., and Cresanta, M. Cigarette smoking, exercise and HDL-C. Atherosclerosis, 52:73, 1984.

74. Rabkin, S. W. Effect of cigarette cessation on risk factors for coronary atherosclerosis-A controlled clinical trial. Atherosclerosis, 53:173, 1984.

75. Karvonen, M., Orma, E., Keys, A., Flaminio, F., and Brozek, J. Cigarette smoking,serum cholesterol, blood pressure and body fatness-observation in Finland. Lancet, 44:23, 1976.

76. Hjermann, I., Helgeland, A., Holmes, I., Larsen, P. G., and Laren, P. The intercorrlation of serum cholesterol,cigarette smoking and body weight: THe Oslo Study. Acta. Med. Scand., 200:479, 1976.

77. Larsen-Lund, P. G., and Tretli, S. Changes in smoking habits and body weight after a three-year period-The Cardiovascular Disease Study in Finnmark. J. Chron. Dis., 35:773, 1982.

78. Blitzer, P. H., Rimin, A. A., and Gietfer, E. E., The effect of cessation of smoking on body weight in 57,032 women: cross-sectional and longitudinal analysis. J. Chron. Dis., 30:415, 1977.

79. Wack, J. T., and Rodin, J. Smoking and its effects on body weight and the systems of caloric regulation. Am. J. Clin. Nut., 35:366, 1982.

80. Gordon, T., Kannel, W. B., Dauber, T. R., and Maltee, D. Changes associated with quitting cigarette smoking. Am. Heart. J., 90:322, 1975.

81. Greene, S. B., Aavedal, M. J., Tyroler, H. A., Davis, C. E., and Holmes, C. G. Smoking habits and blood pressure changes: A seven-year follow-up. J. Chron. Dis., 30:401, 1977.

82. Karvonen, M., Keys, A., Orma, E., Fidanza, P., and Brozek J. Cigarette smoking,serum cholesterol, blood pressure and body fatness observed in Finland. Lancet, I:492, 1959.

83 Blackburn, H., Brozek. J., Taylor, H. L., and Keys, A. Composition of cardiovascular and related characteristics in habitual smokers and non-smokers. Ann. NY. Acad. Sci., 90:277, 1960.

84. Kaplan, J. A., Cox, G. E., and Taylor, C. B. Cholesterol metabolism in man :studies on absorption. Arch. Pathol., 76:358, 1963.

85. Wilson, J. D., and Lindsey, C. A. Jr. Studies on the influence of dietary cholesterol on cholesterol metabolism in the isotopic steady steam. J. Clin. Invest., 44:1805, 1965.

86. Quintao, E. S., Grundy, S. M., and Ahrens, E. H. An evaluation of four methods for measuring cholesterol absorption by the intestine in man. J. Lip. Res., 12:221, 1971.

87. Zeman, F. J. Clinical Nutrition and Disease: Chapter 8, p269. The Collamore Press, Lexington, Massachusets, 1983.

88. Karvinen, E., Lin, T. M., and Ivy, A. C. Capacity of human intestine to absorb exogenous cholesterol. J. Appl. Physiol., 11:143, 1957.

89. Borgstrom, B. Quantification of cholesterol absorption in man by fecal analysis after the feeding of a single isotope-labelled meal. J. Lip. Res., 10:331, 1969.

90. Connor,W. E., Hodges, R. E., and Bleiler, R. E. The serum lipids in men receiving High-cholesterol and cholesterol-free diets. J. Clin. Invest., 40:894, 1961.

91. Mattson, F. H., Erickson, B. A., and Klingman, A. M. Effect of dietary cholesterol in man. Am. J. Clin. Nut., 25:589, 1972.

92. Quintao, E., Grundy, S. M., and Ahren, E. H, Effects of dietary cholesterol on the regulation of total blood cholesterol in man. J. Lip. Res., 12:233, 1971.

93. Packard, C. J., Mckinney, L., Carr, K., and Shepherd J. Cholesterol feeding increases low density lipoprotein synthesis. Am. Soc. Clin. Invest., 72:45, 1983.

94. Brown, M. S., Goldstein, J. L. How LDL receptors influence cholesterol and atherosclerosis. Sci. Am., 251:58, 1984.

95. Tan, M. H., Dickinson, M. A., Alber, J. J., Havel, R. J., Cheung, M. C., and Vigne, J. L. The effect of a high cholesterol and saturated fat on serum HDL-C, apoprotein A-1, and apoprotein E levels in normilipidemic humans. Am. J. Clin. Nut., 33:2559, 1980.

96. Thuesen, L., Henriksen, L. B., Engby, B. One-year experience with a low fat,low cholesterol diet in patients with CHD. Am. J. Clin. Nut., 44:212, 1986.

97. Shepherd, J., Packard, C. J., Grundy, S. M., Yeshurum, D., Gotto, A. M., and Tauton, D. D. Effects of saturated and polysaturated fat diet on the chemical composition and metabolism of LDL-C in man. J. Lip. Res., 21:91, 1980.

98. Nestel, P. J., Connor, W. E., Reardon, F., Connor, S., Wong, S., and Boston, R. Suppression by diets rich in fish oil of VLDL-C production in man. J. Clin. Invest., 74:82, 1984.

99. Harris, W. S,. Connor, W. E., McMurry, M. P. The comparative reductions of the plasma lipids and lipoproteins by dietary polysaturated fats: Salmon oil versus vegetable oils. Metabolism, 32:179, 1983.

100. Iacono, J. M., Marshall, M. W., Doughery, R. M., and Wheeler, M. A. Reduction in blood pressure associated with high polyunsaturated fat diets that reduce blood cholesterol in man. Prev. Med., 4:426, 1975.

101. Newman, W. P., Freedman, D. S., Voors, A. W., Gard, P. D. Srinivasan, S. R., Cresanta, J. L., Williamson, G. D., Webber, L. S., and Berenson, G. S. Relation of serum lipoprotein levels and systolic blood pressure to early atherosclerosis-The Bogalusa Heart Study. N. Eng. J. Med., 314:138, 1986.

102. Rao, R. H., Rao, U. B., and Srikantia, S. G. Effect of polyunsaturated vegetable oils on blood pressure in essential hypertension. Clin. Expt. Hyper., 3:27, 1981.

103. Mattson, F. H., and Grundy, S. M. Comparison of effects of dietary saturated, monosaturated, and polysaturated fatty acids on plasma lipids and lipoprotein in man. J. of Lipid Research, 26:194, 1985.

104. Grundy, S. M. Comparison of monosaturated fatty acids and carbohydrates for lowering plasma cholesterol. New England J. Med., 314:745-748, 1986.

105. Simopoulos, A. P., Itallie, T. B. Body Weight, health and longevity. Ann. Intern. Med., 100:285, 1985.

106. National Institues of Health: Consensus development conference statement: health implication of obesity. Ann. Intern. Med., 103:147, 1985.

107. Sonne-Holm, S., Sorenson, T. I., Christensen U. Risk of early death in extreme over weight young men. Br. Med. J., 287:795, 1983.

108. Epstein, F., Francis, J. T., Hayner, N. Prevalence of chronic diseases and distribution of selected physiologic variables in total community, Tecumseh, Michigan. Am. J. Epidiom., 81:307, 1965.

109. Rimm, A., White, P. Obesity: its risks and hazards. In Obesity in America. Bray G (Ed), Hyattsville, Maryland, DHEW Publication # (NIH), 80-359, 103-124, 1980,

110. Ashley F., Kamel, W. Relation of weight change to changes in atherogenic traits: The Framingham Study. J. Chron Disease, 27:103. 1974.

111. Key, A., Menotti, Araviaris, C. The seven contries study: 2289 death in 15 years. Prev. Med., 13:141, 1984.

112. Rudeman, N., Berentold, P., Schneider, S. Obesity-associated disorders in normal-weight individuals: some speculation. Int. J. Obesity, 6 (supple. 1) :151, 1982.

113. Burack, R. C., Keller, J. C., Higgin, M. W. Cardiovascular risk factors and obesity: are baseline levels of blood pressure, glucose, cholesterol and uric acid elevated prior to weight gain? J. Chro. Disease, 38:865, 1985.

114. Donahue, R. P., Abbott, R. D., Bloom, E., Reed, D. M., Yano, K. Central obesity and coronary heart disease in men. Lancet, April, 1987.

115. Wentworth, J., Choquette, G. Computer analysis of nutrient intake (Mimeo Series) Rev. 1981, VPI & SU, Blacksburg.

116. Lipid Metabolic Branch, Division of Heart and Vascular Diseases, National Heart, Lung and Blood Institute: The lipid research clinics population studies data book, vol. 1: the prevalence study. NIH publication No. 80-1527, US Government Printing Office, Washington, 1980.

117. Pometta, D., Micheli, H., Suenram, A., and Jornot, C. HDL lipids in close relatives of coronary heart disease patients. Atherosclerosis, 34:419, 1979.

APPENDIX A

DESCRIPTION OF THE STUDY

The focus of this study is on children (ages 10-19) whose parents have a serum total cholesterol level of 240 mg/dl or greater. The main purpose is to assess the health status of both these children and their parents. Specifically, the following characteristics of these families will be determined and compared: serum total cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein (LDL-C), very low density lipoprotein (VLDL-C), blood pressure, and weight. Questionaires will be used to obtain the following information: physical activity levels, smoking and drinking habits, and eating habits of both parents and their children.

APPENDIX B

EXPERIMENTAL DESIGN

The study will be carried out in two phases. The first phase consists of a 30-40 minute orientation meeting at which all participants will receive a verbal explanation concerning the study. At the meeting, the participants' role will be outlined clearly and they will be given an opportunity to ask questions about the study. They will be asked to keep a four-day diet record and to respond to questionaires to collect the data outlined in Appendix A. The second phase will involve collection of at least 12-hour fasting blood sample and measurements of height, weight, and blood pressure. At this time, the parents and their children will return the completed diet records to the experimenter. Blood will be drawn by a licensed medical technician between 7 a.m. and 9 a.m. at Wallace Hall on the Virginia Tech campus.

APPENDIX C

INSTRUCTIONS FOR DIETARY RECORD KEEPING

A dietary record is a detailed written record of EVERYTHING (meals and snacks) you eat or drink from the time you wake up in the morning until you go to bed at night. Be sure to indicate the method of preparation, time when foods are eaten, the type of foods, and the amount of foods eaten.

The purpose of this record is to enable us to gather more in-depth information of your normal diet. All information you provide us will be held confidential.

Suggestions

- Record your food intake and the amount IMMEDIATELY after you have finished eating. DO NOT wait until the next day. We are more concerned with the recording ACCURACY of your food intake than the coffee spills or mayonnaise stains on your food record.

- Try to be as specific as you can when listing foods you eat. If you are not sure how to describe a combination type of dish such as chicken casserole, list all the ingredients and seasonings you have in the chicken casserole.

- List the common or brand name if possible. Be sure to also indicate method of preparation, for example, stir-frying, boiling, baking, deep-fat frying, or steaming. Be sure to include the amount of oil you use in food preparation.

- Try to eat your normal meals. DO NOT consciously change your eating habits or portion sizes.

APPENDIX D

PORTION GUIDE
(HOW TO RECORD AMOUNT YOU EAT)

Meats: This should be described in terms of ounces, if possible.

Example: 1 4-oz. hamburger, 1/2 inch thick
3 slices roast beef, 3-oz per slice

Vegetables, Cereals and Beverages:
This should be described in common household measuring cups.

Example: 1 cup whole milk
1/2 cup orange juice
1 cup cooked spaghetti/macaroni/ noodles
1/2 cup steamed broccoli
3 slices of whole wheat breads

Fruits: This should be described in terms of slices or measuring cup.

Example: 1 large fresh apple
1 medium size navel orange
2 cups canned peaches, drained

APPENDIX E

DIET RECORD

NAME_____DATE_____

Please use a SEPARATE SHEET of paper for EACH DAY. Consult the guidelines for diet record when completing this record.

TIME OF DAY	DESCRIPTION OF FOOD CONSUMED	AMOUNT OF FOOD CONSUMED

APPENDIX F

GENERAL QUESTIONAIRE OF BACKGROUND INFORMATION

1) Familial history of cardiovascular disease/events (e.g. heart attack, stroke, angina pectoris, bypass surgery and so forth)

	Yes	No	Problem(please fill in this column if you check Yes)
a. Personal	___	___	_____
Father	___	___	_____
Mother	___	___	_____
Brother	___	___	_____
Brother	___	___	_____
Brother	___	___	_____
Brother	___	___	_____
Sister	___	___	_____
Sister	___	___	_____
Sister	___	___	_____
Sister	___	___	_____

No known history _____

b. Have you ever been diagnosed as having hypercholesterolemia (above 240 mg/dl) ?

Yes No

2) Blood Pressure Status:

a. Is your blood pressure considered high for your age? (Please circle one)

Yes No Don't know

b. What is your usual blood pressure?

_____ /_____ (systolic/diastolic)

APPENDIX F (cont.)

3) Medication:

a. Are you taking any medication on a daily basis? (Please circle one)

Yes No

b. If Yes, what kind (please indicate name and purpose)

_____

4) Smoking Status:

a. Which of the following are you?

_____Non-smoker
_____Ex-smoker
_____Present smoker (pipe, cigar, cigarette)

b. If you are a present-smoker , which of the following are you? (please check)

_____Inhaler _____Filter smoker
_____Non-inhaler _____Non-filter smoker

c. If you are a cigarette smoker:

How many cigarettes do you smoke on a typical day?

How long have you been smoking?

5) Alcohol Consumption:

a. Do you drink alcohol? (Please circle one)

Yes No

b. If Yes, how much do you drink per week? (please be as specific as possible, for example: 3 12-oz cans of beers per week, or 3 4-oz glasses of wine, or 5 8-oz glasses of mixed drinks per week and so forth)

APPENDIX G

DIET HISTROY

1. Are you currently on any modified diet? (Please circle one)

Yes No

If No, please skip the following questions.
If Yes, plese complete the following questions.

Type of Diet	Purpose of Diet	For How Long

2. Where did you receive the instructions for this modified diet ?

_____Dietitian
_____Health Educator
_____Nurse
_____Doctor
_____Newspapers
_____Radio
_____Books (name them) _____
_____Magazines (name them) _____
_____TV program (name them) _____

3. If an individual instructed you on your modified diet, have you had followed up appointments with that individual? (Please circle one)

Yes No

If Yes, how often? _____

4. Is the modified diet served at your family meals? (Please circle one)

Yes No

If Yes, how often? _____

APPENDIX H

PHYSICAL ACTIVITY QUESTIONAIRE

Which of the following descriptions of physical activity best describes how you spend MOST of your time in a TYPICAL DAY. Please check only one letter (A, B, C, or D)

A) reading; knitting; doing office desk work or standing and moving around in the office; general laboratory work and light industrial work such as paring vegetables, cooking, and typing using a mechanical typewriter.

B) Light industrial work such as assembly work; driving a car; light housework such as dusting, scrubbing, setting a dinner table, and washing dishes; playing the piano or a stringed instrument.

C) Light janitorial work; housework such as bed-making, vacuuming, and scrubbing; playing tennis, swimming leisurely.

D) Heavy industrial work such as logging, mining, and construction work; cross-country running, weight lifting, swimming vigorously, dancing actively, country or folk style.

Additional Information

Please indicate ANY FORM of exercise you engage in REGULARLY. Also indicate how long you have been doing this (for example, swimming 3 times a week for the past 2 years, aerobic excercise 4 times a week for the past year, etc.). If none, please check here. [ ] NONE.

APPENDIX I

CONSENT FOR PARTICIPATION

I have received both verbal explanation and a written statement regarding this study and have been given the opportunity to ask questions concerning the procedures and the outcome. I understand the following:

Description of the Study

The focus of this study is on children whose parents have a serum total cholesterol level of 240 mg/dl or greater. The main purpose is to assess the health status of both these children and their parents. Specifically, the following characteristics of these families will be determined and compared: serum total cholesterol, high density lipoprotein cholesterol, low density lipoprotein cholesterol, very low density lipoprotein cholesterol, blood pressure, height, and weight. Questionaires will be used to obtain the following information: physical activity levels, smoking and drinking habits, and eating habits of both parents and children.

Experimental Design

The study will be carried out in two phases. The first pahse consists of a 30-40 minute orientation meeting at which all participants will receive a verbal explanation conerning the study. At the meeting, the participants' role will be outlined clearly and they will be given an opportunity to ask questions about the study. They will be asked to keep a four-day diet record and to respond to questionaires to collect the data outlined above. The second phase will involve: collection of at least 12 hour fasting blood sample and measurements of height, weight, and blood pressure. At this time, the parents and the children will return the completed diet records to the experimenter. Blood will be drawn by a licensed medical technician between 7 a.m. and 9 a.m. at Wallace Hall on the Virginia Tech campus.

APPENDIX I (cont.)

This study does not involve any monetary cost to the participants nor will they receive any monetary reward. However, both parents and children will benefit since they will be provided with detailed information regarding dietary patterns related to preventive aspects of coronary heart disease and the results of all blood analyses. No compensation will be offered should injuries occur as a result of participation in the study. The probability of such an occurence is very low. The identities of all participants will remain confidential as will all information obtained from the participants. Withdrawal from participation is permitted at any time.

Authorization

I authorize the release of medical information indicating my serum cholesterol to the investigators from the Department of Human Nutrition and Foods at Virginia Tech.

My signature and those of my children indicate aggreement to participate in the study described above.

Address:

_____

_____

_____

_____

Telephone: _____

Date: _____

Signature:

_____ (Parent)

_____ (Parent)

_____ (Child)

_____ (Child)

_____ (Child)

Investigators: Ai-Leng Ng, Graduate Student/HNF
Dr. Mary Ann Novascone,
Academic Advisor/HNF

APPENDIX J

DIETARY KNOWLEDGE TEST

MULTIPLE CHOICE

For questions #1-3, choose the ONE answer you think is correct:

1. According to the dietary guidelines of the American Heart Association, the total fat intake should be approximately ___% of total calories.

   a. 30% b. 35% c. 40%

2. According to the dietary guidelines of the American Heart Association, the intake of saturated fats (i.e., fats that stay solid at room temperature) should be approximately ___% of total calories.

   a. 10% b. 20% c. 30%

3. According to the dietary guidelines of the American Heart Association, the intake of polyunsaturated fats (i.e., fats that stay liquid at room and refrigerated temperatures) should be approximately ___% of total calories.

   a. 10% b. 35% c. 40%

TRUE/FALSE QUESTIONS

For questions #4-6 please indicate (TRUE) if you think the statement is true and indicate (FALSE) if you think the statement is false.

_____ 4. Dried beans and peas are meat substitutes since they provide protein and help reduce dietary fat intake.

_____ 5. Meats such as sausage, salami, and hot dogs are considered low fat meat products.

_____ 6. Using vegetable oil such as corn oil or sunflower oil in frying is highly recommended mainly because it has fewer calories than animal fats.

APPENDIX J (cont.)

PLEASE CHECK (X) THE FOOD ITEMS YOU THINK ARE:

7. HIGH in cholesterol

____Egg	____Oyster	____Mayonnaise
____Crab	____Cream	____Salmon
____Shrimp	____Vegetables	____Liver

8. HIGH in polyunsaturated fats

____Coconut oil	____Soybean oil
____Corn	____Sunflower oil
____Palm oil	____Vegetable shortening

9. HIGH in complex carbohydrates

____Gingersnaps	____Beans
____Potatoes	____Corn
____Oatmeal	____Oranges

10. HIGHLY recommended methods of preparing meats, fish, and poultry are:

____Deep-fat frying	____Roasting
____Baking	____Broiling
____Steaming	

APPENDIX K

BLOOD PRESSURES, BODY WEIGHT, AND TEST SCORES ON NUTRITIONAL KNOWLEDGE BASED ON AHA DIETARY GUIDELINES

Subject* code	Gender	Systolic/diastolic (mm Hg)		weight (kg)	test score (%)
11	M	113	83	90	90
12	F	118	94	68	50
13	M	116	98	76	45
21	M	130	90	108	83
22	F	95	66	64	95
23	M	110	59	70	64
24	M	103	56	49	55
31	M	126	99	112	67
32	F	110	77	64	57
33	M	111	80	60	55
34	F	120	84	61	69
41	M	131	89	110	53
42	F	111	71	82	52
43	M	132	68	69	55
44	F	99	55	58	45
45	F	102	58	48	48
51	M	138	99	115	60
52	F	126	79	62	65
53	F	107	62	62	40
54	F	98	55	53	30
61	M	134	84	91	60
62	F	128	80	88	45
63	F	99	59	41	40
64	M	138	73	78	48
71	M	147	93	84	88
72	F	118	81	104	63
73	M	116	78	61	40
74	F	118	69	51	70
75	M	122	64	58	50
81	M	131	91	87	81
82	F	115	73	60	60
83	M	123	58	70	57
84	F	105	55	34	45
91	M	117	77	78	65
92	F	115	75	62	58
93	M	148	82	79	38

APPENDIX K (cont.)

Subject* code	Gender	Systolic/diastolic (mm Hg)		weight (kg)	test score (%)
101	M	146	98	77	80
102	F	123	78	53	70
103	F	106	62	31	63
104	F	140	70	51	53
111	M	122	74	68	71
112	F	125	74	63	88
113	M	122	64	60	43
114	F	118	57	55	31
121	M	116	69	88	67
122	F	107	61	64	45
123	M	140	66	84	36
124	F	94	52	50	60
131	M	138	85	80	38
132	F	112	59	77	83
133	F	109	71	50	45
134	F	118	66	55	64
135	F	109	59	34	57
141	M	140	99	108	29
142	F	118	78	85	74
143	M	123	78	54	17
144	F	110	60	41	38

* the first digit of the subject code is the family of the participant and the second digit denotes the member in the family. The subject code 11 thus means that the participant is from family number 1 and the participant is the first member of the family. The first member is always the father, and the second member is the mother, and the rest are children.

APPENDIX L

SERUM LIPID PROFILES OF PARTICIPANTS

Subject* code	gender	HDL-C (mg/dL)	LDL-C (mg/dL)	VLDL-C (mg/dL)	TC (mg/dL)
11	M	36	156	28	221
12	F	44	164	68	277
13	M	37	147	31	216
21	M	31	204	51	287
22	F	63	88	14	166
23	M	34	105	22	162
24	M	58	115	12	186
31	M	36	185	44	266
32	F	52	135	14	202
33	M	41	96	15	153
34	F	38	121	26	186
41	M	39	189	76	305
42	F	58	159	17	235
43	M	47	89	24	161
44	F	65	116	18	200
45	F	-	-	-	-
51	M	33	190	60	284
52	F	49	93	56	199
53	F	45	168	24	238
54	M	33	87	19	140
61	M	40	172	30	243
62	F	54	82	14	151
63	F	52	69	13	135
64	M	42	102	141	173
71	M	32	74	74	247
72	F	41	142	26	209
73	M	33	150	29	212
74	F	43	117	25	186
75	M	28	86	23	138
81	M	42	119	49	211
82	F	54	105	35	195
83	M	41	104	13	159
84	F	48	170	16	235
91	M	34	107	22	164
92	F	48	104	24	177
93	M	45	176	35	257

APPENDIX L (cont.)

Subject* code	Gender	HDL-C (mg/dL)	LDL-C (mg/dL)	VLDL-C (mg/dL)	TC (mg/dL)
101	M	43	148	32	224
102	F	53	180	21	255
103	F	63	172	15	251
104	F	46	98	12	157
111	M	42	133	31	207
112	F	55	125	23	204
113	M	45	81	23	150
114	F	38	124	24	187
121	M	72	119	10	202
122	F	75	121	12	209
123	M	62	107	20	190
124	F	64	114	16	195
131	M	39	147	26	213
132	F	43	159	31	234
133	F	67	123	30	220
134	F	46	92	29	168
135	F	45	101	22	169
141	M	28	149	69	247
142	F	41	134	20	196
143	M	46	134	16	197
144	F	49	91	18	159

* Refer to Appendix k

APPENDIX M

INTAKES OF DIETARY LIPID COMPONENTS OF PARTICIPANTS

Subject* code	Kcal	Total fat (g)	Saturated fat (g)	MUFA (g)	PUFA (g)	Choles-terol (mg)
11	2816	106	34	35	25	273
12	1674	56	13	19	14	151
13	2032	75	25	24	16	342
21	2041	96	26	37	21	183
22	2204	121	34	36	27	158
23	3085	114	40	35	20	235
24	1811	87	28	29	16	153
31	2102	87	30	25	18	478
32	1402	49	17	16	7	154
33	1744	81	33	28	10	247
34	1156	42	14	15	6	112
41	1485	68	25	21	12	389
42	1110	45	14	15	11	189
43	2870	144	66	39	15	348
44	2244	103	37	32	17	389
45	2351	100	40	25	11	298
51	2134	85	30	28	15	352
52	1275	45	11	9	10	102
53	1110	46	20	18	5	87
54	1296	51	20	18	8	82
61	2699	115	42	37	22	241
62	1428	77	26	26	14	239
63	1592	53	23	14	8	147
64	2079	100	40	37	12	172
71	2138	91	36	32	13	221
72	1706	68	21	22	16	227
73	3655	152	53	35	15	302
74	2501	122	53	35	15	302
75	2064	87	34	30	12	366
81	2746	144	43	37	26	209
82	1815	70	29	24	8	183
83	3459	128	56	42	12	277
84	2051	66	30	21	8	195
91	2907	137	33	41	47	169
92	1934	89	23	21	16	162
93	3055	120	47	35	14	306

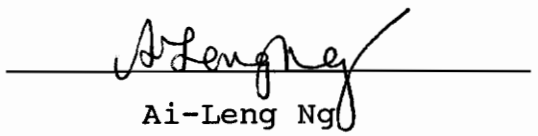
APPENDIX M (cont)

Subject* code	Kcal	Total fat (g)	Saturated fat (g)	MUFA (g)	PUFA (g)	Choles-terol (mg)
101	1536	61	18	18	16	207
102	2084	104	39	32	16	239
103	997	39	15	13	5	183
104	3216	127	59	39	13	537
111	1908	71	20	23	16	138
112	1154	50	18	14	13	223
113	1582	63	27	20	7	353
114	1811	59	23	18	7	307
121	2812	122	45	30	16	162
122	1627	65	23	19	14	139
123	2112	74	31	23	10	156
124	1578	54	23	16	5	75
131	1160	25	9	8	4	110
132	1426	61	13	23	18	83
133	1040	42	14	16	7	42
134	1698	55	15	16	5	255
135	1273	43	10	17	11	84
141	--	-	-	-	-	-
142	--	-	-	-	-	-
143	--	-	-	-	-	-
144	--	-	-	-	-	-

* Refer to Appendix K

VITA

Ai-Leng Ng was born in Mukah, Sarawak, on the island of Borneo. She received her Bachelor of Science degree in Foods and Nutrition from Seattle Pacific University in Seattle, Washington, in 1982. Prior to returning to graduate school, she had worked as a nutritionist for the First Tennessee Regional Health Office. She came to Virginia Tech for the Master of Science in Human Nutrition and Foods and completed the requirements for the degree in the spring of 1988. She is currently resuming her vocation as a nutritionist at the First Tennessee Regional Health Office.



Ai-Leng Ng