



6 JUL 1994

LIPID STATUS IN RAMATHIBODI HOSPITAL STAFF

PANITDA ACHARIYANONT

ถินันทนาการ

จาก

“มหัทธิทธิทธิทธิ”

**A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF
THE REQUIREMENTS FOR THE DEGREE OF
DOCTOR OF SCIENCE
(NUTRITION)**

**IN
FACULTY OF GRADUATE STUDIES
MAHIDOL UNIVERSITY**

1993

Copyright by Mahidol University

26969

Thesis
entitled

LIPID STATUS IN RAMATHIBODI HOSPITAL STAFF

Panitda Achariyanont

Panitda Achariyanont
Candidate

Vichai Tanphaichitr

Vichai Tanphaichitr, M.D., Ph.D., F.A.C.P., F.R.A.C.P.
Major Advisor

Ratana Pakpeankitvatana

Ratana Pakpeankitvatana, D.Sc.
Co-advisor

Preeya Leelahagul

Preeya Leelahagul, D.Sc.
Co-advisor

K. Kraisintu

Krisana Kraisintu, Ph.D.
Co-advisor

M. Chulasamaya

(var. 2001)

Monthree Chulasamaya, M.D., Ph.D.
Dean
Faculty of Graduate Studies

Vichai Tanphaichitr

Vichai Tanphaichitr, M.D., Ph.D., F.A.C.P., F.R.A.C.P.
Chairman
Doctor of Science Program in Nutrition
Faculty of Medicine, Ramathibodi Hospital

Thesis
entitled

LIPID STATUS IN RAMATHIBODI HOSPITAL STAFF

was submitted to the Faculty of Graduate Studies, Mahidol University
for the degree of Doctor of Science (Nutrition)

on

May 13, 1994

Panitda Achariyanont

Panitda Achariyanont
Candidate

Vichai Tanphaichitr

Vichai Tanphaichitr, M.D., Ph.D., F.A.C.P., F.R.A.C.P.
Chairman

Ratana Pakpeankitvatana

Ratana Pakpeankitvatana, D.Sc.
Member

Preeya Leelahagul

Preeya Leelahagul, D.Sc.
Member

K. Kraisintu

Krisana Kraisintu, Ph.D.
Member

Voravarn S Tanphaichitr

Voravarn S Tanphaichitr, M.D., M.S.
Member

M. Chulasamaya
(Vice Dean)

Monthree Chulasamaya, M.D., Ph.D.
Dean
Faculty of Graduate Studies

Athasit Vejjajiva

Athasit Vejjajiva, M.B., B.S., F.R.C.P.
Dean
Faculty of Medicine, Ramathibodi Hospital

BIOGRAPHY

NAME Panitda Achariyanont

DATE OF BIRTH April 27, 1952

PLACE OF BIRTH Nakornpratom, Thailand

INSTITUTIONS ATTENDED :

Kasetsart University, Faculty of Agriculture, Bangkok

April, 1974 Bachelor of Science in Home Economics

Mahidol University, Faculty of Graduate studies, Bangkok

August, 1977 Master of Science in Nutrition

ACKNOWLEDGEMENTS

I would like to express my most sincere gratitude to Professor Dr. Vichai Tanphaichitr, my advisor, for his competent supervision, guidance and encouragement during the entire period of the study and preparation of this thesis. My gratitude is also extended to Professor Dr. Voravarn S Tanphaichitr, Assistant Professor Dr. Ratana Pakpeankitvatana, Assistant Professor Dr. Preeya Leelahagul, and Dr. Krisana Kraisintu for their suggestions and valuable comments.

I am most sincerely thankful to Mr. Sompong Sombatwattanakul, Miss Kesanee Kuhathong and her staff of Clinical Research Ward, Ramathibodi Hospital for their valuable help and cooperation in this study.

Sincere appreciation is likewise extended to Mrs. Kannika Suwan, Miss Niparut Chairungruang, Mrs. Suda Sangplang, and Miss Sunadda Nimnoi for their assistances in statistical work.

My best thanks are given to Mrs. Narangsri Joijsin for her willingness and skill in typing and Miss Suthira Soipet, my classmate.

Finally, I would like to express my utmost gratitude to my parents and my husband for their perfect understanding and love encouragement and moral support.

ชื่อวิทยานิพนธ์ ภาวะโภชนาการด้านไขมันของเจ้าหน้าที่โรงพยาบาลรามารามาศี
ผู้วิจัย มานิตดา อัจฉริยานนท์
ปริญญา วิทยาศาสตร์ดุษฎีบัณฑิต (โภชนาการศาสตร์)
คณะกรรมการควบคุมวิทยานิพนธ์

วิจัย ต้นไมจิตร, พ.บ., Ph.D., F.A.C.P., F.R.A.C.P.

รัตน นากเนตรกิจวัฒนา, ทท.ด.

ปรีชา ลีนิพัทธ์, ทท.ด.

กฤษณา ไกรสินธุ์, Ph.D.

วันที่สำเร็จการศึกษา 13 พฤษภาคม พ.ศ. 2537

บทคัดย่อ

วัตถุประสงค์ของการศึกษานี้ คือ ประเมินปัจจัยต่างๆ ที่มีผลต่อระดับไลโปโปรตีนในซีรัมของเจ้าหน้าที่โรงพยาบาลรามารามาศี ผู้ชาย 88 คน ผู้หญิง 453 คน อายุ 19-61 ปี อายุมีความสัมพันธ์เชิงบวกกับระดับโคเลสเตอรอลทั้งหมด (TC), LDL-C, ไตรกลีเซอไรด์ (TG), ฟอสโฟไลปิด (PL) และ apo B ผู้หญิงมีระดับ HDL-C และ PL สูงกว่าแต่มีระดับ apo B ต่ำกว่าผู้ชาย ผู้ที่มีการสะสมไขมันภายในร่างกายมาก มีความสัมพันธ์เชิงบวกกับ TC, LDL-C, TG, apo B ในซีรัม และระดับ L-, M- และ S-particles ในพลาสมา แต่มีความสัมพันธ์เชิงลบกับระดับ HDL-C และ apo A-I ในซีรัม ผู้ที่เป็นโรคอ้วนลงพุงมีความสัมพันธ์เชิงบวกกับระดับ TG, apo B, L- และ M-particles ในเลือด แต่มีความสัมพันธ์เชิงลบกับระดับ HDL-C และ PL ในซีรัม ผู้ชายและผู้หญิงที่มีภาวะโปรตีนในปัสสาวะน้อยกว่าเกณฑ์มาตรฐานมีระดับ TC, LDL-C, TG, apo B, L-, M- และ S-particles ในเลือดต่ำกว่า แต่มีระดับ HDL-C และ apo A-I สูงกว่าผู้ที่มีภาวะโปรตีนในปัสสาวะน้อยกว่าปกติ

ผู้หญิงที่มีระดับโปรตีนในปัสสาวะน้อยกว่า 35 มก./ดล. มีระดับ TC, LDL-C, TG, PL, apo B, L- และ M-particles ต่ำกว่า แต่มีระดับ HDL-C สูงกว่าผู้หญิงที่มีโปรตีนในปัสสาวะมากกว่า ผู้หญิงที่มีระดับทรานส์เฟอรัลีนต่ำกว่า 2 กรัม/ลิตร มีระดับ TC, TG และ apo B ในซีรัมต่ำกว่าผู้หญิงที่มีระดับทรานส์เฟอรัลีนในซีรัมปกติ

ผู้หญิงที่มีระดับไขมันกลีโกลินต่ำกว่า 120 กรัม/ลิตร มีระดับ TC, HDL-C, TG, PL, apo B, L-, M-, และ S-particles ในเลือดต่ำกว่าผู้ที่ไม่มีภาวะโลหิตจาง

ผู้หญิงที่มีภาวะพร่องของการใช้กลูโคส มีระดับ TC, TG, PL, apo B และ M-particle ในเลือดต่ำกว่าผู้ที่มีระดับกลูโคสในเลือดปกติ ผู้หญิงที่เป็นโรคเบาหวานมีระดับ TG และ apo B ในซีรัมสูงกว่าผู้ที่มีระดับกลูโคสสูงกว่าปกติ

ผู้ที่มีความดันโลหิตสูง มีระดับ TC, LDL-C และ S-particle ในเลือดสูงกว่า แต่มีระดับ HDL-C และ apo A-I ในซีรัมต่ำกว่าผู้ที่มีความดันโลหิตปกติ

ระดับกรดไขมันในเลือดในซีรัมมีความสัมพันธ์เชิงบวกกับระดับ TC, LDL-C, TG, apo B, M- และ S-particles ในเลือด

ความขุ่นของ corneal arcus ในผู้ชาย 66 คน และผู้หญิง 453 คน เท่ากับร้อยละ 18.2 และ 10.2 ตามลำดับ ผู้ที่มี corneal arcus มีระดับ TC และ LDL-C สูงกว่าผู้ที่ไม่ได้มี corneal arcus

ความขุ่นของความผิดปกติของไขมันในเลือดชนิด IIa, IIb, และ IV ในผู้ชาย 66 คน เท่ากับร้อยละ 22.7, 8.1 และ 1.5 ตามลำดับ และในผู้หญิง 453 คนเท่ากับร้อยละ 36.0, 2.6 และ 0.9

ผู้ชาย 44 คน ในผู้ชายทั้งหมด 66 คน ต้มเห็ด และมีระดับ HDL-C และ apo A-I ในซีรัมสูง แต่ระดับ apo B ในซีรัมต่ำกว่าผู้ชาย 22 คน ที่ไม่ต้มเห็ด

กลุ่มผู้ชายที่สูบบุหรี่มีระดับ TC, LDL-C, apo A-I และ S-particle ในเลือดสูงกว่าผู้ชายที่ไม่สูบบุหรี่

ผู้ชาย 47 คน ที่ออกกำลังกายมีระดับ TC, LDL-C และ S-particle สูงกว่าผู้ชายที่ไม่ออกกำลังกาย ในผู้หญิง 270 คนที่ออกกำลังกายมีระดับ TC/HDL-C, LDL-C/HDL-C และ apo B ในซีรัมต่ำกว่าผู้หญิง 177 คนที่ไม่ออกกำลังกาย

การแนะนำให้ความคุมอาหารในผู้ที่มีระดับไขมันในเลือดผิดปกติ 109 คน พบว่ามีอัตราตอบสนองร้อยละ 13.8 เนื่องจากระดับกรดไขมันในเลือดไม่ได้เพิ่มขึ้นอย่างมีนัยสำคัญทางสถิติในผู้ที่มีระดับไขมันกลับเป็นปกติ แสดงว่าการลดลงของระดับ TC และ LDL-C ในซีรัมของบุคคลเหล่านี้จะเกิดจากกลไกอื่น

Thesis Title **Lipid Status in Ramathibodi Hospital Staff**

Name **Panitda Achariyanont**

Degree **Doctor of Science (Nutrition)**

Thesis Supervisory Committee

Vichai Tanphaichitr, M.D., Ph.D., F.A.C.P., F.R.A.C.P.

Ratana Pakpeankitvatana, D.Sc.

Preeya Leelahagul, D.Sc.

Krisana Kraisintu, Ph.D.

Date of Graduation **13 May B.E. 2537 (1994)**

ABSTRACT

The purpose of this study is to assess various factors influencing serum lipoprotein levels in Ramathibodi Hospital Staff (RHS), the prevalences of their dyslipoproteinemia, and the effects of dietary intervention on lipid status in RHS with dyslipoproteinemia. Sixty-six male RHS (MRHS) and 453 female RHS (FRHS), aged 19-61 yrs, participated in the study.

Age had positive correlations with serum total cholesterol (TC), LDL-C, triglyceride (TG), phospholipid (PL) and apo B levels. Total FRHS had significantly higher serum HDL-C and PL levels but significantly lower serum apo B levels than MRHS. Increased energy store assessed by body mass index, body fat in percent of body

weight or mid upper arm circumference (MUAC) positively correlated with serum TC, LDL-C, TG, apo B, plasma L-, M-, and S-particle levels but negatively correlated with serum HDL-C and apo A-I levels. Abdominal obesity assessed by waist-over-hip circumference ratio positively correlated with serum TG, apo B, plasma L- and M-particle levels but negatively correlated with serum HDL-C and PL levels.

Both MRHS and FRHS with inadequate somatic protein status, MUAC <90% std, had significantly lower serum TC, LDL-C, TG and apo B, as well as plasma L-, M- and S-particle levels but significantly higher serum HDL-C and apo A-I levels than those adequate somatic protein status.

FRHS with low serum RBP levels of <35 mg/dL had significantly lower serum TC, LDL-C, TG, PL and apo B as well as plasma L- and M-particle levels but significantly higher serum HDL-C level than FRHS with normal serum RBP levels. FRHS with low serum transferrin levels of < 2g/L also had significantly lower serum TC, TG and apo B levels than FRHS with normal serum transferrin levels.

Anemic FRHS (Hb <120 g/L) had significantly lower serum TC, HDL-C, TG, PL and apo B, as well as plasma L-, M- and S-particle levels than non-anemic FRHS.

FRHS with impaired glucose tolerance had significantly higher serum TC, TG, PL and apo B, as well as plasma M-particle levels than FRHS with euglycemia

whereas FRHS with diabetes mellitus had significantly higher serum TG and apo B levels than FRHS with euglycemia.

Subjects with definite hypertension had significantly higher serum TC, LDL-C and plasma S-particle levels but significantly lower serum HDL-C and apo A-I levels than normotensive subjects.

There were significantly negative correlations between serum linoleate levels and serum TC, LDL-C, TG, apo B, plasma M- and S-particle levels.

The prevalences of corneal arcus in 66 MRHS and 453 FRHS were 18.2 and 10.20%, respectively. This suggests the high prevalence of heterozygous familial hypercholesterolemia in Thai population, ie, 56: 500 in RHS. Subjects with corneal arcus had significantly higher serum TC and LDL-C levels than those without corneal arcus.

However, the prevalences of type IIa, IIb and IV hyperlipoproteinemias in 66 MRHS with were 22.7, 6.1, and 1.5% respectively, whereas the corresponding figures in 453 FRHS were 36, 2.6, and 0.9%.

Out of 66 MRHS, 44 were alcohol drinkers. They had significantly higher serum HDL-C and apo A-I but lower apo B levels than 22 non-drinkers. Male smokers had significantly higher serum TC, LDL-C, apo A-I and plasma S-particle levels than male non-smokers.

Forty-seven MRHS who exercised had significantly lower serum TC, LDL-C and plasma S-particle levels than 19 MRHS who did not exercised whereas 270 FRHS who exercised had significantly lower serum TC/HDL-C and LDL-C/HDL-C ratios as well as serum apo B level than 177 FRHS who did not exercise.

The responding rate to dietary advice in 109 dyslipidemia RHS was 13.8%. Though those becoming normolipidemia at the third visit had higher mean serum linoleate level than those with persistent dyslipidemia at the third visit, the difference did not reach statistical significance. Thus there might be other dietary factors lowering their serum TC and LDL-C levels.

TABLE OF CONTENTS

	PAGE
ABSTRACT	i
LIST OF ABBREVIATIONS	xi
LIST OF FIGURE	xiv
LIST OF TABLES	xv
CHAPTER	
I STATEMENT OF THE PROBLEM AND OBJECTIVES	1
II METERIALS AND METHODS	18
Subjects	18
Baseline information	18
Dietary assessment	18
Anthropometric assessment	19
Physical examination	19
Blood collection	20
Determination of serum lipoprotein	20
Preparation of FA methyl esters of total erythrocyte lipids	21
Preparation of FA methyl ester of total serum lipids	22
Determination of FA by the capillary GLC	22
Statistical analysis	23

III RESULTS	30
Baseline information	30
Dietary intake	30
Body composition	35
Influence of gender and age on serum lipids, apo, plasma lipoprotein and fibrinogen levels	40
Influence of body composition on serum lipids, apo, plasma lipoprotein and fibrinogen levels	54
Influence of anemia on serum lipids, apo, plasma lipoprotein and fibrinogen levels	76
Influence of blood glucose levels on serum lipids, apo, plasma lipoprotein and fibrinogen levels	81
Influence of blood pressure on serum lipids, apo, plasma lipoprotein and fibrinogen levels	85
Serum lipid, apo, plasma lipoprotein and fibrinogen levels in RHS by corneal arcus	94
Influence of levels of education and income on serum lipids, apo, plasma lipoprotein and fibrinogen levels	99
Influence of smoking habit on serum lipids, apo, plasma lipoprotein and fibrinogen levels	107
Influence of alcohol consumption on serum lipids, apo, plasma lipoprotein and fibrinogen levels	112
Influence of coffee consumption on serum lipids, apo, plasma lipoprotein and fibrinogen levels	116

PAGE

Influence of physical activity on serum lipids, apo, plasma lipoprotein and fibrinogen levels	121
Study in 241 dyslipidemic RHS	125
Effect of dietary intervention on lipid status in dyslipidemic RHS. Serum lipid, plasma lipoprotein and fibrinogen and apoprotein levels	130
Relative of FA composition of total serum and erythrocyte lipids	142
IV DISCUSSION	168
Population profile	168
Serum lipid profile	169
Factors affecting serum lipid and lipoprotein levels	171
Age	174
Gender	175
Obesity	176
Inadequate somatic protein status	178
Inadequate visceral protein status	179
Anemia	180
Blood glucose status	180
Blood pressure	182
Dietary factors	183
Linoleic acid	184
Alcohol	187

	PAGE
Coffee	188
Tobacco	188
Physical activity	189
Levels of education and income	190
Corneal arcus	191
Prevalences of dyslipidemia and lipoprotein phenotypes	192
Effects of dietary intervention on serum lipid status in dyslipopro- teinemic subjects	196
Plasma fibrinogen	197
IV SUMMARY	199
REFERENCES	207

LIST OF ABBREVIATIONS

ALB	Albumin
Apo	apoprotein
BHT	Butylated hydroxytoluene
BMI	Body mass index
CHD	Coronary heart disease
DBP	Diastolic blood pressure
DL	Deciliter
DM	Diabetes mellitus
EFA	Essential fatty acid
EGAT	Electricity Generating Authority of Thailand
F	Female
FA	Fatty acid
FBG	Fasting blood glucose
FRHS	Female Ramathibodi Hospital staff
g	Gram
GLC	Gas liquid chromatograph
Hb	Haemoglobin
HDL-C	High density lipoprotein-cholesterol
hr	Hour
IDL	Intermediate density lipoprotein
IGT	Impaired glucose tolerance
kcal	Kilocalorie
kg	Kilogram

LCAT	Lecithin: cholesterol acyltransferase
L	Liter
LDL-C	Low density lipoprotein-cholesterol
M	Male
mg	Milligram
mL	Milliliter
min	minute
MRHS	Male Ramathibodi Hospital staff
MUAC	Mid upper arm muscle circumference
MUFA	Monounsaturated fatty acid
N	Number
NEFA	Non essential fatty acid
NIDDM	Non-insulin-dependent diabetes mellitus
PBG	Postprandial blood glucose
PL	Phospholipid
PUFA	Polyunsaturated fatty acid
RBP	Retinal binding protein
RHS	Ramathibodi Hospital staff
SBP	Systolic blood pressure
SEM	Standard error of mean
SFA	Saturated fatty acid
SD	Standard deviation
std	Standard
TC	Total cholesterol
TG	Triglyceride

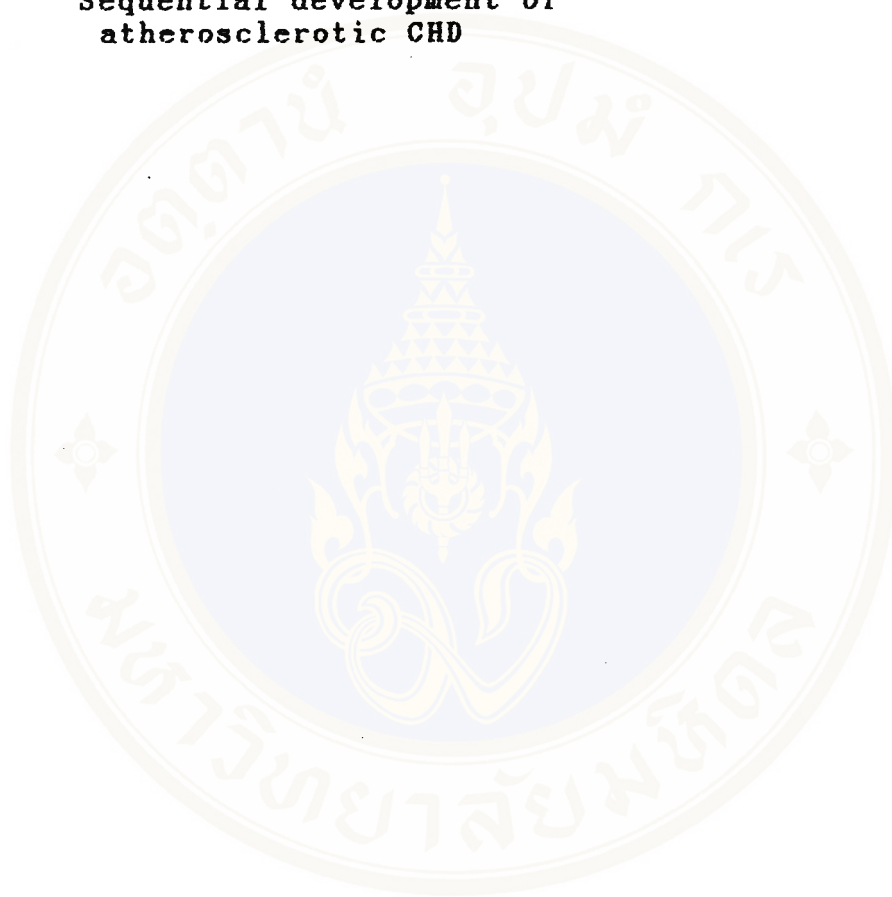
TRA	Transferrin
TST	Triceps skinfold thickness
UAMC	Upper arm muscle circumference
uL	Microliter
VLDL	Very low density lipoprotein
v/v	Volume/volume
WHR	Waist-over-hip circumference
wk	Week
yr	Year

LIST OF FIGURE

FIGURE

PAGE

- | | | |
|---|--|---|
| 1 | Sequential development of
atherosclerotic CHD | 7 |
|---|--|---|



LIST OF TABLES

TABLE		PAGE
1	Number and percentage of 519 RHS at the first visit by sex and age	25
2	Number and percentage of 513 RHS by sex and educational level	26
3	Number and percentage of 513 RHS by sex and income	27
4	Means±SEM of total energy, protein, fat and carbohydrate intake in 513 RHS by sex and age	28
5	Means±SEM of dietary energy distribution in 513 RHS by sex and age	29
6	Means±SEM of saturated, n-7 and n-9 fatty acid intake in 513 RHS by sex and age	30
7	Means±SEM of n-6 and n-3 fatty acid and cholesterol intakes in 513 RHS by sex and age	32
8	Means±SEM of fatty acid and cholesterol intakes in 66 MRHS by types of dyslipidemia	33
9	Means±SEM of fatty acid and cholesterol intakes in 447 FRHS by types of dyslipidemia	34
10	Means±SEM of height, weight, and BMI in 519 RHS by sex and age	36
11	Prevalences of underweight, acceptable weight, grades I and II obesity in 519 RHS by sex and age	37

TABLE

PAGE

12	Means±SEM of body fat in 519 RHS by sex and age	38
13	Means±SEM of triceps skinfold thickness, mid upper arm and upper arm muscle circumferences in 519 RHS by sex and age	39
14	Means±SEM of waist and hip circumferences and WHR in 519 RHS by sex and age	41
15	Prevalences of abdominal obesity in 519 RHS by sex and age	42
16	Means±SEM of anthropometric parameters in 519 RHS by sex and WHR	43
17	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and age	44
18	Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and age	46
19	Means±SEM of serum apoprotein A- I and apoprotein B levels in 519 RHS by sex and age	47
20	Number and percentage of 519 RHS by serum TC categories in each age group of each sex	49
21	Number and percentage of 519 RHS by serum LDL-C categories in each age group of each sex	50
22	Number and percentage of 519 RHS by serum HDL-C categories in each age group of each sex	51
23	Number and percentage of 519 RHS by serum TG categories in each age group of each sex	52

TABLE		PAGE
24	Number and percentage of 519 RHS by serum lipid categories in each age group of each sex	53
25	Means $\pm$ SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and BMI categories	55
26	Means $\pm$ SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and BMI categories	57
27	Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and BMI categories	59
28	Means $\pm$ SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and mid upper arm circumferences	61
29	Means $\pm$ SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and mid upper arm circumferences	62
30	Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and mid upper arm circumferences	64
31	Means $\pm$ SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and mid upper arm muscle circumferences	65
32	Means $\pm$ SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and mid upper arm muscle circumferences	66
33	Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and mid upper arm muscle circumferences	68

TABLE

PAGE

34	Means $\pm$ SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and triceps skinfold thickness	69
35	Means $\pm$ SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and triceps skinfold thickness	70
36	Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and triceps skinfold thickness	71
37	Means $\pm$ SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and WHR	73
38	Means $\pm$ SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and WHR	74
39	Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and WHR	75
40	Means $\pm$ SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and visceral protein status	77
41	Means $\pm$ SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and visceral protein status	78
42	Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and visceral protein status	79
43	Means $\pm$ SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 506 RHS by sex and Hb levels	80

TABLE

PAGE

44	Means±SEM of plasma lipoprotein and fibrinogen levels in 506 RHS by sex and Hb levels	82
45	Means±SEM of serum apoprotein A-I and apoprotein B levels in 506 RHS by sex and Hb levels	83
46	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and PBG levels	84
47	Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and PBG levels	86
48	Means±SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and PBG levels	87
49	Age and anthropometric parameters in 453 FRHS by PBG criteria	88
50	Serum lipid levels, serum lipid ratios, plasma lipoprotein, fibrinogen level, serum apoprotein levels, serum uric acid, serum creatinine, FBG, PBG and blood pressure levels in 453 FRHS by PBG criteria	89
51	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and blood pressure categories	90
52	Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and blood pressure categories	92
53	Means±SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and blood pressure categories	93

TABLE		PAGE
54	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and corneal arcus	95
55	Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and corneal arcus	96
56	Means±SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and corneal arcus	97
57	Means±SEM of age and anthropometric levels in 513 RHS by sex and corneal arcus	98
58	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and educational level	100
59	Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and educational level	101
60	Means±SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and educational level	102
61	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and income	103
62	Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and income	105
63	Means±SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and income	106
64	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and smoking habit	108

TABLE

PAGE

65	Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and smoking habit	109
66	Means±SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and smoking habit	110
67	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and intensity of smoking	111
68	Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and intensity of smoking	113
69	Means±SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and intensity of smoking	114
70	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and alcohol consumption	115
71	Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and alcohol consumption	117
72	Means±SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and alcohol consumption in each sex	118
73	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and coffee consumption	119
74	Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and coffee consumption	120

TABLE

PAGE

75	Means±SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and coffee consumption	122
76	Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and exercise	123
77	Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and exercise	124
78	Means±SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and exercise	126
79	Prevalences of dyslipidemia and lipoprotein phenotypes in 241 RHS by sex and age	127
80	Age and anthropometric parameter in 229 FRHS by dyslipidemic criteria	128
81	Serum lipid levels, serum lipid ratios, plasma lipoprotein and fibrinogen levels, and serum apoprotein levels in 229 FRHS by dyslipidemic criteria	
82	Relative FA composition of total serum lipids in 229 FRHS by dyslipidemic criteria	131
83	Prevalences of dyslipidemic types in 110 RHS on the first visit by sex and age	132
84	Prevalences of dyslipidemic types in 110 RHS on the second visit by sex and age	134
85	Prevalences of dyslipidemic types in 110 RHS on the third visit by sex and age	135

TABLE

PAGE

86	Number and percentage of 110 dyslipidemic RHS on dietary treatment by serum total choles- terol levels in each sex and age groups	136
87	Number and percentage of 110 dyslipidemic RHS on dietary treatment by serum triglyceride levels in each sex and age group	137
88	Means±SEM of serum lipid levels and TC/HDL-C and LDL-C/HDL-C ratios in 110 dyslipidemic RHS on dietary treatment	138
89	Means±SEM of serum lipoprotein and plasma fibrinogen levels in 110 dyslipidemic RHS on dietary treatment	140
90	Means±SEM of serum apoprotein levels in 110 dyslipidemic RHS on dietary treatment	141
91	Relative FA composition of total serum lipids in 110 dyslipidemic RHS on dietary treatment	143
92	Relative FA composition of total erythrocyte lipids in 110 dyslipidemic RHS on dietary treatment	144
93	Relative FA composition of total serum lipids in 110 dyslipidemic RHS at the third visit by dyslipidemic criteria	145
94	Relative FA composition of total erythrocyte lipids in 110 dyslipidemic RHS at the third visit by dyslipidemic criteria	147

TABLE

PAGE

95	Relative FA composition of total serum lipids in 9 MRHS at the third visit by dyslipidemic criteria	148
96	Relative FA composition of total erythrocyte lipids in 9 male RHS at the third visit by dyslipidemic criteria	149
97	Relative FA composition of total serum lipids in 101 FRHS at the third visit by dyslipidemic criteria	150
98	Relative FA composition of total erythrocyte lipids in 101 F RHS at the third visit by dyslipidemic criteria	151
99	Relationship between serum TC, TG or HDL-C levels in mmol/L and other serum lipid levels, serum lipid ratios, serum apoprotein, plasma lipoprotein and fibrinogen levels in 519 RHS	152
100	Relationship between age in yrs and serum lipid levels, serum lipid ratios, serum apoprotein, plasma lipoprotein and fibrinogen levels in 519 RHS	153
101	Relationship between BMI in kg/m ² and serum lipid levels, serum lipid ratios, serum apoprotein, plasma lipoprotein and fibrinogen levels in 519 RHS	154
102	Relationship between body fat in percent of body weight and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RHS	155

TABLE

PAGE

103	Relationship between body fat in kg and serum lipid levels, serum lipid ratios, serum apoprotein levels, and plasma lipoprotein levels in 519 RHS	156
104	Relationship between mid upper arm circumferences in cm and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RHS	157
105	Relationship between mid upper arm muscle circumferences in cm and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RHS	158
106	Relationship between triceps skinfold thicknesses in mm and serum lipid levels, serum lipid ratios, serum apoprotein levels, and plasma lipoprotein and fibrinogen levels in 519 RHS	159
107	Relationship between waist or hip circumferences in cm and serum lipid levels, serum lipid ratios, and plasma lipoprotein levels in 519 RHS	160
108	Relationship between WHR and serum lipid levels, serum lipid ratios, serum apoprotein, and plasma lipoprotein levels in 519 RHS	161
109	Relationship between serum linoleate levels and serum lipid levels, serum lipid ratios, serum apoprotein, and plasma lipoprotein levels in 241 RHS	162

TABLE

PAGE

110	Relationship between serum transferrin (TRA) levels in g/L or RBP in mg/dL and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RHS	163
111	Relationship between Hb levels in g/L and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RHS	164
112	Relationship between systolic blood pressure in mmHg and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RHS	165
113	Relationship between diastolic blood pressure in mmHG and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RHS	166
114	Relationship between FBG and serum lipid levels, lipid ratios, apoprotein levels in 519 RHS	167
115	Guidelines for the diagnosis of dyslipidemia	193
116	NCEP Expert Panel's Criteria for dyslipidemia	194

CHAPTER I

STATEMENT OF THE PROBLEM AND OBJECTIVES

Role of lipids

When lipids are liquid at room temperature, they are called oils, and when they are solid at room temperature, they are called fats. Fats and oils can be either visible or invisible to the human eyes. The so-called invisible fats are those dispersed within a food, such as that within nuts, whole milk and egg yolks, whereas the visible fats such as butter or vegetable oil are readily apparent to the consumer. In western population, about 60% of their dietary fat comes from animal sources and the remainder from vegetable sources, primarily vegetable oils.¹

In the human body, lipid has many important roles. Quantitatively, the most important role is that of fuel, since lipid provides twice the energy of carbohydrate and protein. One g of fat supplies 9 kcal, while 1 g of protein or carbohydrate provides 4 kcal. Lipid can be stored in almost unlimited quantity. All cells of the body, except for those of the central nervous system and the erythrocytes, are able to use fatty acids as a source of energy.²

A growing incidence of coronary heart disease (CHD) has prompted epidemiological surveys of risk factors among our population. Since increased levels of serum total cholesterol (TC) and triglyceride (TG) have been identified as associated risk factors, much attention has been focused on dietary lipids and their role in the causation, treatment, and prevention of CHD.^{3,4}

Functions of dietary lipids

The major substance of dietary lipids is TG which has various functions summarized below.

Source of energy One g of fat, whether animal or vegetable, liquid or solid, provides 9 kcal, 2.25 times as much energy as an equal weight of either carbohydrate or protein.²

Satiety value Fat tends to leave the stomach slowly, being released approximately 35 hrs after ingestion. This slow emptying time of the stomach helps to delay the onset of hunger pangs and contributes to a feeling of satiety after a meal.

Carrier of fat-soluble vitamins The 4 fat-soluble vitamins: A, D, E and K, require dietary fat as a carrier for their intestinal absorption.

Source of essential fatty acids (EFAs) Various vegetable oils provides different amount and type of EFAs. Soybean oil provide both linoleic acid and alpha-linolenic acids: 53% and 7% of total fatty acids (FAs).⁵⁻⁷

Palatability The role of fat in contributing to the palatability of food is appreciated best by those forced to exist on a low-fat diet. The use of fat for frying food, as a spread, as a base of salad dressing, and as a flavor adjunct for vegetables, does much to improve the taste appeals of meals. It has been suggested that dietary fat stimulates the flow of digestive juices.

Functions of body lipids

Lipids are ubiquitous constituents of body tissues and fluids with various functions summarized below.⁸ The major type of body lipids is also TG consisting of one molecule of glycerol and 3 molecules of FAs.

Energy reserve Body fat represents the primary form in which energy is stored in the body. Since it is an essential constituent of the cell membrane, all tissues contain some fat. In addition, the body has a group of specialized cells called adipose cells, whose main function is the storage of fat. Once fat has been formed and deposited in the adipose tissues, the body has no way of excreting it. Thus body fat can be reduced only by

oxidizing or burning it as a source of energy when caloric intake is less than caloric expenditure. A certain amount 15% for men, is considered normal and desirable. Fat reserve excess of this represents, at first, overweight and in extreme cases, obesity with all the associated physical, physiological, and esthetic disadvantage.²

Body regulator As an essential constituent of the membrane of each individual cell, fat helps to regulate the uptake and excretion of various nutrients by the cell.

Insulation Subcutaneous fat, fat deposit beneath the skin, serves as an insulating material for the body, and protects the body against shock from changes in environmental temperature. A certain minimum layer is desirable to prevent excessive heat loss from the body. However, if the layer is too thick it slows down the rate of heat loss during hot weather, resulting discomfort to the individual.

Protection of vital body organs The fat deposits that surround certain vital organs serve to hold them in position and shield them from physical shock. The kidneys and heart are protected in this way. These are the last fat deposits to be reduced when there is a caloric deficit.

Precursors of eicosanoids Polyunsaturated fatty acids (PUFAs) of n-3 and n-6 families are the precursors of eicosanoids which function in various processes in the body.⁵

Blood lipids and the risk of CHD

Although most cells have the capacity to synthesize and degrade lipids, the arrangement of tissues into organs has necessitated a specific transport system for lipids in the form of the lipoproteins. These components are particularly important because of their intrinsic function as a transport system, being subject to many metabolic defects, and particularly playing an important role in the development of atherosclerotic heart disease.^{8,9}

CHD accounts for about one-quarter of all deaths worldwide, which is the highest proportion of all causes of death. They are sometimes referred to as "killer number one". In developed countries, about one-half of all deaths are caused by CHD. Although in developing countries the proportion of CHD as a cause of death is estimated to be only about 16% (in third place after respiratory and infectious disease).¹⁰ In Thailand, an agricultural and developing country, the occurrence of CHD among urban population seems to be progressively increased each year. This is, at least, due to the change of life style towards westernized-society pattern.^{4,11}

A number of risk factors have been identified including dyslipidemia, hypertension, obesity, cigarette smoking, diabetes mellitus (DM), sedentary living,

personality factors, certain pattern of behavior, and a family history of premature clinical atherosclerosis.¹¹ However, one of the most clearly established factors that numerous published studies have pointed out the strong correlation with CHD, is serum lipid levels.^{4,12} An elevated serum cholesterol level is one of the principle risk factors of CHD.^{13,14}

Sequential development of CHD

The sequential development of CHD is schematically shown in Figure 1.

Several steps in Figure 1 deserve emphasis.¹⁵ First, the production of clinically recognized disease is the result of a sequence of pathogenic events. Secondly, the rates of conversion from one step to another may vary greatly from person to person and may possibly be independent of one another. Thirdly, the probability of the conversion may be influenced by one or more risk factors. Fourthly, clinically recognized disease takes several forms of these clinical manifestations: chest pain (angina pectoris), myocardial infarction, and sudden death. Fifthly, the modification of risk factors causing an asymptomatic stenosis to become symptomatic would probably also be important in the secondary prevention of myocardial infarction or death in a person with angina or prior myocardial infarction.

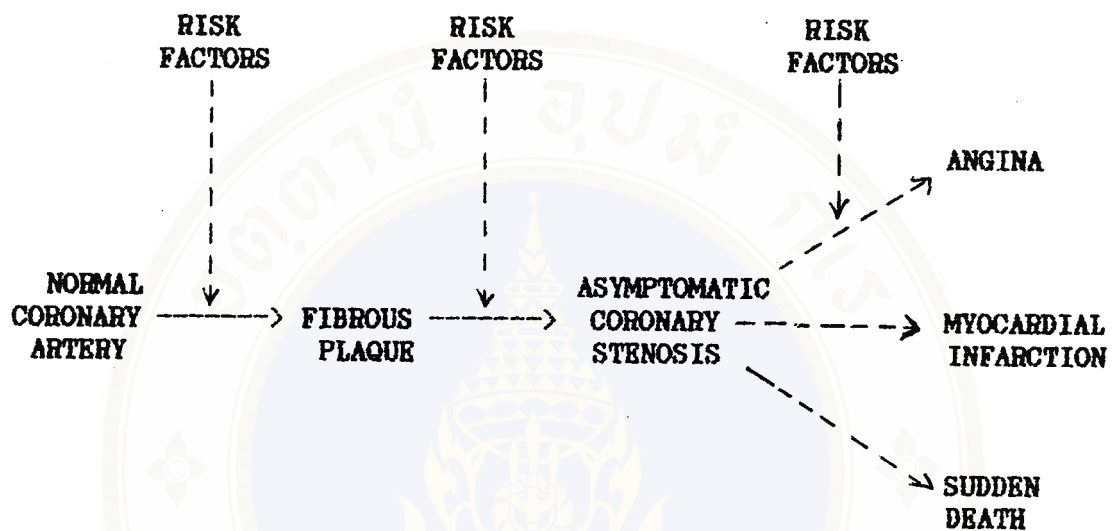


Fig. 1 Sequential development of atherosclerotic CHD.

Atherosclerosis

Coronary atherosclerosis, the major cause of CHD, is characterised histologically by the accumulation of lipid, predominantly cholesterol, in the arterial wall together with a local connective tissue reaction.¹⁶ Four points related to atherosclerotic lesions are brought up here.

Both the early and advanced lesions of atherosclerosis consist of smooth muscle cells and macrophages. Macrophages predominate in fatty streaks, which may represent a form of inflammatory response, whereas advanced lesions contain a fibrous cap of smooth muscle that covers proliferated smooth muscle and macrophages and variable numbers of other types of leukocytes, including lymphocytes.

Diet-induced hypercholesterolemia leads to changes in endothelium and monocytes that result in increased monocyte adherence, subendothelial migration, accumulation of lipid to form foam cells, and gradual accumulation of smooth muscle to form fatty streaks.^{17,18}

All four principle cells are involved in atherosclerosis, ie, endothelial cells, smooth muscle cells, platelets and monocytes/macrophages. For instance, platelets contain or can synthesize and release

chemoattractants and growth factors, at least one of which is identical or very closely related to platelet-derived growth factor (PDGF). It is thus possible that *injury* or other stimulus may induce growth-factor release and autocrine or paracrine stimulation of cell in the artery.

A modified response to injury hypothesis of atherogenesis suggests that at least two pathways may lead to formation of intimal smooth muscle proliferation lesions. The first pathway, demonstrated in hypercholesterolemia, involves monocyte and possibly platelet interactions, which may stimulate fibrous plaque formation by growth-factor release from the different cells. The second pathway involves direct stimulation of endothelium, which may release growth factors that can induce smooth muscle migration and proliferation. This pathway may be important in diabetes mellitus, hypertension, cigarette smoking, or other circumstance associated with an increased incidence of atherosclerosis.^{19,20}

Risk factors

Scientific studies in laboratory, clinic, and population have pointed to the contributing roles of dyslipidemia²¹⁻²⁹ including high serum TC, low serum high density lipoprotein-cholesterol (HDL-C) and high serum triglyceride (TG) levels; hypertension,^{30,31} cigarette

smoking,^{21,22,28,32,33} overweight,^{28,34-36} DM, and impaired glucose tolerance (IGT),^{28,37,38} physical inactivity^{28,39} in the genesis of CHD.^{21,22} The risk factors represent statistical diagnosis which are accurate for large populations but which may have less predictive value for individuals.

Cholesterol Even a single measurement of serum TC is significantly predictive of the risk of CHD in epidemiological studies. This reflects the risk conferred by low density lipoprotein-cholesterol (LDL-C) and intermediate density lipoprotein-cholesterol (IDL-C). The relationship between CHD and plasma cholesterol levels is continuous, graded and curvilinear. It is consistent in many studies of single population and in comparisons between populations.²⁸

CHD risk spans a very wide range of serum TC levels, increasing progressively from levels of about 3.5 mmol/L; the rise in risk becomes increasingly steep in the upper limit of the distribution. Among usually observed levels of TC, there is no threshold at which risk begins.

HDL-C It is also now recognized that low levels of serum HDL-C are an independent and powerful predictor for CHD incidence.²⁹ This inverse association is particularly powerful in women. Low HDL-C often reflects obesity,

cigarette smoking, lacked of physical exercise and IGT, and may also be inherited.

In the Framingham Heart Study, the Lipid Research Clinics (LRC) Mortality Followup Study, the LRC Coronary Primary Prevention Trial, and the Multiple Risk Factor Intervention Trial (MRFIT), quantitative analysis of the data was consistent with a 2 to 3% decrease in CHD risk for each 1 mg/dL increase in HDL-C level, after adjustment to control for other risk factors. Follow-up extended from 6 to 10 yrs, and similar results were found in men and women. In some, but not all trials of lipid lowering there has been a statistical association between rising levels of HDL-C and decreasing incidence of CHD. It must be emphasized that there is no evidence from a specifically designed trial to show that treating initial low levels of HDL-C will lessen risk of CHD; subject analysis of one drug trial has, however, suggested that increasing HDL-C is associated with reduction of risk.²⁸

Triglyceride There is increasing evidence, though less strong than that for serum TC that elevated serum TG levels, when accompanied by a low HDL-C are predictive of increased CHD incidence. It is not clear as yet whether the TG level is independently predictive or whether the association is dependent on coexisting factors. But it has emerged from recent studies that a high TG when

associated with a low HDL-C level, even without markedly elevated LDL-C, reflects a cluster of findings that is predictive of high risk. This cluster often includes truncal obesity, hypertension, IGT, and hyperinsulinemia with insulin resistance.²⁸

Hypertension Systolic, diastolic and pulse pressures increase with age, and all have been incriminated as risk factors for CHD.³⁰ A positive association has been reported between hypertension and elevated serum TC levels, DM, obesity and body weight. In the Framingham Study, the subsequent stroke rate in subjects with borderline diastolic blood pressures of 90-94 mmHg was three times that of subjects with diastolic pressures under 90 mmHg; CHD was increased 50% in this group.³¹

Cigarette smoking Proppective studies consistently show that cigarette use is directing related to CHD risk both in men and women.²⁹ The Framingham Study has revealed that in men, the incidence of CHD was twofold greater in cigarette smokers than in nonsmokers, and fourfold greater in heavy smokers; in women, the rate of CHD was lower than in men, but was commensurately higher when the smoking pattern was similar to that of men; the risk of developing CHD increased with the duration (in years) of cigarette smoking; and the cessation of smoking;

lead to CHD death rate that was substantially lower in the stopped smokers than in continuing smokers.^{32,33}

The two major cigarette components are nicotine and carbon monoxide that can be hazardous to a heart already in jeopardy because of a narrowed, compromised coronary circulation. The tendency of inhaled tobacco smoke to increase platelet adhesiveness, to raise carboxyhaemoglobin levels and to increase myocardial irritability could precipitate a myocardial infarction or sudden death.³³

Overweight Keys and Fidanza³⁴ found that subjects with CHD had higher serum TC levels and greater relative body weight than control subjects. Osterman et al³⁵ reported that patients with obesity and hypercholesterolemia favorable changes in cholesterol profile following weight loss, and that relative reduction of cholesterol levels depended on initial levels. Other have also noted an association between body weight or body mass index (BMI) with serum TC.³⁶ The International Task Forces for the Prevention of Coronary Heart Disease has recently stated that weight control contributes to the prevention and reduction of high blood pressure, high serum TC and TG, prevention or correction of low HDL-C and to the prevention of non-insulin-dependent diabetes mellitus (NIDDM), which are commonly associated with

overweight; in addition obesity is an independent risk factor for CHD, at least in persons aged less than 50 yrs, especially when it is of truncal distribution.²⁸

DM and IGT DM has an aggravating effect on atherosclerosis and will thereby enhance the development of CHD. There is an association between CHD and DM and IGT.³⁷ DM and IGT are both age dependent. An integral relationship exists between abnormalities of insulin and glucose metabolism and the etiology and clinical course of hypertension.³⁸ Moreover, several antihypertensive drugs adversely affect glucose, insulin and lipid metabolism.²²

Weight control is of central importance in the prevention of NIDDM, especially in persons with a family history of this disorder. This is particularly necessary because of the cardiovascular risk possibly conferred by IGT, which may be operative years before DM becomes manifest.²⁸

Stress Epidemiologic studies of coronary risk show stress (particularly time: urgency, competitive, drive, frustration, and somewhat hostile attributes) as a factor. Castelli³¹ found that coronary patients were those person who constantly worked at maximum capacity, incessantly striving for success in professional, commercial or political life. Lately it has been stated that therapy designed to alter such behaviors improves risk.

Physical inactivity Sedentary habits are associated with increased risk of CHD. There is a considerable consensus that lack of exercise is an independent source of CHD risk, interacting with other risk determinants.²⁸

Exercise certainly changes a number of coronary risk factors for the better, including LDL-C, very low density lipoprotein-cholesterol (VLDL-C) and HDL-C levels, blood pressure, weight, and depression.³⁹ Exercise trials in postmyocardial infarction patients generally show no benefit in life expectancy but great benefit in lifestyle and sense of well-being, often reflected in an increased return to work. These trials show no danger from exercise; although death rates were not statistically lower in the patients who exercised, they were generally lower in the group undergoing exercise intervention.³⁹

Alcohol consumption Alcohol consumption also influences the occurrence of CHD. A slightly lower risk of CHD in high-to moderate drinkers than in abstainers was shown in a number of observational studies in Israel, Scotland, the USA and Yugoslavia.⁴⁰ However, a study from the United Kingdom suggests that this association can be partly or wholly explained by the inclusion in the group of abstainers of ex-drinkers who had stopped drinking for health reasons.⁴¹

Most reports have shown that moderate to excessive alcohol consumption leads to increased blood lipid level, especially TG levels, in chronic alcoholics and in patients with type IV hyperlipoproteinemia.⁴² Though alcohol ingestion does cause a favorable rise in serum HDL-C level many epidemiological studies show that moderate and heavy drinkers have higher blood pressure than non-drinkers, and that abstinence from alcohol is followed by a fall in blood pressure.⁴⁰

Genetic factors A familial tendency to complication of severe atherosclerosis has long been recognized clinically. Genetic factors also appear to determine blood cholesterol, blood pressure, blood glucose levels, body build and obesity.¹³ Familial hypercholesterolemia and hereditary connective tissue disorders could aggravate the effects of atherosclerosis. If heredity plays an important role in these risk factors, the genetic disorders are more likely to become manifest in subjects in younger population groups usually studies clinically.⁴³

Objectives

The Division of Nutrition and Biochemical Medicine, Faculty of Medicine, Ramathibodi Hospital has shown high prevalences of CHD risk factors in urban Thais including 2,703 male and 792 female officials of

Electricity Generating Authority of Thailand (EGAT), aged 35-54 yrs. Fifty-two and 18% of men had serum TC levels of 200-260 and greater than 260 mg/dL, respectively, whereas the corresponding figures for women were 50 and 15%. Nineteen and 23% of men had serum TG levels of 150-200 mg/dL and greater than 200 mg/dL, respectively, whereas the corresponding figures for women were 9 and 6%.^{44,45} Thus it is the purpose of this study to assess various factors influencing serum lipoprotein levels in Ramathibodi Hospital staff (RHS) and the prevalences of dyslipoproteinemia

Specific aims

The specific aims of the study are

1. to assess the influences of age, gender, body composition, visceral protein status, anemia, blood glucose status, blood pressure, dietary factors, smoking habit, levels of education and income, and inherited traits on serum lipoprotein and plasma fibrinogen levels in RHS,
2. to determine the prevalences of dyslipoproteinemia in RHS and
3. to assess the effects of dietary intervention on lipid status in RHS with dyslipoproteinemia.

CHAPTER II

MATERIALS AND METHODS

Subjects

Questionnaires were sent to 3,977 RHS, aged 19-61 yrs, requesting them to take part in the investigation for nutritional risk factors. The responding rate to participate in the study was 13.1%. The subjects consisted of 66 males (MRHS) and 453 females (FRHS).

Baseline information

The following baseline informations were obtained from each individual: personal history, educational attainment, title of position, monthly income, and habits of food intake, smoking and drinking.

Dietary assessment

Each subject was asked to record their 24-hr dietary intake prior to the first visit. However, the data were available from 513 subject (98.84%) for the evaluation by a nutritionist on the day of the first visit. The data were calculated for dietary energy intake and its distribution derived from protein, fat and carbohydrate, amount of animal and vegetable protein, and FA intake in each subject were then calculated.⁴⁶⁻⁵¹

Anthropometric assessment

After a 12-14 hr fast, height, body weight, mid upper-arm circumference (MUAC), triceps skinfold thickness (TST), and waist and hip circumferences in each subject were measured by the standard technique.⁵²⁻⁵⁴

Upper arm muscle circumference (UAMC) was calculated from the following formula⁵²: $UAMC = MUAC - 3.1416 \times TST$. Standards of TST, MUAC, and UAMC were taken from the standard source.^{52,53} BMI⁵⁵ was calculated from body weight in kg/(height in m)². Waist-over-hip circumference ratio (WHR) was computed. Body fat was measured by using infrared light absorption analysis with the Futrex 5000-A Body Composition Analyzer (Futrex Inc, Gaithersburg, MD, USA).⁵⁶

Physical examination

At each visit, every subject was physically examined by Professor Vichai Tanphaichitr whereas blood pressure was measured by nurses of Clinical Research Ward, Faculty of Medicine, Ramathibodi Hospital, Bangkok, Thailand, using a sphygmomanometer.

Blood collection

Venous blood was obtained from each patient after a 12-14 hr fast. Whole blood, serum or plasma was appropriately prepared for biochemical determination.

Determination of serum lipoproteins

Serum TC, free cholesterol and TG were measured by enzymatic-colorimetric methods.⁵⁷⁻⁵⁹ Serum HDL-C was also measured by the enzymatic-colorimetric method as the determination of serum TC⁵⁷ after precipitating chylomicron, VLDL and LDL by adding phosphotungstic acid and magnesium ions to the serum.^{60,61} LDL-C was calculated from Friedewald's formula⁶²: $\text{LDL-C in mmol/L} = \text{TC} - (\text{TG}/2.2) - \text{HDL-C}$. Then serum TC/HDL-C and LDL-C/HDL-C ratios were calculated.

Serum apoprotein (apo) A-I and apo B were measured by immunochemical reaction with specific antibodies.⁶³

Plasma L-, M- and S-particles (equivalent to chylomicron, VLDL, and LDL, respectively) and fibrinogen were determined by micronephelometric method.⁶⁴ Serum phospholipid (PL) was measured by Bartlett's method.⁶⁵

Determination of FA composition of total serum and erythrocyte lipids

Preparation of FA methyl esters of total erythrocyte lipids^{66,67}

Four hundred μL of erythrocytes suspended in isotonic NaCl were added into Teflon-lined screw-cap test tubes containing of 100 μL of pentadecanoic acid in methanol (1 mg/mL) used as an internal standard and 100 μL of butyrate hydroxytoluene (BHT) (4 g of BHT/L of methanol) used as an antioxidant. After adding 2 mL of solution containing 6 N HCl in methanol (1:5 v/v), the samples were transesterified by heating the tubes at 90°C for 2.5 hrs under nitrogen. Lipids were extracted by shaking with 4 mL of hexane and 100 μL of amyl alcohol (1-pentanol) followed by centrifugation at 800 g for 5 min. After evaporation under nitrogen, the FAs were methylated by heating with 0.5 mL of boron trifluoride in methanol 14 g/L at 60°C for 10 min. The FA methyl esters were extracted into 5 mL of hexane, washed twice with saturated NaCl solution 400 g/L and dried with anhydrous sodium sulfate. After evaporation under nitrogen, the residue was resuspended in 200 μL of hexane and stored in sealed ampoules at -20°C until analysis was performed by capillary gas liquid chromatograph (GLC).

Preparation of FA methyl esters of total serum lipids

Two hundred μL of serum were added to teflon-lined screw-cap test tubes containing 100 μL of pentadecanoic acid used as the internal standard. Two mL of methanol-hexane 4:1 (v/v) was added to the serum samples which were then mixed by Vortex mixer vigorously. The amount of 0.2 mL of acetyl chloride was slowly added to the stirring tube over the period of 1 min. Tubes were tightly closed with Teflon-lined caps and subjected to methanolysis at 100°C for 1 hr. After tubes had been cooled at room temperature, 5 mL of 6% K_2CO_3 solution was slowly added to stop reaction and neutralized the mixture. The tubes was then shaken vigorously and centrifuged at 2,000 rpm for 5 min and an aliquot of the hexane upper phase was injected into the capillary GLC.

Determination of FA by the capillary GLC

The fatty acid methyl esters were separated by the capillary GLC (Model 5890 Gas Chromatographic System, Palo Alto, California: Hewlette-Packard Co.) with a 12 m x 0.2 mm x 0.33 mm film thickness of fused-silica capillary column and a flame ionization detector. Nitrogen was used as a carrier gas at the rate of 39 mL per min. The column temperature was programmed from 150°C to 170°C at

2°C per min, from 170°C to 192°C at 1°C per min, and from 192°C to 200°C at 10°C per min with a final hold of 15 min. The detector temperature was 250°C.

The emergent peaks were identified by comparing their retention time with the standard FA methyl esters. The relative proportion of FA was derived by determining the area under each peak divided by the total area under each peak of all FAs appeared in the chromatogram. The value was expressed as percent of total FAs.

Statistical Analysis

Statistical analysis was performed by the Statistical Package for the Social Sciences (SPSS^x)⁶⁸ on an IBM computer. The Means and standard error of means (SEM) of various parameters were calculated. Comparisons were made between 2 groups by Student's t test (2-tailed) and among the groups, by the analysis of variance. Pearson product moment correlations were used in evaluating the relative importance of correlation between various parameters. The significance of prevalences of various parameters were tested by chi-square test.

CHAPTER III

RESULTS

Baseline information

Table 1 shows number and percentage of 519 RHS at the first visit by sex and age which was categorized into 4 groups: 19-29, 30-39, 40-49 and 50-61 yrs. Only 4 women were 19 yrs old and 1 women was 61 yrs old. Men had lower education level (Table 2) and income (Table 3) than women.

Dietary intake

Table 4 shows that FRHS had lower energy, protein, fat, and carbohydrate intakes than MRHS with the corresponding age groups. However, their dietary energy distributions were not significantly different (Table 5).

Table 6 shows that FRHS 19-29, 40-49 yrs old and total FRHS had significantly lower total saturated fatty acid (SFA) intake than men with the corresponding age groups, FRHS 40-49 yrs old had significantly lower 16:0 intake than MRHS with the same age group, and total FRHS had significantly lower 16:0 and 18:0 than total men. FRHS 19-29 and 40-49 yrs old and total FRHS had significantly lower total monounsaturated fatty acid (MUFA) intake than MRHS with the corresponding age groups, and FRHS 19-29, 30-39 and 40-49 yrs old and total FRHS

Table 1. Number and percentage of 519 RBS at the first visit by sex and age

Age yr	Male		Female		Total	
	n	% [*]	n	% [*]	n	% ⁺
19-29	22	33.3	97	21.4	119	22.9
30-39	22	33.3	154	34.0	176	33.9
40-49	12	18.2	158	34.9	170	32.8
50-61	10	15.2	44	9.7	54	10.4
Total	66	100	453	100	519	100

*Percentage of total males or females.

+Percentage of total males and females.

Only 4 females were 19 yrs old.

Only 1 female was 61 yrs old.

Table 2. Number and percentage of 513 RHS by sex and educational level

Education level	Male		Female		Total	
	n	% [*]	n	% [*]	n	% ⁺
School	58	88.0	221	49.5	279	54.4
Primary	35	53.1	102	22.8	137	26.7
Secondary	23	34.9	119	26.7	142	27.7
College	2	3.0	23	5.0	25	4.9
Vocational	2	3.0	23	5.0	25	4.9
University	6	9.0	203	45.5	209	40.7
Bachelor degree	3	4.5	142	31.8	145	28.3
Master degree	1	1.5	53	11.9	54	10.5
Doctorate degree	2	3.0	8	1.8	10	1.9
Total	66	100	447	100	513	100

*Percentage of total males or females.

⁺Percentage of total males and females.

Table 3. Number and percentage of 513 EHS by sex and income

Income baht/month	Male*		Female*		Total	
	n	%	n	%	n	%
≤3000	35	53.0	80	17.9	115	22.4
3001-5000	13	19.7	110	24.6	123	24.01
5001-7000	14	21.2	96	21.5	110	21.4
≥7001	4	6.1	161	36.0	165	32.2
Total	66	100	447	100	513	100

*P value <0.0005 by chi-square test.

Table 4. Means±SEM of total energy, protein, fat and carbohydrate intake in 513 RHS by sex and age

Sex	Age	N	Energy	Protein			Fat			Carbohydrate
				Animal	Vegetable	Total	Animal	Vegetable	Total	
yr				g/caput/day						
M	20-29	22	1,749±99	29.8±2.6	19.5±2.3	50.6±3.1	29.9±2.8	25.7±2.5	55.6±3.4	246.1±24.6
	30-39	22	1,748±97	27.6±2.4	20.1±1.5	47.7±2.3	30.3±3.3	27.7±2.2	58.1±3.5	265.2±18.3
	40-49	12	1,582±139	29.7±3.9	18.3±1.6	48.2±5.2	24.3±5.5	27.0±3.4	51.4±4.0	227.9±22.3
	50-59	10	1,630±183	24.1±5.7	18.6±2.6	42.6±7.1	34.3±5.5	17.2±3.8	51.4±5.0	244.7±33.0
	Total	66	1,700±57	28.2±1.6	19.3±1.0	48.0±1.8	29.7±1.9	25.3±1.4	55.6±1.9	248.9±11.6
F	19-29	94	1,258±34	20.3±0.9	15.5±0.8	35.8±1.1	23.5±1.4	20.1±1.0	43.6±1.5	183.8±6.5
	30-39	152	1,302±25	22.0±0.7	15.0±0.4	37.1±0.8	23.5±1.0	22.3±0.9	45.9±1.4	184.3±4.2
	40-49	157	1,294±29	21.0±0.8	15.9±0.6	37.0±1.0	22.1±1.0	23.7±0.9	45.7±1.5	185.3±5.2
	50-61	44	1,282±35	20.2±1.4	15.3±0.7	35.4±1.4	22.9±2.0	22.7±1.8	44.1±2.1	184.1±5.5
	Total	447	1,291±15	21.1±0.4	15.5±0.3	36.6±0.5	22.9±0.6	22.4±0.5	45.2±0.8	184.5±2.7

Comparisons were made within the same sex: significant difference from 19-29 yr: p<0.02; b1 c1

significant difference from 30-39 yr: p<0.02; significant difference from 40-49 yr: p<0.02.

Significant difference from males with the corresponding age groups: p<0.0005, p<0.002, p<0.005, p<0.05, p<0.0005, p<0.005, p<0.05, p<0.005. A1 A2 A3 A4 B1 B2 B3 C1 D1

Significant difference from total males: p<0.0005, p<0.002. F1 F2

Table 5. Means±SEM of dietary energy distribution in 513 EHS by sex and age

Sex	Age	N	Energy distribution						
			Protein			Fat			Carbohydrate
			Animal	Vegetable	Total	Animal	Vegetable	Total	
yr			<----- % of total energy ----->						
M	20-29	22	7.2±0.7	4.3±0.3	11.5±0.5	16.1±1.6	13.8±1.4	30.0±2.0	58.0±2.2
	30-39	22	6.5±0.6	4.7±0.4	10.9±0.5	15.5±1.4	15.1±1.5	29.8±1.6	58.6±1.8
	40-49	12	7.5±0.7	4.6±0.2	12.2±0.7	13.4±2.6	16.2±2.0	29.6±1.2	57.3±1.1
	50-60	10	5.9±1.3	4.5±0.3	10.3±1.1	18.8±2.6	10.3±2.4	29.1±2.2	59.4±2.9
	Total	66	6.8±0.4	4.5±0.2	11.3±0.3	15.8±0.9	14.1±0.8	29.7±0.9	58.3±1.0
F	19-29	94	6.6±0.3	4.7±0.2	11.3±0.3	16.9±0.9	14.6±0.8	31.5±1.0	57.0±1.0
	30-39	152	6.9±0.2	4.6±0.1	12.2±0.7	16.0±0.6	15.7±0.6	31.7±0.7	56.6±0.7
	40-49	157	6.8±0.3	4.9±0.1	11.6±0.2	15.5±0.7	16.7±0.6	32.1±0.8	56.6±0.8
	50-61	44	6.3±0.4	4.8±0.2	11.2±0.4	15.7±1.3	16.3±1.5	30.5±1.0	57.7±1.0
	Total	447	6.7±0.1	4.8±0.1	11.4±0.1	16.0±0.4	15.9±0.4	31.7±0.4	56.8±0.4

Table 6. Means±SEM of saturated, n-7 and n-9 fatty acid intake in 513 RHS by sex and age

Sex	Age	N	SFAs	14:0	16:0	18:0	MUFAs	16:1 n-7	18:1 n-9	20:3 n-9
yr <----- g/d ----->										
M	20-29	22	12.2±2.2	2.2±1.7	8.5±1.9	4.6±1.8	15.9±2.3	2.6±1.7	13.8±2.1	2.1±1.7
	30-39	22	9.7±1.1	0.6±0.1	6.2±0.7	2.6±0.3	13.1±1.3	0.7±0.2	11.6±1.0	0.4±0.1
	40-49	12	12.5±2.1	1.0±0.3	7.3±1.1	3.2±0.6	15.3±2.1	0.9±0.2	12.5±1.8	0.4±0.1
	50-60	10	10.6±2.2	0.6±0.3	6.5±1.2	3.1±0.6	13.1±2.0	0.9±0.6	11.6±1.7	0.6±0.1
	Total	66	11.2±1.0	1.2±0.6	7.2±0.6	3.5±0.6	14.5±1.0	1.4±0.1	12.5±0.9	1.0±0.6
F	19-29	94	6.9±0.4	0.6±0.1	4.6±0.3	2.0±0.1	9.2±0.5	0.6±0.1	8.3±0.4	0.4±0.1
	30-39	152	8.2±0.4	0.8±0.1	5.2±0.2	2.3±0.1	10.7±0.4	0.7±0.1	9.3±0.4	0.4±0.1
	40-49	157	7.9±0.3	0.8±0.1	5.0±0.3	2.0±0.1	10.3±0.4	0.6±0.1	9.3±0.4	0.5±0.1
	50-61	44	8.4±1.2	0.7±0.2	4.9±0.4	2.3±0.3	10.0±0.7	0.5±0.1	9.1±0.7	0.3±0.04
	Total	447	7.8±0.2	0.7±0.05	5.0±0.1	2.2±0.1	10.1±0.2	0.6±0.04	9.1±0.2	0.4±0.04

Comparisons were made within the same sex: significant difference from 19-29 yr: p<0.01, p<0.02, p<0.05; b1

significant difference from 30-39 yr: p<0.05.

Significant difference from males with the corresponding age group: p<0.01, p<0.02, p<0.05, p<0.005, B2 C1 C2 C3

p<0.05, p<0.005, p<0.02, p<0.05. F1 F2 F3 F4

Significant difference from total males: p<0.0005, p<0.002, p<0.005, p<0.05.

had significantly lower 18:1 n-9 intake than MRHS with the corresponding age groups. FRHS 30-39 yrs old had significantly higher SFA and MUFA intakes than FRHS 19-29 yrs old whereas FRHS 50-61 yrs old had significantly lower 16:1 n-7 intake than FRHS 30-39 yrs old.

Table 7 shows that FRHS 19-29 yrs old and total FRHS had significantly lower PUFA and 18:2 n-6 intake than MRHS with the corresponding age groups whereas FRHS 19-29 and 30-39 yrs old and total FRHS had significantly lower cholesterol intake than MRHS with the corresponding age groups. FRHS 30-39 yrs old had significantly lower 18:2 n-6 intake than FRHS 19-29 yrs old whereas FRHS 40-49 yrs old had significantly lower 18:2 n-6 and 18:3 n-3 intake than FRHS 19-29 yrs old.

Table 8 shows that type IIa hyperlipoproteinemic MRHS had significantly lower total PUFA and 18:2 n-6 intakes than normolipidemic MRHS whereas type IIb hyperlipoproteinemic MRHS had significantly lower total SFA and 16:0 intake than normolipidemic MRHS.

Table 9 shows that type IIb hyperlipoproteinemic FRHS had significantly lower 16:0, total MUFA, 18:1 n-9, 18:3 n-3 and 22:6 n-3 intakes than normolipidemic FRHS.

Table 8. Means±SEM of fatty acid and cholesterol intakes in 66 MRHS by types of dyslipidemia

Fatty acid	Normal	TC≥5.2 LDL-C≥3.4 & TG<2.3 mmol/L	TG≥2.3 mmol/L only	TC≥5.2, LDL-C≥3.4 & TG≥2.3 mmol/L	HDL-C<0.9 mmol/L only	Total dyslipidemia
	n=39	n=15	n=1	n=4	n=7	n=27
SFAs (g)	11.4±1.3	10.3±1.6	13.7	6.2±0.6 ^{a1}	14.0±3.8	10.8±1.4
14:0 (g)	1.5±0.9	0.6±0.2	1.6	1.1±0.6 ^{a1}	1.0±0.3	0.8±0.2
16:0 (g)	7.8±1.1	6.4±0.9	5.6	3.6±0.4 ^{a1}	8.1±2.0	6.4±0.8
18:0 (g)	3.8±1.0	2.9±0.5	3.1	1.4±0.2 ^{a1}	3.8±1.1	2.9±0.4
MUFAs (g)	15.3±1.4	12.9±1.7	10.5	8.3±1.5	17.2±3.5	13.2±1.4
16:1 n-7 (g)	1.9±1.0	0.8±0.2	0.7	0.6±0.4	1.1±0.3	0.8±0.1
18:1 n-9 (g)	13.2±1.2	11.3±1.4	7.8	7.1±1.7	14.9±2.9	11.5±1.2
20:3 n-9 (g)	1.4±0.9	0.4±0.1 ^{a3}	0.3	0.8±0.3	0.5±0.2	0.5±0.1
PUFAs (g)	15.7±1.4	11.4±1.4 ^{a3}	13.2	11.4±2.9	15.2±3.5	12.4±1.3
18:2 n-6 (g)	13.8±1.3	9.9±1.2	11.2	9.9±2.6	13.3±3.1	10.8±1.1
18:3 n-3 (g)	2.7±1.0	1.3±0.2	1.5	1.4±0.3	4.6±3.2	2.2±0.8
20:4 n-6 (g)	1.1±0.9	0.1±0.02	0.08	0.1±0.03	0.5±0.4	0.2±0.1
20:5 n-3 (g)	1.0±0.9	0.05±0.02	0.14	0.04±0.03	0.1±0.02	0.1±0.01
22:6 n-3 (g)	1.1±0.9	0.1±0.03	0.7	0.2±0.1	0.2±0.1	0.2±0.04
PUFAs/SFAs	1.7±0.1	1.3±0.2	0.9	1.8±0.5	1.8±0.5	1.5±0.2
PUFAs/MUFAs/SFAs	1.7:1.4:1	1.3:1.3:1	0.9:0.8:1	1.8:1.4:1	1.8:1.4:1	1.5:1.3:1
Cholesterol (mg)	337.6±42.5	348.6±77.0	229.5	147.6±12.8	430.6±165.5	335.6±61.8

Significant difference from normal: ^{a1} p<0.002, ^{a2} p<0.02, ^{a3} p<0.05.

Table 9. Means±SEM of fatty acid and cholesterol intakes in 447 FHHS by types of dyslipidemia

Fatty acid	Normal	TC>5.2 mmol/L only	TC>5.2 & LDL-C >3.4 mmol/L	TG>2.3 mmol/L only	TC>5.2, LDL-C>3.4 & TG>2.3 mmol/L	HDL-C<0.9 mmol/L only	Total dyslipidemia
	n=260	n=18	n=143	n=4	n=12	n=10	n=187
SFAs (g)	7.9±0.3	12.7±2.7	7.3±0.3	7.7±1.6	6.8±0.7	7.5±1.4	7.8±0.4
14:0 (g)	0.8±0.1	1.7±0.6	0.7±0.1	0.4±0.2	0.6±0.1	0.5±0.1	0.8±0.1
16:0 (g)	5.0±0.2	6.5±0.8	4.8±0.2	5.3±1.2	4.0±0.5	4.7±0.8	4.9±0.2
18:0 (g)	2.2±0.1	3.4±0.6	2.1±0.1	1.8±0.4	1.8±0.2	2.2±0.5	2.2±0.1
MUFAs (g)	10.3±0.4	11.4±1.0	9.9±0.4	12.1±2.9	8.2±0.8	10.2±1.4	10.0±0.3
16:1 n-7 (g)	0.6±0.1	1.1±0.4	0.5±0.03	0.9±0.4	0.4±0.1	0.4±0.1	0.6±0.1
18:1 n-9 (g)	9.2±0.3	9.8±0.9	8.9±0.4	10.5±2.5	7.3±0.7	9.6±1.2	8.9±0.3
20:3 n-9 (g)	0.5±0.1	0.6±0.4	0.3±0.02	0.8±0.5	0.3±0.1	0.3±0.04	0.4±0.04
PUFAs (g)	12.1±0.5	12.2±2.1	11.7±0.5	17.0±6.7	9.9±1.2	12.9±1.4	11.8±0.5
18:2 n-6 (g)	10.6±0.5	10.8±1.9	10.2±0.5	15.1±6.0	8.1±1.0	11.3±1.3	10.4±0.4
18:3 n-3 (g)	1.5±0.1	1.8±0.5	1.4±0.1	21.1±0.9	1.2±0.1	1.5±0.2	1.4±0.1
20:4 n-6 (g)	0.2±0.1	0.5±0.4	0.1±0.01	0.1±0.02	0.1±0.01	0.1±0.01	0.1±0.04
20:5 n-3 (g)	0.1±0.05	0.4±0.4	0.04±0.01	0.05±0.02	0.03±0.01	0.02±0.01	0.1±0.04
22:6 n-3 (g)	0.2±0.05	0.5±0.4	0.1±0.01	0.2±0.2	0.1±0.03	0.04±0.02	0.1±0.04
PUFAs/SFAs	1.7±0.1	1.3±0.2	1.8±0.1	2.0±0.3	1.6±0.2	2.1±0.3	1.8±0.1
PUFAs/MUFAs/SFAs	1.7:1.4:1	1.3:1.4:1	1.8:1.4:1	2.0±1.5:1	1.6:1.2:1	2.1:1.4:1	1.8:1.4:1
Cholesterol (mg)	119.7±9.7	193.8±30.1	191.8±14.24	332.4±101.04	168.1±56.0	182.6±40.2	192.9±12.2

Significant difference from normal: ^{al} p<0.05.

Body composition

Table 10 shows that women had significantly lower height than men with the corresponding age groups and women 19-29 and 30-39 yrs old had significantly lower body weight than men of the same age. In women, those 50-60 yrs old had the lowest height whereas but highest body weight and BMI increased with age. Only men 50-59 yrs old had significantly higher BMI than men 30-39 yrs old.

Table 11 shows that in 453 FRHS the prevalences of grade II under weight, grade I underweight, acceptable weight, grade I obesity and grade II obesity based on BMI of <18.5, 18.5-19.9, 20.0-24.9, 25.0-29.9 and ≥ 30.0 kg/m², respectively, were 7.7, 13.0, 52.3, 23.2 and 3.8%, respectively, whereas in 66 MRHS the corresponding prevalences were 4.5, 13.6, 63.6, 15.2 and 3.0%. The prevalences of acceptable weight in FRHS decreased significantly with age.

Table 12 shows that women had significantly higher body fat expressed as kg and/or % of body weight than men with the same age. In women, their body fat expressed as kg and/or % body weight increased with age.

Table 13 shows that women had significantly higher TST but lower MUAC and UAMC than men with the same age. In women, their TST, MUAC and UAMC also increased with age.

Table 10. Means $\pm$ SEM of height, weight, and BMI in 519 RHS by sex and age

Sex	Age	N	Height	Weight	BMI
	yr		m	kg	kg/m ²
M	20-29	22	1.64 $\pm$ 0.01	60.0 $\pm$ 2.5	22.2 $\pm$ 0.9
	30-39	22	1.67 $\pm$ 0.01	60.7 $\pm$ 1.9	22.1 $\pm$ 0.6
	40-49	12	1.65 $\pm$ 0.02	64.1 $\pm$ 3.6	23.5 $\pm$ 1.1
	50-60	10	1.62 $\pm$ 0.02	63.3 $\pm$ 3.1	24.1 $\pm$ 0.8
	Total	66	1.65 $\pm$ 0.01	61.8 $\pm$ 1.3	22.7 $\pm$ 0.4
F	19-29	97	1.56 $\pm$ 0.01 ^{A1}	51.7 $\pm$ 0.8 ^{A2}	21.3 $\pm$ 0.3
	30-39	154	1.56 $\pm$ 0.01 ^{B1}	54.6 $\pm$ 0.8 ^{a4,B2}	22.8 $\pm$ 0.3 ^{a2}
	40-49	158	1.55 $\pm$ 0.01 ^{C1}	56.6 $\pm$ 0.64 ^{a1,b4}	23.6 $\pm$ 0.3 ^{a1,b4}
	50-61	44	1.53 $\pm$ 0.01 ^{a3,b2,c2,D1}	59.5 $\pm$ 1.3 ^{a1,b3,c3}	25.5 $\pm$ 0.5 ^{a1,b1,c1}
	Total	453	1.55 $\pm$ 0.002 ^F	55.1 $\pm$ 0.4 ^F	23.0 $\pm$ 0.2

Comparisons were made within the same sex:

significant difference from 19-29 yr: ^{a1} ^{a2} $p < 0.0005$, $p < 0.001$,
^{a3} ^{a4} $p < 0.002$, $p < 0.005$;

significant difference from 30-39 yr: ^{b1} ^{b2} $p < 0.0005$, $p < 0.002$,
^{b3} ^{b4} $p < 0.005$, $p < 0.05$;

significant difference from 40-44 yr: ^{c1} ^{c2} ^{c3} $p < 0.002$, $p < 0.02$, $p < 0.05$.

Significant difference from males with the corresponding

^{A1} ^{A2} ^{B1} ^{B2} age groups: $p < 0.0005$, $p < 0.005$, $p < 0.0005$, $p < 0.005$,
^{C1} ^{D1} $p < 0.002$, $p < 0.0005$.

^F Significant difference from total males: $p < 0.0005$.

Table 11. Prevalences of underweight, acceptable weight, grades I and II obesity in 519 RHS by sex and age

Sex	Age	N	BMI, kg/m ²											
			<18.5		18.5-19.9		20-24.9		25-29.9		≥30		n	%
yr			n	%	n	%	n	%	n	%	n	%		
M	20-29	22	2	9.1	4	18.2	12	54.5	3	13.6	1	4.5		
	30-39	22	1	4.5	3	13.6	16	72.7	2	9.1	-	-		
	40-49	12	-	-	1	8.3	8	66.7	2	16.7	1	8.3		
	50-59	10	-	-	1	10.0	6	60.0	3	30.0	-	-		
	Total	66	3	4.5	9	13.6	42	63.6	10	15.2	2	3.0		
* F	19-29	97	14	14.4	21	21.6	51	52.6	9	9.3	2	2.1		
	30-39	154	16	10.4	19	12.3	86	55.8	29	18.8	4	2.6		
	40-49	158	3	1.9	18	11.4	85	53.8	43	27.2	9	5.7		
	50-61	44	2	4.5	1	2.3	15	34.1	24	54.5	2	4.5		
	Total	453	35	7.7	59	13.0	237	52.3	105	23.2	17	3.8		

*P value <0.0005 by chi-square test

Table 12. Means $\pm$ SEM of body fat in 519 RHS by sex and age

Sex	Age	N	Body fat	
			% of body weight	kg
M	20-29	22	24.7 $\pm$ 1.2	15.1 $\pm$ 1.3
	30-39	22	26.5 $\pm$ 1.2	16.5 $\pm$ 1.1
	40-49	12	27.8 $\pm$ 1.6	18.1 $\pm$ 1.9
	50-59	10	29.6 $\pm$ 0.9	18.8 $\pm$ 1.3
	Total	66	26.6 $\pm$ 0.7	16.7 $\pm$ 0.7
F	19-29	97	36.2 $\pm$ 0.4	18.7 $\pm$ 0.4
	30-39	154	37.8 $\pm$ 0.3	21.1 $\pm$ 0.5
	40-49	158	38.8 $\pm$ 0.3	22.1 $\pm$ 0.4
	50-61	44	39.4 $\pm$ 0.6	23.5 $\pm$ 0.8
	Total	453	38.0 $\pm$ 0.2	21.2 $\pm$ 0.2

Comparisons were made within the same sex:

al
significant difference from 19-29 yr: $p < 0.0005$

a2 a3
 $p < 0.001$, $p < 0.02$;

b1 b2
significant difference from 30-39 yr: $p < 0.01$, $p < 0.01$.

Significant difference from males with the corresponding

A1 A2 B1 B2
age groups: $p < 0.0005$, $p < 0.002$, $p < 0.0005$, $p < 0.002$

C1 D1 D2
 $p < 0.0005$, $p < 0.0005$, $p < 0.005$.

F1
Significant difference from total males: $p < 0.0005$.

Table 13. Means±SEM of triceps skinfold thickness, mid upper arm and upper arm muscle circumferences in 519 EHS by sex and age

Sex	Age	N	TST	MUAC		UAMC	
	yr		mm	%std	cm	%std	cm
M	20-29	22	8.7±0.9	69.8±7.4	27.2±0.7	92.8±2.3	24.5±0.5
	30-39	22	8.9±0.9	71.3±7.1	27.4±0.6	93.4±1.9	24.6±0.5
	40-49	12	9.7±1.2	77.8±9.3	27.8±1.1	94.8±3.6	24.7±0.9
	50-59	10	10.5±0.9	84.2±7.0	28.8±0.8	98.4±2.6	25.5±0.8
	Total	66	9.2±0.5	73.9±3.8	27.6±0.4	94.2±1.2	24.7±0.3
F	19-29	97	17.6±0.6	106.4±3.8	24.7±0.3	86.8±1.1	19.3±0.2
	30-39	154	20.8±0.6	125.8±3.3	26.5±0.3	92.9±1.0	19.9±0.2
	40-49	158	22.1±0.5	134.0±3.2	27.4±0.2	96.0±0.8	20.4±0.2
	50-61	44	23.5±1.1	142.2±6.5	28.8±0.5	101.1±1.6	21.5±0.4
	Total	453	20.8±0.3	126.1±2.0	26.6±0.2	93.5±0.6	20.1±0.1

Comparisons were made within the same sex: significant difference from 19-29 yr: p<0.005, p<0.02; b1 b2 b3

significant difference from 30-39 yr: p<0.005, p<0.02, p<0.05; significant difference from 40-44 yr: p<0.005, c1

Significant difference from males with the corresponding age groups: p<0.0005, p<0.002, p<0.0005, A1 A2 A3 B1
C1 D1 D2
p<0.0005, p<0.0005, p<0.05.

Significant difference from total males: p<0.0005, p<0.05. F1 F2

Table 14 shows that except those 50-61 yrs old, women had significantly lower WHR than men with the same age and WHR of both sexes increased with age.

Table 15 shows that the prevalences of abdominal obesity in 66 men (WHR >1.0) and 413 women (WHR >0.8) were 1.5 and 54.1%, respectively, and the prevalences of abdominal obesity in women increased significantly with age.

Table 16 shows that women without abdominal obesity had significantly lower WHR, BMI, MUAC and UAMC but higher percent body fat than men without abdominal obesity. Women and combined men and women with abdominal obesity had higher WHR, % body fat, BMI, MUAC and UAMC than the corresponding groups without abdominal obesity. Except UAMC in combined men and women, the differences in the other parameters reached statistical significance.

Influence of gender and age on serum lipid, apo, plasma lipoprotein and fibrinogen levels

Table 17 shows that in 66 MRHS, those with 50-60 yrs old had the highest levels of serum TC, LDL-C, TC/HDL-C and LDL-C/HDL-C ratios whereas serum HDL-C levels in men with 30-39 and 40-49 yrs old and serum TG levels in men 40-49 yrs old were significantly lower and higher than men 20-29 yrs old, respectively. In 453 FRHS, women with 50-

Table 14. Means $\pm$ SEM of waist and hip circumferences and WHR in 519
RHS by sex and age

Sex	Age	N	Circumference		WHR
			Waist	Hip	
	yr		<----- cm ----->		
M	20-29	22	73.59 $\pm$ 2.18	88.49 $\pm$ 1.53	0.83 $\pm$ 0.01
	30-39	22	76.68 $\pm$ 1.70 a2,b4	90.17 $\pm$ 5.74	0.86 $\pm$ 0.01 a1,b2
	40-49	12	83.14 $\pm$ 3.06 a2	91.33 $\pm$ 2.04	0.91 $\pm$ 0.02 a1,b1
	50-59	10	83.83 $\pm$ 1.43	91.35 $\pm$ 1.48	0.92 $\pm$ 0.01
	Total	66	77.91 $\pm$ 1.19	90.00 $\pm$ 0.78	0.87 $\pm$ 0.01
F	19-29	97	69.34 $\pm$ 0.80 A2 a2	89.82 $\pm$ 0.61 a2	0.77 $\pm$ 0.01 A1 a1,B
	30-39	154	74.66 $\pm$ 0.75 a2,b3	92.81 $\pm$ 0.53 a2	0.80 $\pm$ 0.01 a1,b1,C
	40-49	158	79.30 $\pm$ 0.70 a2,b3,C2	93.89 $\pm$ 0.46 a2,b3	0.84 $\pm$ 0.01 a1,b1,c1
	50-61	44	85.53 $\pm$ 1.23	96.07 $\pm$ 1.45 F2	0.89 $\pm$ 0.01 F1
	Total	453	76.19 $\pm$ 0.47	92.86 $\pm$ 0.32	0.82 $\pm$ 0.003

Comparisons were made within the same sex:

significant difference from 19-29 yr: a1 a2
p<0.0005, p<0.01;

significant difference from 30-39 yr: b1 b2
p<0.0005, p<0.005,
b3 b4
p<0.01, p<0.05;

significant difference from 40-49 yr: c1 c2
p<0.0005, p<0.01.

Significant difference from males with the corresponding

age groups: A1 A2 B C D
p<0.0005, p<0.05, p<0.0005, p<0.002, p<0.05.

Significant difference from total males: F1 F2
p<0.0005, p<0.005.

Table 15. Prevalences of abdominal obesity in 519

RHS by sex and age

Sex	Age	N	Abdominal obesity*	
			N	%
M	20-29	22	-	-
	30-39	22	-	-
	40-49	12	1	8.3
	50-59	10	-	-
	Total	66	1	1.5
F ⁺	19-29	97	23	23.7
	30-39	154	70	45.5
	40-49	158	111	70.3
	50-61	44	41	93.2
	Total	453	245	54.1

*WHR >1.0 in male and >0.8 in female.

⁺P value <0.0005 by chi-square test.

Table 16. Means±SEM of anthropometric parameters in 519 RBS by sex and WHR

Sex	WHR	N	WHR	Body fat	BMI	MUAC	UAMC		
				%	kg/m ²	cm			
M	≤1.0	65	0.86±0.007	26.43±0.66	22.52±0.39	27.54±0.36	94.00±1.22	24.65±0.28	97.42±1.11
	>1.0	1	1.06	36.90	32.05	31.50	107.51	28.10	111.22
F	≤0.8	208	0.76±0.002	36.67±0.29	21.42±0.19	25.24±0.21	88.55±0.74	19.34±0.13	83.37±0.57
	>0.8	245	0.87±0.003	39.05±0.23	24.42±0.24	27.83±0.20	97.66±0.71	20.76±0.14	89.51±0.60
M&F	M≤1.0/F≤0.8	273	0.78±0.004	34.23±0.38	21.68±0.18	25.79±0.19	89.85±0.65	20.60±0.18	86.71±0.62
	M>1.0/F>0.8	246	0.87±0.003	39.06±0.23	24.45±0.25	27.85±0.20	97.70±0.71	20.79±0.14	89.59±0.60

Significant difference from females with WHR ≤0.8: p<0.0005.

Significant difference from males with WHR ≤1.0: p<0.0005, p<0.01.

Significant difference from males with WHR ≤1.0 and females with WHR ≤0.8: p<0.0005.

Table 17. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and age

Sex	Age	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC/HDL-C	LDL-C/HDL-C
mmol/L									
yr									
M	20-29	22	5.1±0.2	3.1±0.2	1.5±0.1	1.1±0.1	2.6±0.1	3.6±0.3	2.2±0.2
	30-39	22	5.4±0.2	3.3±0.2	1.3±0.1	1.3±0.2	2.6±0.2	4.2±0.3	2.7±0.2
	40-49	12	5.3±0.3	3.3±0.3	1.2±0.1	1.9±0.4	2.4±0.2	4.9±0.6	3.0±0.5
	50-60	10	6.9±0.5	4.9±0.4	1.3±0.2	1.5±0.3	2.4±0.1	5.8±0.7	4.1±0.6
	Total	66	5.4±0.2	3.4±0.2	1.3±0.04	1.4±0.1	2.5±0.1	4.3±0.2	2.8±0.2
F	19-29	97	5.0±0.1	3.1±0.1	1.4±0.03	1.1±0.1	2.6±0.1	3.6±0.1	2.2±0.1
	30-39	154	5.1±0.1	3.2±0.1	1.5±0.1	1.1±0.1	2.7±0.1	3.7±0.1	2.3±0.1
	40-49	158	5.3±0.1	3.3±0.1	1.4±0.02	1.3±0.1	2.9±0.1	3.8±0.1	2.4±0.7
	50-61	44	5.8±0.2	3.5±1.2	1.5±0.1	1.6±0.1	3.2±0.1	4.1±0.2	2.5±0.2
	Total	453	5.2±0.1	3.2±0.1	1.5±0.1	1.2±0.1	2.8±0.1	3.8±0.1	2.3±0.1

Comparisons were made within the same sex:

significant difference from 19-29 yr: $p < 0.0005$, $p < 0.002$, $p < 0.005$, $p < 0.02$, $p < 0.05$;

significant difference from 30-39 yr: $p < 0.0005$, $p < 0.005$, $p < 0.02$, $p < 0.05$;

significant difference from 40-44 yr: $p < 0.005$, $p < 0.02$, $p < 0.05$.

Significant difference from males with the corresponding age groups: $p < 0.005$, $p < 0.01$, $p < 0.05$, $p < 0.002$, $p < 0.02$, $p < 0.05$.

Significant difference from total males: $p < 0.005$, $p < 0.01$.

61 yrs old had significantly higher serum TC, TG and PL levels than women of the other age groups. Women with 40-49 yrs old had significantly higher serum TC, LDL-C, TG, PL, and TC/HDL-C ratio than women 19-29 yrs old whereas women with 50-61 yrs old also had significantly higher serum LDL-C level and TC/HDL-C ratio than women 19-29 yrs old. Serum TG and PL levels, TC/HDL-C and LDL-C/HDL-C ratios in women 40-49 yrs old as well as serum HDL-C and PL levels and TC/HDL-C and LDL-C/HDL-C ratios in total women were significantly different from men of the corresponding age groups.

Table 18 shows that men with 40-49 yrs old had significantly lower plasma fibrinogen level than the men 20-29 yrs old. In 453 FRHS, women 50-61 yrs old had plasma L-particle level higher than women 19-29 yrs old, plasma M-particle level in women 50-56 yrs old was significantly higher than the other groups of women, and women 50-61 yrs old had significantly higher plasma S-particle and fibrinogen levels than women 19-29 and 30-39 yrs old.

Table 19 shows that there were no significant differences in serum apo A-I and apo B levels between men and women with the same age. In women, their serum apo B levels increased significantly with age whereas men 50-59 yrs old had significantly higher serum apo B levels than

Table 18. Means $\pm$ SEM of plasma lipoprotein and fibrinogen levels in 519 RBS by sex and age

Sex	Age	N	L-particle	M-particle	S-particle	Fibrinogen
	yr		<----- mg/dL ----->			
M	20-29	22	6.1 $\pm$ 1.2	180.8 $\pm$ 21.9	401.7 $\pm$ 22.9	287.1 $\pm$ 16.3
	30-39	22	7.4 $\pm$ 1.8	157.9 $\pm$ 18.2	414.9 $\pm$ 21.9	258.4 $\pm$ 11.0
	40-49	12	6.2 $\pm$ 1.0	197.7 $\pm$ 32.0	383.8 $\pm$ 27.4	232.6 $\pm$ 16.4
	50-60	10	9.6 $\pm$ 3.2	158.1 $\pm$ 20.2	361.1 $\pm$ 27.1	257.9 $\pm$ 31.7
	Total	66	7.1 $\pm$ 0.8	172.8 $\pm$ 11.2	396.7 $\pm$ 12.1	262.8 $\pm$ 8.5
F	19-29	97	A2 4.3 $\pm$ 0.3	A1 127.2 $\pm$ 6.2	A2 375.3 $\pm$ 7.2	249.4 $\pm$ 6.2
	30-39	154	B 4.9 $\pm$ 0.3	a2 145.7 $\pm$ 6.9	a4 381.5 $\pm$ 6.9	267.6 $\pm$ 5.6
	40-49	158	a3,D 4.9 $\pm$ 0.3	a1,b1,c1 153.6 $\pm$ 5.9	a2,b3 392.5 $\pm$ 6.6	a1,b4,C 283.5 $\pm$ 5.2
	50-61	44	7.3 $\pm$ 1.7	a1,b1,c1 204.6 $\pm$ 16.9	a2,b3 419.1 $\pm$ 13.9	a1,b2 330.8 $\pm$ 14.7
	Total	453	5.1 $\pm$ 0.3	150.2 $\pm$ 3.9	387.7 $\pm$ 3.9	272.7 $\pm$ 3.3

Comparisons were made within the same sex:

significant difference from 19-29 yr: $p < 0.0005$, $p < 0.005$, $p < 0.02$,
a4
 $p < 0.05$;

significant difference from 30-39 yr: $p < 0.0005$, $p < 0.01$, $p < 0.02$,
b4
 $p < 0.05$;

significant difference from 40-49 yr: $p < 0.0005$, $p < 0.02$.

Significant difference from males with the corresponding age groups:

A1 A2 B C D
 $p < 0.002$, $p < 0.05$, $p < 0.05$, $p < 0.01$, $p < 0.002$

Table 19. Means±SEM of serum apoprotein A-I and apoprotein B levels in 519 RBS by sex and age

Sex	Age	N	Apoprotein A-I	Apoprotein B
	yr		<----- mg/dL ----->	
M	20-29	22	155.73±7.28	110.85±5.93
	30-39	22	147.45±6.71	124.58±8.63
	40-49	12	155.83±17.38	129.59±10.47
	50-60	10	147.73±15.11	148.84±13.99 ^a
	Total	66	151.78±4.99	124.59±4.66
F	19-29	97	146.24±2.77	99.92±2.50 ^a
	30-39	154	147.84±2.58	107.65±2.18 ^{a,b}
	40-49	158	148.63±2.50	117.47±2.30 ^{a,b,c}
	50-61	44	151.44±4.29	131.54±5.63 ^F
	Total	453	148.12±1.43	111.74±1.40

Comparisons were made within the same sex:

^a
significant difference from 19-29 yr: $p < 0.01$;

^b
significant difference from 30-39 yr: $p < 0.01$;

^c
significant difference from 40-49 yr: $p < 0.01$.

^F
Significant difference from total males: $p < 0.01$.

men 20-29 yrs old. Total men also had significantly higher serum apo B than total women.

Table 20 shows that when serum TC levels were categorized into <5.20 , $5.20-6.20$ and >6.21 mmol/L as desirable, borderline-high and high serum TC levels, respectively, both men and women showed increases in the prevalences of hypercholesterolemia with age.

Table 21 shows that when serum LDL-C levels were categorized into <3.36 , $3.36-4.11$ and ≥ 4.12 mmol/L as desirable, borderline-high risk and high risk levels, respectively, the prevalences of elevated serum LDL-C levels both in men and women increased significantly with age.

Table 22 shows that when serum HDL-C levels were categorized into <0.90 , $0.90-1.54$ and ≥ 1.55 mmol/L, as high risk, borderline-high risk and desirable levels, respectively, both men and women showed decreases in the prevalences of serum HDL-C levels with age.

Table 23 shows that when serum TG levels were categorized into <2.26 , $2.26-4.51$ and $4.52-11.29$ mmol/L as desirable, borderline-high risk and high risk levels, respectively, both men and women showed increases in the prevalences of hypertriglyceridemia with age.

Table 24 shows that when 519 RHS were categorized into normolipidemia and dyslipidemia including type IIa

Table 20. Number and percentage of 519 BHS by serum TC categories in each age group of each sex

Sex	Age	N	TC (mmol/L)					
			<5.20		5.20-6.20		≥6.21	
			N	%	N	%	N	%
	yr							
M [*]	20-29	22	17	77.3	2	9.1	3	13.6
	30-39	22	17	77.3	1	4.5	4	18.2
	40-49	12	8	66.7	1	8.3	3	25.0
	50-60	10	2	20.0	-	-	8	80.0
	Total	66	44	66.7	4	6.1	18	27.3
F ⁺	19-29	97	67	69.1	20	20.6	10	10.3
	30-39	154	94	61.0	33	21.4	27	17.5
	40-49	158	93	58.9	34	21.5	31	19.6
	50-61	44	17	38.6	11	25.0	16	36.4
	Total	453	271	59.8	98	21.6	84	18.6

*P <0.01 by chi-square test.

+P <0.02 by chi-square test.

Table 21. Number and percentage of 519 RHS by serum LDL-C categories in each age group of each sex

Sex	Age	N	LDL-C (mmol/L)					
			<3.36		3.36-4.11		≥4.12	
			N	%	N	%	N	%
	yr							
M	20-29	22	16	72.7	2	9.1	4	18.2
	30-39	22	17	77.3	2	9.1	3	13.6
	40-49	12	9	75.0	1	8.3	2	16.7
	50-60	10	2	20.0	-	-	8	80.0
		+						
	Total	66	44	66.7	5	7.6	17	25.8
F	19-29	67	73	75.3	13	13.4	11	11.3
	30-39	154	100	64.9	24	15.6	30	19.5
	40-49	158	100	63.3	22	13.9	36	22.8
	50-61	44	22	50.0	9	20.5	13	29.5
		+						
	Total	453	295	65.1	68	15.0	90	19.9

*P<0.0054 by chi-square test.

+

+

8 MRHS (12.1%) and 26 FRHS having serum LDL-C levels ≥4.92 mmol/L.

Table 22. Number and percentage of 519 RBS by serum HDL-C categories in each age group of each sex

Sex	Age	N	HDL-C (mmol/L)					
			<0.90		0.90-1.54		≥1.55	
	yr		N	%	N	%	N	%
M*	20-29	22	-	-	12	54.5	10	45.5
	30-39	22	1	4.5	18	81.8	3	13.6
	40-49	12	4	33.3	6	50.0	2	16.7
	50-60	10	2	20.0	5	50.0	3	30.0
	Total	66	7	10.6	41	62.1	18	27.3
F	19-29	97	1	1.0	60	61.9	36	37.1
	30-39	154	2	1.3	57	63.0	55	35.7
	40-49	158	5	3.2	92	58.2	61	38.6
	50-61	44	2	4.5	21	47.7	21	47.7
	Total	453	10	2.2	220	59.6	173	38.2

*P<0.02 by chi-square test

Table 23. Number and percentage of 519 RHS by serum TG categories in each age group of each sex

Sex	Age	N	TG (mmol/L)					
			<2.26		2.26-4.51		4.52-11.29	
	yr		N	%	N	%	N	%
M	20-29	22	21	95.5	1	4.5	-	-
	30-39	22	20	90.9	2	9.1	-	-
	40-49	12	7	58.3	4	33.3	1	8.3
	50-60	10	8	80.0	2	20.0	-	-
		+						
	Total	66	56	84.8	9	13.6	1	1.5
F	19-29	97	96	99.0	1	1.0	-	-
	30-39	154	149	96.8	4	2.6	1	0.6
	40-49	158	148	93.7	10	6.3	-	-
	50-61	44	38	86.4	5	11.4	1	2.3
		+						
	Total	453	431	95.1	20	4.4	2	0.4

*P<0.05 by chi square test

+ +
50 (75.8%) of 66 MEHS and 376 (83.0%) of 453 FRHS having serum TG levels of <1.69 mmol/L; none of MEHS and FRHS having serum TG levels $\geq$ 11.30 mmol/L.

Table 24. Number and percentage of 519 EHS by serum lipid categories in each age group of each sex

Sex	Age	N	Normal	TC $\geq$ 5.2 mmol/L only	TC $\geq$ 5.2, LDL-C $\geq$ 3.4& TG $\geq$ 2.3 mmol/L	TC $\geq$ 5.2, IDL-C $\geq$ 3.4& TG $\geq$ 2.3 mmol/L	HDL-C $\leq$ 0.9 mmol/L only						
	yr	N	%	N	%	N	%	N	%				
* M	20-29	22	77.3	-	4	18.2	-	1	4.5				
	30-39	22	72.7	-	4	18.2	-	1	4.5				
	40-49	12	41.7	-	1	8.3	1	8.3	4	33.3			
	50-60	10	10.0	-	6	60.0	-	1	1.0	2	20.0		
	Total	66	59.1	-	15	22.7	1	1.5	4	6.1	7	10.6	
+ F	19-29	97	68.0	5	5.2	24	24.7	-	1	1.0	1	1.0	
	30-39	154	60.4	4	2.6	51	33.1	1	0.6	3	1.9	2	1.3
	40-49	158	57.0	7	4.4	49	31.0	1	0.6	6	3.8	5	3.2
	50-61	44	34.1	3	6.8	20	45.5	2	4.5	2	4.5	2	4.5
	Total	453	58.3	19	4.2	144	31.8	4	0.9	12	2.6	10	2.2

*P<0.005 by chi-square test.
+P<0.05 by chi-square test.

hyperlipoproteinemia (TC ≥ 5.2 and LDL-C ≥ 3.4 mmol/L), type IIb hyperlipoproteinemia (TC ≥ 5.2 and LDL-C ≥ 3.4 and TG ≥ 2.3 mmol/L), type IV hyperlipoproteinemia (TG ≥ 2.3 mmol/L only) and HDL-C ≤ 0.9 mmol/L only, the prevalences of normolipidemia decreased significantly with age.

Influence of body composition on serum lipid, apo, plasma lipoprotein and fibrinogen levels

When protein-calorie status in each subject was categorized by BMI into 5 categories: grade II underweight, grade I underweight, acceptable weight, grade I obesity and grade II obesity based on BMI of <18.5 , $18.5-19.9$, $20.0-24.9$, $25.0-29.9$ and ≥ 30.0 kg/m², respectively, Table 25 shows that grade I obese MRHS had significantly higher serum TC, LDL-C and TG levels as well as TC/HDL-C and LDL-C/HDL-C ratios than grade II and grade I underweight MRHS; grade I obese MRHS also had significantly lower serum HDL-C level than grade II underweight MRHS; acceptable weight MRHS had significantly higher serum TG level and serum TC/HDL-C and LDL-C/HDL-C ratios than grade II underweight MRHS; and grade II obese MRHS had significantly higher serum TG level than grade I underweight MRHS whereas their serum TC/HDL-C and LDL-C/HDL-C ratios were significantly higher than grade II underweight, grade I underweight, and acceptable MRHS. In

Table 25. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 PHS by sex and BMI categories

Sex	BMI	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC/HDL-C	LDL-C/HDL-C
kg/m ² <----- mmol/L ----->									
M	<18.5	3	4.2±0.4	2.3±0.4	1.5±0.1	0.9±0.1	2.6±0.1	2.9±0.3	1.6±0.3
	18.5-19.9	9	5.0±0.2	3.1±0.2	1.5±0.1	0.8±0.1	2.5±0.1	3.5±0.3	2.2±0.2
	20.0-24.9	42	5.3±0.2	3.4±0.2	1.4±0.1	1.4±0.1	2.5±0.1	4.2±0.2	2.7±0.2
	25.0-29.9	10	6.4±0.5	4.2±0.4	1.2±0.1	1.7±0.3	2.7±0.1	5.8±0.7	3.9±0.5
	≥30	2	5.6±0.8	3.6±1.0	1.0±0.2	2.4±0.9	2.8±0.5	5.6±0.2	3.5±0.2
	Total	66	5.4±0.2	3.4±0.2	1.3±0.4	1.4±0.1	2.5±0.1	4.3±0.2	2.8±0.2
F	<18.5	35	5.0±0.9	3.1±0.2	1.8±0.2	1.0±0.1	2.7±0.1	3.3±0.1	2.0±0.1
	18.5-19.9	59	5.0±0.1	3.1±0.1	1.6±0.4	1.0±0.1	2.8±0.1	3.4±0.1	2.1±0.1
	20.0-24.9	237	5.2±0.1	3.2±0.1	1.5±0.02	1.1±0.04	2.8±0.1	3.6±0.1	2.2±0.1
	25.0-29.9	105	5.5±0.1	3.5±0.3	1.4±0.03	1.5±0.1	2.8±0.1	4.2±0.1	2.7±0.1
	≥30	17	5.4±0.3	3.2±0.3	1.3±0.1	2.0±0.2	2.9±0.1	4.5±0.5	2.7±0.3
	Total	453	5.2±0.1	3.2±0.1	1.5±0.2	1.2±0.03	2.8±0.03	3.8±0.1	2.3±0.04

Comparisons were made within the same sex:

significant difference from $\langle 18.5 \text{ kg/m}^2 \rangle$: a1 p<0.0005, a2 p<0.002, a3 p<0.005, a4 p<0.01, a5 p<0.002, a6 p<0.05; b1 b2 b3 b4 b5 b6

significant difference from 18.5-19.9 kg/m²: p<0.0005, p<0.002, p<0.005, p<0.01, p<0.02, p<0.05;

χ^2 test: $p < 0.0005$, $p < 0.002$, $p < 0.01$, $p < 0.02$, $p < 0.05$.

significant difference from 20.0-24.9 kg/m²: p<0.0005, p<0.002, p<0.01, p<0.02, p<0.05 E)

Significant difference from total males: $p < 0.005$, $p < 0.01$.

453 FRHS, grade I obese FRHS had significantly higher serum TC and TG levels as well as serum TC/HDL-C and LDL-C/HDL-C ratios but significantly lower serum HDL-C level than grade II underweight, grade I underweight, and acceptable weight FRHS whereas their serum LDL-C level was also significantly higher than those in grade I underweight and acceptable weight FRHS; and grade II obese FRHS had significantly higher serum TG but significantly lower serum HDL-C levels than grade I underweight and acceptable weight FRHS, had significantly higher serum TC/HDL-C and LDL-C/HDL-C ratios than grade II underweight, grade I underweight, and acceptable weight FRHS, and had significantly higher serum PL level than acceptable weight FRHS. Acceptable weight FRHS had significantly lower serum TG level and TC/HDL-C and LDL-C/HDL-C ratios but significantly higher serum PL level than acceptable weight MRHS whereas serum TC/HDL-C ratios in acceptable weight, grade I obese, and grade II obese FRHS as well as serum LDL-C/HDL-C ratios in acceptable weight and grade I obese FRHS were significantly higher than those in MRHS with the corresponding BMI categories.

Table 26 shows that grade I obese MRHS had significantly higher plasma L-particle and M-particle, levels than grade I underweight MRHS and had significantly higher plasma S-particle level than acceptable weight

Table 26. Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and BMI categories

Sex	BMI	N	L-particle	M-particle	S-particle	Fibrinogen
			kg/m ² <----- mg/dL ----->			
M	<18.5	3	5.1±2.1	134.1±24.1	311.3±27.7	277.2±27.4
	18.5-19.9	9	6.1±1.2	117.5±11.4	373.8±16.3	281.5±21.1
	20.0-24.9	42	6.8±1.2	166.7±13.1	391.4±14.2	260.6±11.5
	25.0-29.9	10	8.5±2.3	246.6±34.2	464.2±42.7	252.7±18.9
	≥30	2	13.5±4.5	238.8±95.2	401.7±61.4	254.9±17.3
	Total	66	7.1±0.8	172.8±11.2	396.7±12.1	262.8±8.5
F	<18.5	35	6.0±1.9	128.6±15.9	383.2±17.3	242.5±8.7
	18.5-19.9	59	3.7±0.4	111.6±6.5	380.6±9.3	259.3±9.9
	20.0-24.9	237	4.9±0.3	146.0±5.1	382.9±5.4	275.3±4.3
	25.0-29.9	105	5.9±0.5	179.9±8.6	404.3±7.8	285.3±7.1
	≥30	17	6.4±1.2	202.9±17.4	384.7±21.8	279.5±18.0
	Total	453	5.1±0.3	150.2±3.9	387.7±3.9	272.7±3.3

Comparisons were made within the same sex:

significant difference from <18.5 kg/m²: p<0.0005, p<0.005, p<0.01, p<0.05;
 significant difference from 18.5-19.9 kg/m²: p<0.0005, p<0.005, p<0.01, p<0.05;
 significant difference from 20.0-24.9 kg/m²: p<0.0005, p<0.005, p<0.02, p<0.05.
 Significant difference from males with the corresponding BMI groups; p<0.05, p<0.05.
 Significant difference from total males: p<0.005.

MRHS; and grade II obese MRHS had significantly higher plasma M-particle level than acceptable weight MRHS. In 453 FRHS, grade I obese FRHS had significantly higher plasma L-particle level than grade I underweight FRHS, had significantly higher plasma S-particel level than acceptable weight FRHS, and had significantly higher plasma fibrinogen level than grade II underweight and grade I underweight FRHS; grades I and II obese FRHS had significantly higher plasma M-particle level than grade II underweight, grade I underweight, and acceptable weight FRHS; grade II obese FRHS had significantly higher plasma fibrinogen level than grade II underweight FRHS; and acceptable weight FRHS had significantly higher plasma L-particle and M-particle levels than grade I underweight FRHS and had significantly higher plasma fibrinogen level than grade II underweight FRHS; plasma L-particle level in grade I underweight and M-particle level in grade I obese FRHS were significantly lower than those in MRHS with the corresponding BMI categories.

Table 27 shows that grade I obese and acceptable weight FRHS had significantly lower serum apo B levels than the corresponding groups of MRHS. In FRHS, their serum apo A-I decreased significantly with increased BMI whereas opposite resulty were observed for their serum apo B levels. In men, those with acceptable weight and those

Table 27. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RBS by sex and BMI categories

Sex	BMI	N	Apoprotein A-I	Apoprotein B
	kg/m ²		<----- mg/dL ----->	
M	<18.5	3	182.67 $\pm$ 13.12	105.13 $\pm$ 19.23
	18.5-19.9	9	177.56 $\pm$ 21.66 _{b2}	100.54 $\pm$ 6.17
	20.0-24.9	42	145.41 $\pm$ 5.18	123.70 $\pm$ 6.28 _{b1}
	25.0-29.9	10	138.80 $\pm$ 10.78	150.90 $\pm$ 7.89
	≥ 30	2	188.00 $\pm$ 16.00	149.00 $\pm$ 7.00
	Total	66	151.78 $\pm$ 4.99	124.59 $\pm$ 4.66
F	<18.5	35	161.58 $\pm$ 6.25	95.93 $\pm$ 4.14
	18.5-19.9	59	157.17 $\pm$ 4.15 _{a1,b}	100.22 $\pm$ 3.49 _{a2,b2,C}
	20.0-24.9	237	145.86 $\pm$ 1.79 _{a1,b}	109.10 $\pm$ 1.75 _{a1,b1,c,D}
	25.0-29.9	105	144.63 $\pm$ 2.97	126.11 $\pm$ 3.11 _{a1,b1,c}
	≥ 30	17	142.19 $\pm$ 9.50	132.39 $\pm$ 7.42 _F
	Total	453	148.12 $\pm$ 1.43	111.74 $\pm$ 1.40

Comparisons were made within the same sex:

significant difference from <18.5 kg/m²: ^{a1} p<0.01, ^{a2} p<0.05;

significant difference from 18.5-19.9 kg/m²: ^{b1} p<0.01, ^{b2} p<0.05;

significant difference from 20-24.9 kg/m²: ^c p<0.01.

Significant difference from males with the corresponding BMI groups:

^C ^D
p<0.05, p<0.05.

^F
Significant difference from total males: p<0.01.

with grade I obesity had significantly lower serum apo A-I and higher apo B levels than those with grade I underweight. Acceptable weight MRHS had significantly lower serum apo A-I level than grade I underweight MRHS whereas grade I obese MRHS had significantly higher serum apo B level than grade I underweight MRHS; acceptable weight FRHS had significantly lower serum apo A-I but significantly higher serum apo B levels than grade II underweight and grade I underweight FRHS; grade I obese FRHS had significantly lower serum apo A-I level than grade II underweight and grade I underweight FRHS; and grade I and II obese FRHS had significantly higher serum apo B levels than the remaining FRHS.

Table 28 shows that both MRHS and FRHS with MUAC <90% standard (std) had significantly lower serum TC, LDL-C, and TG levels as well as, TC/HDL-C and LDL-C/HDL-C ratios but significantly higher serum HDL-C level than those with MUAC $\geq$ 90% std. FRHS with MUAC <90% std had significantly higher serum PL level whereas FRHS with MUAC $\geq$ 90% std had significantly higher serum HDL-C level but significantly lower serum TC/HDL-C and LDL-C/HDL-C ratios than MRHS with the corresponding MUAC.

Table 29 shows that MRHS and FRHS with MUAC <90% std had significantly lower plasma L-, M-, and S-particle levels than men and women with MUAC $\geq$ 90% std. MRHS with

Table 28. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 EHS by sex and mid upper arm circumferences

Sex	MUAC	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC/HDL-C	LDL-C/HDL-C
<----- mmol/L ----->									
M	<90 % std	23	4.9±0.2	2.9±0.2	1.5±0.1	0.9±0.1	2.5±0.1	3.3±0.2	2.0±0.2
			a3	a5	a4	a1		a1	a1
	≥90 % std	43	5.7±0.2	3.7±0.2	1.2±0.1	1.6±0.2	2.6±0.1	4.9±0.3	3.2±0.2
	Total	66	5.4±0.2	3.4±0.2	1.3±0.04	1.4±0.1	2.5±0.1	4.3±0.2	2.8±0.2
F	<90 % std	189	5.0±0.1	3.0±0.1	1.6±0.04	1.0±0.03	2.7±0.04	3.4±0.1	2.1±0.1
			a1	a1	a2,B1	a1		a1,B2	a1,B1
	≥90 % std	264	5.4±0.1	3.4±0.1	1.4±0.02	1.4±0.04	2.8±0.04	3.9±0.02	2.5±0.1
					F2		F1	F2	F2
	Total	453	5.2±0.1	3.2±0.1	1.5±0.1	1.2±0.1	2.8±0.1	3.8±0.1	2.3±0.1

Comparisons were made within the same sex:

significant difference from <90 % std: p<0.0005, p<0.001, p<0.002, p<0.005, p<0.01.
 Significant difference from males with the corresponding UAC groups: p<0.005, p<0.01, p<0.002.
 Significant difference from total males: p<0.005, p<0.01.

Table 29. Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and mid upper arm circumferences

Sex	MUAC	N	L-particle	M-particle	S-particle	Fibrinogen
<----- mg/dl. ----->						
M	<90 % std	23	4.6±0.7 a3	129.2±10.3 a2	360.2±14.02 a4	292.2±14.2 a4
	≥90 % std	43	8.4±1.2	196.1±15.04	416.2±16.1	247.1±9.8
	Total	66	7.1±0.8	172.8±11.2	396.7±12.1	262.8±8.5
F	<90 % std	189	4.3±0.4 a4,B2	125.0±4.7 a1	376.3±5.7 a4	256.2±4.7 a1,B1
	≥90 % std	264	5.7±0.4 F	168.2±5.5 F	395.8±5.3	285.3±4.3
	Total	453	5.1±0.3	150.2±3.9	387.7±3.9	272.7±3.3

Comparisons were made within the same sex:

· significant difference from <90 % std: p<0.0005, p<0.001, p<0.01, p<0.02.

Significant difference from males with the corresponding UAC groups: p<0.02, p<0.001, B2 p<0.05.

Significant difference from total males: p<0.05.

MUAC <90% std had significantly lower plasma fibrinogen level than MRHS with MUAC $\geq$ 90% std whereas opposite results was observed for FRHS. FRHS with MUAC $\geq$ 90% std had significantly lower plasma L-particle level than MRHS with MUAC $\geq$ 90% std. FRHS with MUAC <90% std had significantly lower plasma fibrinogen level than MRHS with MUAC <90% std whereas opposite result was observed between men and women with MUAC $\geq$ 90% std.

Table 30 shows that MRHS with MUAC $\geq$ 90% std had significantly higher apo B level than MRHS with MUAC <90% std. Similar finding was also observed for FRHS $\geq$ 90% std and FRHS with MUAC <90% std also had significantly lower apo A-I level than women with MUAC <90% std; and FRHS with MUAC $\geq$ 90% std had significantly lower serum apo B level than MRHS with the same MUAC.

Table 31 shows that MRHS with UAMC $\geq$ 90% std had significantly higher serum TC/HDL-C and LDL-C/HDL-C ratios than MRHS with UAMC <90% std; FRHS with UAMC $\geq$ 90% std had significantly higher serum TC, LDL-C and TG levels as well as serum TC/HDL-C and LDL-C/HDL-C ratios but had significantly lower serum HDL-C levels than FRHS with UAMC <90% std; and FRHS with UAMC $\geq$ 90% std had significantly higher serum PL level than MRHS with the same UAMC.

Table 32 shows that FRHS with UAMC $\geq$ 90% std had significantly higher plasma M-particle and fibrinogen levels than FRHS with UAMC 90% std.

Table 30. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and mid upper arm circumferences

Sex	MUAC	N	Apoprotein A-I	Apoprotein B
$\langle \text{----- mg/dL -----} \rangle$				
M	<90 % std	23	162.21 $\pm$ 10.74	108.68 $\pm$ 5.64 ^{a2}
	$\geq$ 90 % std	43	146.20 $\pm$ 4.97	133.10 $\pm$ 6.14
	Total	66	151.78 $\pm$ 4.99	124.59 $\pm$ 4.66
F	<90 % std	189	152.71 $\pm$ 2.30 ^{a2}	101.05 $\pm$ 1.83 ^{a1,B}
	$\geq$ 90 % std	264	144.84 $\pm$ 1.80	119.40 $\pm$ 1.87 ^F
	Total	453	148.12 $\pm$ 1.43	111.74 $\pm$ 1.40

Comparisons were made within the same sex:

significant difference from <90 % std: ^{a1} $p < 0.0005$, ^{a2} $p < 0.01$.

Significant difference from males with the corresponding UAC^B groups: $p < 0.05$.

Significant difference from total males: ^F $p < 0.01$.

Table 31. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and mid upper arm muscle circumferences

Sex	UAMC	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC/HDL-C	LDL-C/HDL-C
<----- mmol/L ----->									
M	<90 % std	14	4.9±0.3	2.9±0.3	1.5±0.1	1.1±0.2	2.5±0.1	3.5±0.3	2.1±0.3
	≥90 % std	52	5.5±0.2	3.6±0.2	1.3±0.05	1.4±0.1	2.5±0.1	4.6±0.2	3.0±0.2
	Total	66	5.4±0.2	3.4±0.2	1.3±0.04	1.4±0.1	2.5±0.1	4.3±0.2	2.8±0.2
F	<90 % std	295	5.2±0.1	3.2±0.1	1.5±0.03	1.1±0.03	2.8±0.04	3.6±0.05	2.2±0.05
	≥90 % std	158	5.4±0.1	3.4±0.1	1.3±0.02	1.4±0.1	2.8±0.05	4.1±0.1	2.6±0.1
	Total	453	5.2±0.1	3.2±0.1	1.5±0.1	1.2±0.1	2.8±0.1	3.8±0.1	2.3±0.1

Comparisons were made within the same sex:

significant difference from <90 % std: $p<0.0005$, $p<0.001$, $p<0.002$, $p<0.005$.

Significant difference from males with the corresponding UAMC groups: $p<0.01$.

Significant difference from total males: $p<0.005$, $p<0.01$.

Table 32. Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and mid upper arm muscle circumferences

Sex	UAMC	N	L-particle	M-particle	S-particle	Fibrinogen
<----- mg/dL ----->						
M	<90 % std	14	6.4±1.2	144.4±16.3	362.4±22.7	262.9±15.9
	≥90 % std	52	7.2±1.1	180.4±13.3	405.9±13.7	262.8±9.9
	Total	66	7.1±0.8	172.8±11.2	396.7±12.1	262.8±8.5
F	<90 % std	295	4.9±0.4	137.3±4.1	383.1±4.7	266.8±3.9 ^{a2}
	≥90 % std	158	5.6±0.4 ^F	174.2±7.6 ^{a1}	396.2±6.9	285.0±5.8
	Total	453	5.1±0.3 ^F	150.2±3.9	387.7±3.9	272.7±3.3

Comparisons were made within the same sex:

^{a1} significant difference from <90 % std: $p < 0.0005$, $p < 0.01$.

^F Significant difference from total males: $p < 0.05$.

Table 33 shows that FRHS with UAMC $\geq 90\%$ std had significantly lower apo A-I but higher apo B levels than FRHS with UAMC $< 90\%$ std.

Table 34 shows that FRHS with TST $< 90\%$ std had significantly lower serum LDL-C level and TC/HDL-C and LDL-C/HDL-C ratios but higher serum HDL-C level than FRHS with TST $\geq 90\%$ std; and FRHS with TST $< 90\%$ std had significantly higher serum HDL-C and PL levels but lower serum TC/HDL-C and LDL-C/HDL-C ratios than MRHS with the same TST whereas similar findings, except serum PL level, were also observed between MRHS and FRHS with TST $\geq 90\%$ std.

Table 35 shows that MRHS with TST $< 90\%$ std had significantly higher plasma fibrinogen level than MRHS with TST $\geq 90\%$ std whereas opposite result was observed in FRHS; and FRHS with TST $< 90\%$ std had significantly lower plasma fibrinogen level than MRHS $< 90\%$ std whereas opposite result was observed between FRHS with TST $\geq 90\%$ std and MRHS the same TST.

Table 36 shows that MRHS and FRHS with TST $\geq 90\%$ std had significantly lower serum apo A-I levels than MRHS and FRHS with TST $< 90\%$ std whereas FRHS with TST $< 90\%$ std had significantly lower serum apo B than MRHS with the same TST.

Table 33. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and mid upper arm muscle circumferences

Sex	UAMC	N	Apoprotein A-I	Apoprotein B
$\langle \text{----- mg/dL -----} \rangle$				
M	<90 % std	14	161.00 $\pm$ 16.06	108.30 $\pm$ 6.60
	$\geq$ 90 % std	52	149.29 $\pm$ 4.69	128.97 $\pm$ 5.51
	Total	66	151.78 $\pm$ 4.99	124.59 $\pm$ 4.66
F	<90 % std	295	151.42 $\pm$ 1.81 ^{a2}	106.33 $\pm$ 1.61 ^{a1}
	$\geq$ 90 % std	158	141.96 $\pm$ 2.26	121.85 $\pm$ 2.47 ^F
	Total	453	148.12 $\pm$ 1.43	111.74 $\pm$ 1.40

Comparisons were made within the same sex:

significant difference from <90 % std: ^{a1} $p < 0.0005$, ^{a2} $p < 0.005$.

^F Significant difference from total males: $p < 0.01$.

Table 34. Mean±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 BHS by sex and triceps skinfold thickness

Sex	TST	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC/HDL-C	LDL-C/HDL-C
<----- mmol/L ----->									
M	<90 % std	47	5.3±0.2	3.3±0.2	1.4±0.05	1.3±0.1	2.5±0.1	4.1±0.2	2.6±0.2
	≥90 % std	19	5.7±0.3	3.7±0.3	1.2±0.10	1.6±0.2	2.6±0.1	4.9±0.4	3.3±0.3
	Total	66	5.4±0.2	3.4±0.2	1.3±0.04	1.4±0.1	2.5±0.1	4.3±0.2	2.8±0.2
F	<90 % std	94	5.1±0.1	3.0±0.1	1.6±0.1	1.1±0.1	2.8±0.1	3.5±0.1	2.0±0.1
	≥90 % std	359	5.3±0.1	3.3±0.1	1.4±0.01	1.2±0.03	2.8±0.03	3.9±0.1	2.4±0.05
	Total	453	5.2±0.1	3.2±0.1	1.5±0.1	1.2±0.1	2.8±0.1	3.8±0.1	2.3±0.1

Comparisons were made within the same sex: significant difference from <90 % std: p<0.0005, p<0.05.

Significant difference from males with the corresponding TSF groups: p<0.01, p<0.02, p<0.05.

B1 B2 p<0.01, p<0.02.

F1 F2

Significant difference from total males: p<0.005, p<0.01.

Table 35. Means±SRM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and triceps skinfold thickness

Sex	TST	N	L-particle	M-particle	S-particle	Fibrinogen
$\langle \text{-----} \text{mg/dL} \text{-----} \rangle$						
M	<90 % std	47	6.5±0.9	164.2±12.0	389.2±13.7	273.3±9.9
	≥90 % std	19	8.5±1.9	193.9±24.4	415.2±24.4	235.6±15.4 ^{a2}
	Total	66	7.1±0.8	172.8±11.2	396.7±12.1	262.8±8.5
F	<90 % std	94	4.5±0.8	135.5±10.2	379.0±8.2	248.0±6.6 ^A
	≥90 % std	359	5.3±0.3 ^F	154.0±4.0 ^F	389.9±4.4	289.2±3.7 ^{a1,B}
	Total	453	5.1±0.3	150.2±3.9	387.7±3.9	272.7±3.3

Comparisons were made within the same sex:

significant difference from <90 % std: ^{a1} $p<0.0005$, ^{a2} $p<0.05$.

Significant difference from males with the corresponding TSF groups: ^A $p<0.05$, ^B $p<0.02$.

Significant difference from total males: ^F $p<0.05$.

Table 36. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and triceps skinfold thickness

Sex	TST	N	Apoprotein A-I	Apoprotein B
<----- mg/dL ----->				
M	<90 % std	47	158.4 $\pm$ 6.3 ^{a2}	121.4 $\pm$ 5.1
	$\geq$ 90 % std	19	135.3 $\pm$ 6.2	132.5 $\pm$ 10.2
	Total	66	151.7 $\pm$ 4.9	124.6 $\pm$ 4.7
F	<90 % std	94	154.7 $\pm$ 3.3 ^{a2}	99.3 $\pm$ 2.6 ^A
	$\geq$ 90 % std	359	146.44 $\pm$ 1.6	144.9 $\pm$ 1.6 ^{a1}
	Total	453	148.1 $\pm$ 1.4	111.7 $\pm$ 1.4 ^F

Comparisons were made within the same sex:

a1 a2

significant difference from <90 % std: p<0.0005, p<0.02.

Significant difference from males with the corresponding TSF

A

groups: p<0.0005.

F

Significant difference from total males: p<0.01.

Table 37 shows that FRHS with abdominal obesity had significantly lower serum HDL-C level but higher serum TG and PL levels as well as serum TC/HDL-C and LDL-C/HDL-C ratios than FRHS without abdominal obesity. FRHS without abdominal obesity had significantly higher serum HDL-C level but lower serum TG level as well as serum TC/HDL-C and LDL-C/HDL-C ratios than MRHS without abdominal obesity.

Table 38 shows that FRHS with abdominal obesity had significantly higher plasma L-particle, M-particle and fibrinogen levels than FRHS without abdominal obesity. FRHS without abdominal obesity had significantly lower plasma L-particle and fibrinogen levels than MRHS without abdominal obesity. Combined MRHS and FRHS with abdominal obesity had significantly higher plasma M-particle and fibrinogen levels than those without abdominal obesity.

Table 39 shows that FRHS without abdominal obesity had significantly lower serum apo B level than MRHS without abdominal obesity; and FRHS with abdominal obesity as well as combined MRHS and FRHS with abdominal obesity had significantly higher serum apo B level than those without abdominal obesity.

Table 37. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and WHR

Sex	WHR	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC/HDL-C	LDL-C/HDL-C
mmol/L									
M	≤1.0	65	5.4±0.2	3.4±0.1	1.4±0.04	1.3±0.1	2.5±0.1	4.3±0.2	2.8±0.2
	>1	1	5.1	2.9	0.9	3.0	2.3	5.9	3.3
F	≤0.8	208	5.2±0.1	3.2±0.1	1.6±0.04	1.0±0.02	2.7±0.05	3.6±0.1	2.2±0.1
	>0.8	245	5.3±0.1	3.3±0.1	1.4±0.02	1.4±0.05	2.9±0.04	3.9±0.1	2.4±0.1
M&F	M≤1.0/F≤0.8	273	5.2±0.1	3.3±0.1	1.5±0.03	1.1±0.03	2.7±0.04	3.7±0.1	2.4±0.1
	M>1.0/F>0.8	246	5.3±0.1	3.3±0.1	1.4±0.02	1.4±0.05	2.8±0.04	3.9±0.1	2.4±0.1

Comparisons were made within the same sex: significant difference from ≤0.8 WHR: p<0.0005, p<0.002, p<0.02.
Significant difference from males with the corresponding WHR groups: p<0.001, p<0.002, p<0.005.
Significant difference from males with WHR ≤1.0 and females with WHR ≤0.8: p<0.001, p<0.002, p<0.02.

Table 38. Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and WHR

Sex	WHR	N	L-particle	M-particle	S-particle	Fibrinogen
(<----- mg/dL ----->)						
M	≤1.0	65	6.9±0.9	170.3±10.9	397.6±12.1	263.2±8.5
	>1	1	18.0±0.0	334.0±0.0	340.3±0.0	237.6±0.0
F	≤0.8	208	4.3±0.2 ^A	128.3±3.7 ^A	388.4±5.7	265.6±4.7 ^{a4}
	>0.8	245	5.8±0.4 ^{a2}	168.8±6.2 ^{a1}	387.0±5.4	279.6±4.5
M&F	M≤1.0/F≤0.8	273	4.9±0.3	138.3±4.0	396.6±5.2	265.0±4.1 ^{a3}
	M>1.0/F>0.8	246	5.9±0.5	169.5±6.1 ^{a1}	386.9±5.3	279.4±4.5

Comparisons were made within the same sex:

significant difference from ≤0.8 WHR: ^{a1} p<0.0005, ^{a2} p<0.002, ^{a3} p<0.02, ^{a4} p<0.05.

Significant difference from males with the corresponding WHR groups: ^{A1} p<0.001, ^{A2} p<0.005.

Significant difference from males with WHR ≤1.0 and females with WHR ≤0.8: ^{a1} p<0.0005, ^{a3} p<0.02.

Table 39. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and WHR

Sex	WHR	N	Apoprotein A-I	Apoprotein B
mg/dL				
M	≤ 1.0	65	151.47 $\pm$ 5.06	124.32 $\pm$ 4.72
	> 1.0	1	172	142
F	≤ 0.8	208	150.33 $\pm$ 2.18	104.18 $\pm$ 1.81 ^A
	> 0.8	245	146.25 $\pm$ 1.89	118.16 $\pm$ 2.00 ^{a1}
M&F	M ≤ 1.0 /F ≤ 0.8	273	150.60 $\pm$ 2.05	108.98 $\pm$ 1.85 ^{a2}
	M > 1.0 /F > 0.8	246	146.36 $\pm$ 1.88	118.26 $\pm$ 1.99

Significant difference from females with ≤ 0.8 WHR: ^{a1} $p < 0.0005$.

Significant difference from males with the corresponding WHR groups:
A
 $p < 0.0005$.

Significant difference from males with WHR ≤ 1.0 and females with
^{a2}
WHR ≤ 0.8 : $p < 0.001$.

Influence of visceral protein status on serum lipid, apo, plasma lipoprotein and fibrinogen levels

Table 40 shows that FRHS with TRA <2 g/L had significantly lower serum TC and TG levels than FRHS with TRA ≥ 2 g/L. FRHS with RBP <35 mg/L had significantly lower serum TC, LDL-C and TG levels as well as, TC/HDL-C and LDL-C/HDL-C ratios but significantly higher serum HDL-C level than those with RBP ≥ 35 mg/L.

Table 41 shows that MRHS with RBP <35 mg/L had significantly lower plasma L-particle level than MRHS with RBP ≥ 35 mg/L. FRHS with RBP <35 mg/L had significantly lower plasma L- and M-particle levels than women with RBP ≥ 35 mg/L.

Table 42 shows that FRHS with TRA <2 g/L had significantly lower apo B level than FRHS with TRA ≥ 2 g/L whereas FRHS with RBP <35 mg/L had significantly lower apo B level than FRHS with RBP ≥ 35 mg/L.

Influence of anemia on serum lipid, apo, plasma lipoprotein and fibrinogen levels

Table 43 shows that non-anemic MRHS (Hb ≥ 130 g/L) had significantly higher serum PL level than anemic MRHS (Hb <130 g/L); non-anemic FRHS (Hb ≥ 120 g/L) had significantly higher serum TC, TG, and PL levels than

Table 40. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and visceral protein status

Sex	Serum protein	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC HDL-C	LDL-C HDL-C
(<----- mmol/L ----->)									
M	ALB <35 g/L	1	7.2	5.4	1.4	1.0	4.0	5.1	3.8
	≥35 g/L	65	5.4±1.2	3.4±0.1	1.3±0.04	1.4±0.1	2.5±0.1	4.3±0.2	2.8±0.2
	TRA <2 g/L	4	6.1±1.3	4.1±1.3	1.4±0.2	1.4±0.5	2.7±0.2	4.4±0.07	2.9±0.8
	≥2 g/L	62	5.4±0.1	3.4±0.1	1.3±0.04	1.4±0.1	2.5±0.1	4.3±0.02	2.8±0.2
	RBP <35 mg/L	5	5.3±0.6	3.2±0.6	1.4±0.1	1.7±0.7	2.3±0.2	4.0±0.06	2.4±0.5
	≥35 mg/L	61	5.4±0.2	3.5±0.2	1.3±0.04	1.3±0.1	2.6±0.1	4.4±0.2	2.8±0.2
F	ALB <35 g/L	27	5.4±0.2	3.4±0.2	1.4±0.05	1.2±0.1	2.9±0.1	3.8±0.2	2.4±0.2
	≥35 g/L	426	5.3±0.05	3.2±0.05	1.5±0.02	1.2±0.03	2.8±0.03	3.7±0.05	2.3±0.04
	TRA <2 g/L	42	4.9±0.2	3.0±0.2	1.4±0.05	1.0±0.01	2.8±0.1	3.6±0.2	2.2±0.2
	≥2 g/L	411	5.3±0.05	3.3±0.05	1.5±0.02	1.2±0.03	2.8±0.03	3.8±0.1	2.3±0.04
	RBP <35 mg/L	175	5.1±0.1	3.1±0.1	1.5±0.05	1.0±0.02	2.7±0.05	3.5±0.1	2.2±0.1
	≥35 mg/L	278	5.4±0.1	3.3±0.1	1.4±0.02	1.4±0.04	2.9±0.04	3.9±0.1	2.4±0.05

Significant difference from normal: a1 a2 a3 a4 p<0.0005, p<0.002, p<0.01, p<0.05.

Table 41. Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RBS by sex and visceral protein status

Sex	Serum protein	N	L-particle	M-particle	S-particle	Fibrinogen
<----- mg/dL ----->						
M	ALB <35 g/L	1	3.6	199.6	538.9	313.2
	≥35 g/L	65	7.1±0.9	172.4±11.3	394.5±12.0	262.1±8.5
	TRA <2 g/L	4	6.5±3.6	149.2±24.8	459.7±101.1	297.5±42.2
	≥2 g/L	62	7.1±0.9	174.3±11.7	392.6±11.2	260.6±8.6
	RBP <35 mg/L	5	3.1±0.9	178.2±44.6	389.9±46.9	250.1±30.9
	≥35 mg/L	61	7.4±0.9 ^{a2}	172.3±11.6	397.2±12.5	263.9±8.8
F	ALB <35 g/L	27	6.7±1.2	172.1±19.9	394.7±18.6	273.2±13.2
	≥35 g/L	426	5.0±0.3	148.8±3.9	387.2±4.0	272.7±3.4
	TRA <2 g/L	42	4.6±0.6	137.5±12.6	367.6±13.4	268.9±10.7
	≥2 g/L	411	5.2±0.3	151.5±4.1	389.7±4.1	273.1±3.5
	RBP <35 mg/L	175	3.8±0.2	118.8±4.0	378.6±6.2	265.9±5.4
	≥35 mg/L	278	5.9±0.4 ^{a1}	169.9±5.4 ^{a1}	393.4±5.0	277.0±4.2

Significant difference from normal: ^{a1} p<0.0005, ^{a2} p<0.005.

Table 42. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and visceral protein status

Sex	Serum protein	N	Apoprotein A-I	Apoprotein B
			$\langle$ ----- mg/dL ----- $\rangle$	
M	ALB <35 g/L	1	206.0	177.0
	≥ 35 g/L	65	150.9 $\pm$ 5.0	123.8 $\pm$ 4.6
	TRA <2 g/L	4	143.8 $\pm$ 23.6	120.6 $\pm$ 17.1
	≥ 2 g/L	62	152.3 $\pm$ 5.1	124.8 $\pm$ 4.9
	RBP <35 mg/L	5	146.6 $\pm$ 15.5	110.6 $\pm$ 17.5
	≥ 35 mg/L	61	152.2 $\pm$ 5.3	125.7 $\pm$ 4.8
F	ALB <35 g/L	27	142.7 $\pm$ 5.6	117.8 $\pm$ 0.7
	≥ 35 g/L	426	148.5 $\pm$ 1.5	111.4 $\pm$ 1.5
	TRA <2 g/L	42	139.7 $\pm$ 4.6	94.7 $\pm$ 4.1
	≥ 2 g/L	411	148.9 $\pm$ 1.5	113.5 $\pm$ 1.5 ^a
	RBP <35 mg/L	175	150.7 $\pm$ 2.5	102.3 $\pm$ 1.9
	≥ 35 mg/L	278	146.5 $\pm$ 1.7	117.7 $\pm$ 1.8 ^a

^a
Significant difference from normal: $p < 0.0005$.

Table 43. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 506 RHS by sex and Hb levels

Sex	Hb	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC HDL-C	LDL-C HDL-C
g/L									
mmol/L									
M	<130	6	5.0±0.3	3.0±0.5	1.5±0.1	1.3±0.4	3.05±0.3	3.6±0.5	2.2±0.5
	≥130	60	5.4±0.2	3.5±0.2	1.3±0.4	1.4±0.1	2.50±0.04	4.4±0.7	2.8±0.2
	Total	66	5.4±0.2	3.4±0.1	1.4±0.04	1.4±0.1	2.50±0.1	4.3±0.2	2.8±0.2
F	<120	114	5.0±0.1	3.1±0.1	1.4±0.03	1.1±0.1	2.60±0.1	3.6±0.1	2.3±0.1
	≥120	326	5.1±0.1	3.3±0.1	1.5±0.03	1.3±0.04	2.90±0.04	3.8±0.1	2.4±0.1
	Total	440	5.3±0.1	3.3±0.1	1.5±0.02	1.2±0.03	2.80±0.03	3.8±0.1	2.4±0.04

Comparisons were made within the same sex: significant difference from <130 g/L: ^{a3} p<0.02;
significant difference from <120 g/L: ^{a1} p<0.002, ^{a2} p<0.005, ^{a4} p<0.05.
Significant difference from males with the corresponding Hb levels: ^{B1} p<0.0005, ^{B2} p<0.005, ^{B3} p<0.02.
Significant difference from total males: ^{F1} p<0.005, ^{F2} p<0.01, ^{F3} p<0.02.

anemic FRHS (Hb <120 g/L); and non-anemic FRHS had significantly higher serum HDL-C and PL levels but lower serum TC/HDL-C and LDL-C/HDL-C ratios than non-anemic MRHS.

Table 44 shows that non-anemic FRHS had significantly higher plasma L-, M-, and S-particle and fibrinogen levels than anemic FRHS.

Table 45 shows that non-anemic FRHS had significantly higher serum apo B level than anemic FRHS whereas opposite result was observed between non-anemic women and men.

Influence of blood glucose levels on serum lipid, apo, lipoprotein and fibrinogen levels

Table 46 shows that when RHS were categorized into euglycemia (FBG <6.7 and PBG <7.8 mmol/L), IGT (FBG <6.7 and PBG 7.8-11.1 mmol/L), and DM (FBG >6.7 and PBG ≥11.1 mmol/L), FRHS with IGT had significantly higher serum TC, TG, and PL levels and TC/HDL-C and LDL-C/HDL-C ratios than euglycemic FRHS; diabetic FRHS had significantly higher serum TG level than euglycemic FRHS; and euglycemic FRHS had significantly higher serum HDL-C level but lower serum TC/HDL-C and LDL-C/HDL-C ratios than euglycemic MRHS.

Table 44. Means±SEM of plasma lipoprotein and fibrinogen levels in 506 BHS by sex and Hb levels

Sex	Hb	N	L-particle	M-particle	S-particle	Fibrinogen
g/L						
----- mg/dl. ----->						
M	<130	6	6.5±2.6	132.4±16.4	371.7±29.8	249.5±26.9
	≥130	60	7.1±0.9	176.8±12.0	399.2±12.9	264.2±8.9
	Total	66	7.1±0.9	172.8±11.1	396.7±11.9	262.8±8.4
F	<120	114	4.3±0.4	132.1±6.1	374.7±7.6	257.1±5.2
	≥120	326	5.5±0.4	157.5±4.8	394.5±4.6	278.5±4.1
	Total	440	5.2±0.3	150.9±3.9	398.3±3.9	272.9±3.4

Comparisons were made within the same sex:

al a2 a3
significant difference from <120 g/L: p<0.002, p<0.02, p<0.03.

F
Significant difference from total males: p<0.05.

Table 45. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 506 RHS by sex and Hb levels

Sex	Hb	N	Apoprotein A-I	Apoprotein B
	g/L		<----- mg/dL ----->	
M	<130	6	155.5 $\pm$ 14.4	108.5 $\pm$ 9.7
	$\geq$ 130	60	151.4 $\pm$ 5.3	126.2 $\pm$ 5.0
	Total	66	151.8 $\pm$ 5.0	124.6 $\pm$ 4.7
F	<120	114	146.5 $\pm$ 2.8	107.1 $\pm$ 3.0
	$\geq$ 120	326	148.2 $\pm$ 1.7	113.7 $\pm$ 1.6 ^{a,B}
	Total	440	147.8 $\pm$ 1.4	111.9 $\pm$ 1.4 ^F

Comparisons were made within the same sex:

^a
significant difference from <120 g/L: $p < 0.05$.

Significant difference from males with the corresponding Hb
^B
levels: $p < 0.05$.

^F
Significant difference from total males: $p < 0.02$.

Table 46. Mean±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and PBG levels

Sex	PBG	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC	LDL-C
								HDL-C	HDL-C
mmol/L <----- mmol/L ----->									
M	<7.8	64	5.4±0.2	3.4±0.1	1.3±0.04	1.4±0.1	2.5±0.1	4.3±0.2	2.8±0.2
	≥7.8-11.0	1	6.4	4.6	1.3	1.2	2.5	4.9	3.5
	≥11.1	1	7.6	5.1	1.9	1.4	3.4	4.1	2.7
F	<7.8	392	5.2±0.05	3.2±0.05	1.5±0.03	1.2±0.03	2.8±0.03	3.7±0.1	2.3±0.05
	≥7.8-11.0	51	5.6±0.2	3.5±0.2	1.4±0.04	1.4±0.1	2.9±0.1	4.0±0.2	2.6±0.1
	≥11.1	10	5.2±0.3	3.0±0.3	1.4±0.1	1.8±0.3	2.8±0.2	4.1±0.4	2.4±0.3

Comparisons were made within the same sex:

significant difference from <7.8 mmol/L: a1 a2 a3 p<0.005, p<0.01, p<0.05.
Significant difference from males with the corresponding PBG groups: A1 A2 p<0.005, p<0.01.

Table 47 shows that FRHS with IGT had significantly higher plasma M-particle level than euglycemic FRHS whereas euglycemic FRHS had significantly lower plasma L- and M-particle levels than euglycemic MRHS.

Table 48 shows that FRHS with IGT and diabetic FRHS had significantly higher serum apo B levels than euglycemic FRHS whereas euglycemic FRHS had significantly lower serum apo B level than euglycemic MRHS.

Table 49 shows that FRHS with PBG <7.8 mmol/L had significantly lower age, weight, BMI, Body fat expressed as kg, waist circumference and WHR than FRHS with PBG ≥ 7.8 mmol/L.

Table 50 shows that FHS with PBG <7.8 mmol/L had significantly lower serum TC, TG, TC/HDL-C and LDL-C/HDL-C, apo B plasma M-particle, and FBG than FRHS with PBG ≥ 7.8 mmol/L.

Influence of blood pressure on serum lipid, apo, lipoprotein and fibrinogen levels

Table 51 shows that when RHS were categorized into normal blood pressure (BP) (systolic BP ≤ 140 and diastolic BP ≤ 90 mmHg), borderline hypertension (systolic BP >140 - 160 and diastolic BP >90 - 95 mmHg), and definite hypertension (systolic BP >160 and diastolic BP >95 mmHg),

Table 47. Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and

PBG levels

Sex	PBG	N	L-particle	M-particle	S-particle	Fibrinogen
mmol/L						
<----- mg/dL ----->						
M	<7.8	64	7.1±0.9	171.9±14.4	392.8±11.9	260.5±8.4
	>7.8-11.0	1	4.5	188.4	477.1	371.5
	>11.1	1	8.1	210.8	567.1	307.6
F	<7.8	392	5.1±0.3 ^A	146.3±4.1 ^A	385.7±4.2	272.2±3.6
	>7.8-11.0	51	5.5±0.5	172.7±12.0 ^a	404.8±12.2	274.2±9.2
	>11.1	10	4.3±0.7	188.4±30.8	378.3±19.9	285.3±22.8

Comparisons were made within the same sex:

^a significant difference from <7.8 mmol/L: p<0.05.

^A Significant difference from males with the corresponding PBG groups: p<0.05.

Table 48. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and PBG levels

Sex	PBG	N	Apoprotein A-I	Apoprotein B
	mmol/L		←————— mg/dL —————→	
M	<7.8	64	151.8±5.1	124.4±4.8
	≥7.8-11.0	1	187.0	132.0
	≥11.1	1	117.0	129.0
F	<7.8	392	148.0±1.5	109.5±1.5 ^A
	≥7.8-11.0	51	147.3±4.1	122.9±4.3 ^a
	≥11.1	10	156.6±13.5	142.4±9.4 ^a

Comparisons were made within the same sex:

significant difference from <7.8 mmol/L: ^a p<0.01.

Significant difference from males with the corresponding PBG groups:

^A p<0.005.

Table 49. Age and anthropometric parameters in 453 FHHS by PBG criteria

Parameters	Mean±SEM	
	PBG <7.8 mmol/L n=392	PBG ≥7.8 mmol/L n=61
Age	37.1±0.5	42.1±1.1 ^{a1}
Height (m)	1.65±0.03	1.55±0.05
Weight (kg)	54.7±0.5	57.8±1.1 ^{a3}
BMI (kg/m ²)	22.9±0.2	23.9±0.4 ^{a4}
Body fat (% body weight)	37.8±0.2	38.8±0.4
Body fat (kg)	20.9±0.3	22.5±0.6 ^{a4}
Waist (cm)	75.6±0.5	79.9±1.1 ^{a2}
Hip (cm)	92.7±0.3	94.1±0.7
WHR	0.81±0.004	0.85±0.1 ^{a2}

Significant difference from normal subject: ^{a1} p<0.0005, ^{a2} p<0.005,
^{a3} p<0.02, ^{a4} p<0.05

Table 50. Serum lipid levels, serum lipid ratios, plasma lipoprotein, fibrinogen level, serum apoprotein levels, serum uric acid, serum creatinine, FBG, PBG and blood pressure levels in 453 FRHS by PBG criteria

Lipid status	Mean±SEM	
	PBG <7.8 mmol/L n=392	PBG ≥7.8 mmol/L n=61
TC (mmol/L)	5.2±0.05	5.5±0.1 ^{a4}
LDL-C (mmol/L)	3.2±0.05	3.4±0.1
HDL-C (mmol/L)	1.5±0.03	1.4±0.04
TG (mmol/L)	1.2±0.03	1.5±0.1 ^{a1}
Phospholipid (mmol/L)	2.8±0.03	2.9±0.1
TC/HDL-C	3.7±0.1	4.1±0.2 ^{a3}
LDL-C/HDL-C	2.3±0.05	2.6±0.1 ^{a4}
L-particle (mg/dL)	5.1±0.3	5.3±0.4
M-particle (mg/dL)	146.3±4.1	175.3±11.2 ^{a3}
S-particle (mg/dL)	385.7±4.2	400.4±10.7
Fibrinogen (mg/dL)	272.2±3.6	276.1±8.5
Apoprotein A-I (mg/dL)	147.9±1.5	149.2±4.1
Apoprotein B (mg/dL)	109.5±1.5	126.4±4.0 ^{a1}
Uric acid (mg/dL)	4.8±0.1	4.9±0.2
Creatinine (mg/dL)	0.7±0.01	0.7±0.02
FBG (mmol/L)	4.9±0.02	5.4±0.1 ^{a2}
SBP (mmHg)	107.3±0.6	110.0±1.7
DBP (mmHg)	69.9±0.5	72.5±1.2

Significant difference from normal subject: ^{a1} p<0.0005, ^{a2} p<0.005,
^{a3} p<0.02, ^{a4} p<0.05

Table 51. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RHS by sex and blood pressure categories

Sex	Blood pressure	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC/HDL-C	LDL-C/HDL-C
(<----- mmol/l. ----->)									
M	Normal	63	5.3±0.1	3.4±0.1	1.4±0.04	1.4±0.1	2.5±0.1	4.2±0.2	2.7±0.2
	Borderline	1	7.3	5.4	1.4	1.0	4.0	5.1	3.8
	Defenite	2	7.7±0.9 ^{a1}	5.4±0.6 ^{a2}	1.3±0.4	2.2±1.4	3.5±0.2 ^{a3}	6.7±2.6 ^{a3}	4.7±1.8 ^{a3}
F	Normal	445	5.3±0.1	3.2±0.05	1.5±0.02	1.2±0.3	2.8±0.03	3.7±0.1	2.3±0.04
	Borderline	1	4.6	2.3	1.4	2.2	2.8	3.4	1.7
	Defenite	7	5.8±0.5	3.8±0.4	1.1±0.1 ^{a3}	2.1±0.5	3.0±0.2	3.9±0.9 ^{a3}	3.8±0.6 ^{a1}
M&F	Normal	508	5.3±0.05	3.3±0.05	1.5±0.02	1.2±0.1	2.8±0.03	3.8±0.1	2.4±0.04
	Borderline	2	6.0±1.3	3.8±0.2	1.4±0.02	1.6±0.6	3.4±0.6	4.3±0.9	2.7±1.1
	Defenite	9	6.2±0.5 ^{a1}	4.1±0.4 ^{a2}	1.1±0.1 ^{a3}	2.2±0.4	3.0±0.1	6.0±0.8 ^{a3}	3.9±0.6 ^{a2}

Comparisons were made within the same sex:

significant difference from normal: ^{a1} p<0.01, ^{a2} p<0.02, ^{a3} p<0.05.

Significant difference from males with the corresponding blood pressure categories: ^{A1} p<0.0005, ^{A2} p<0.01, ^{A3} p<0.02, ^{A4} p<0.05.

MRHS with definite hypertension had significantly higher serum TC, LDL-C, and PL levels as well as serum TC/HDL-C and LDL-C/HDL-C ratios than normal MRHS; FRHS with definite hypertension had significantly higher serum TC/HDL-C and LDL-C/HDL-C ratios but lower serum HDL-C level than normal FRHS; combined MRHS and FRHS with definite hypertension had significantly higher serum TC and LDL-C levels as well as TC/HDL-C and LDL-C/HDL-C ratios but lower serum HDL-C level than normal subjects whereas normal FRHS had significantly higher serum HDL-C and PL levels but lower serum TC/HDL-C and LDL-C/HDL-C ratios than normal MRHS.

Table 52 shows that MRHS and combined MRHS and FRHS with definite hypertension had significantly higher plasma S-particle level than normal subjects whereas normal FRHS had significantly lower plasma L- and M-particle levels than normal MRHS.

Table 53 shows that combined MRHS and FRHS with borderline hypertension had significantly higher serum apo A-I and apo B levels than normal subjects; combined MRHS and FRHS with definite hypertension had significantly lower serum apo A-I level than those with normal blood pressure; and normal FRHS had significantly lower serum apo B level than normal MRHS.

Table 52. Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHS by sex and blood pressure categories

Sex	Blood pressure	N	L-particle	M-particle	S-particle	Fibrinogen
← mg/dL →						
M	Normal	63	7.1±0.9	172.0±11.3	338.6±11.5	262.6±8.9
	Borderline	1	3.6	199.6	538.9	317.2
	Defenite	2	8.6±5.8	182.8±117.6	580.8±53.8 ^{a1}	243.0±33.4
F	Normal	445	^A 5.1±0.3	^A 149.3±3.9	387.2±3.9	272.8±3.3
	Borderline	1	1.8	166.0	334.1	200.9
	Defenite	7	4.5±0.4	205.1±22.3	422.8±35.6	273.4±30.2
M&F	Normal	508	5.4±0.3	152.1±3.7	387.4±3.7	271.6±3.1
	Borderline	2	2.7±0.9	182.8±16.8	436.5±102.4 ^{a2}	257.0±56.2
	Defenite	9	5.4±1.2	200.2±26.2	457.9±36.9	266.6±24.2

Comparisons were made within the same sex:

significant difference from normal: ^{a1} p<0.01, ^{a2} p<0.02.

Significant difference from males with the corresponding blood pressure categories: ^{A1} p<0.05.

Table 53. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and blood pressure categories

Sex	Blood pressure	N	Apoprotein A-I	Apoprotein B
<----- mg/dL ----->				
M	Normal	63	151.4 $\pm$ 5.1	121.6 $\pm$ 4.3
	Borderline	1	206.0	127.0
	Defenite	2	137.5 $\pm$ 5.5	194.0 $\pm$ 6.6
F	Normal	445	148.1 $\pm$ 1.4	111.3 $\pm$ 1.5 ^A
	Borderline	1	246.0	179.0
	Defenite	7	135.2 $\pm$ 1.5	133.3 $\pm$ 7.3
M&F	Normal	508	148.5 $\pm$ 1.3 ^{a1}	112.5 $\pm$ 1.3 ^{a2}
	Borderline	2	226.0 $\pm$ 20.0 ^b	178.0 $\pm$ 1.0
	Defenite	9	135.7 $\pm$ 11.8	146.8 $\pm$ 15.2

Comparisons were made within the same sex:

a1 a2

significant difference from normal: $p < 0.002$, $p < 0.005$;

b

significant difference from borderline: $p < 0.01$.

Significant difference from males with the corresponding blood pressure categories: ^A $p < 0.02$.

**Serum lipid, apo, plasma lipoprotein and fibrinogen levels
in RHS by corneal arcus**

Table 54 shows that MRHS with corneal arcus had significantly higher serum PL level than those without corneal arcus; FRHS with corneal arcus had significantly higher serum TC, LDL-C, HDL-C and PL levels than those without corneal arcus; and FRHS without corneal arcus had significantly higher serum HDL-C and PL levels but lower serum TC/HDL-C ratio than MRHS without corneal arcus.

Table 55 shows that FRHS with corneal arcus had significantly higher plasma S-particle level than FRHS without corneal arcus whereas FRHS without corneal arcus had significantly lower plasma L-particle level than MRHS without corneal arcus.

Table 56 shows that FRHS with corneal arcus had significantly higher serum apo B level than FRHS without corneal arcus whereas FRHS without corneal arcus had significantly lower serum apo B level than MRHS without corneal arcus.

Table 57 shows that MRHS with corneal arcus had significantly higher age, waist circumference and WHR but lower height than MRHS without corneal arcus; FRHS with corneal arcus had significantly higher age, and BMI but lower height than FRHS without corneal arcus; FRHS with corneal arcus had significantly lower weight and height

Table 54. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 519 RBS by sex and corneal arcus

Sex	Corneal arcus	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC		LDL-C	
								HDL-C		HDL-C	
<----- mmol/L ----->											
M	Negative	54	5.3±0.1	3.3±0.1	1.3±0.04	1.3±0.1	2.5±0.1		4.1±0.2	2.6±0.2	
	Positive	12	6.1±0.5	4.0±0.5	1.3±0.1	1.5±0.3	2.9±0.2		5.2±0.7	3.5±0.6	
F	Negative	407	5.2±0.1	3.2±0.1	1.5±0.03	1.2±0.03	2.8±0.03		3.7±0.1	2.3±0.04	
	Positive	46	6.0±0.2	3.8±0.2	1.6±0.04	1.3±0.1	3.1±0.1		3.9±0.1	2.5±0.1	

Comparisons were made within the same sex:

significant difference from negative corneal arcus: $p<0.0005$, $p<0.002$, $p<0.01$, $p<0.05$.

Significant difference the corresponding group of males with or without corneal arcus: $p<0.005$, $p<0.05$.

Table 55. Means±SEM of plasma lipoprotein and fibrinogen levels in 519 RHB by sex and corneal arcus

Sex	Corneal arcus	N	L-particle	M-particle	S-particle	Fibrinogen
$\leftarrow \text{mg/dL} \rightarrow$						
M	Negative	54	7.2±1.0	165.6±11.04	386.2±11.2	259.7±9.0
	Positive	12	6.4±1.6	205.2±35.3	443.2±41.2	276.8±22.5
F	Negative	407	5.1±0.3 ^A	148.5±4.1	380.9±3.9 ^a	273.2±3.4
	Positive	46	5.4±0.9	165.1±10.4	447.5±14.4	268.3±12.5

Comparisons were made within the same sex:

significant difference from negative corneal arcus: p<0.0005.^a

Significant difference the corresponding group of males with or without corneal arcus: p<0.05.^A

Table 56. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 519 RHS by sex and corneal arcus

Sex	Corneal arcus	N	Apoprotein A-I	Apoprotein B
<----- mg/dL ----->				
M	Negative	54	149.2 $\pm$ 4.4	121.2 $\pm$ 4.8
	Positive	12	163.4 $\pm$ 19.3	139.9 $\pm$ 13.7
F	Negative	407	147.4 $\pm$ 1.5	109.9 $\pm$ 1.5 ^A
	Positive	46	154.3 $\pm$ 4.5	127.5 $\pm$ 4.3 ^a

Comparisons were made within the same sex:

a

significant difference from negative corneal arcus: $p < 0.0005$.

Significant difference the corresponding group of males with or without corneal arcus: ^A $p < 0.02$.

Table 57. Means±SEM of age and anthropometric levels in 513 RHS by sex and corneal arcus

Sex	Corneal arcus	N	Age	Weight	Height	BMI	Circumference		
							Waist	Hip	WHR
			yr	kg	m	kg/m ²	cm	cm	
M	Negative	54	33.8±1.3	61.7±1.4	1.7±0.01	22.3±0.4	73.2±2.1	82.4±5.1	0.9±0.01
	Positive	12	47.5±2.3	62.4±2.9	1.6±0.01	24.2±0.9	82.4±2.5	82.5±7.3	0.9±0.01
F	Negative	407	36.8±0.4	55.0±0.5	1.6±0.02	22.9±0.2	73.8±0.7	80.5±1.4	0.8±0.004
	Positive	46	46.2±1.0	56.7±1.2	1.5±0.01	24.2±0.5	77.7±2.2	76.3±5.0	0.9±0.01

Comparisons were made within the same sex:

significant difference from negative corneal arcus: $p < 0.0005$, $p < 0.005$, $p < 0.002$, $p < 0.01$, $p < 0.05$.

Significant difference the corresponding group of males with or without corneal arcus: $p < 0.0005$, $p < 0.05$, $p < 0.002$, $p < 0.05$.

than MRHS with corneal arcus and similar findings were observed between MRHS and FRHS without corneal arcus.

Influence of levels of education and income on serum lipid, apo, plasma lipoprotein and fibrinogen levels

Table 58 shows that FRHS with university education had significantly lower serum LDL-C level and serum TC/HDL-C and LDL-C/HDL-C ratios but higher serum PL level than FRHS with school education whereas FRHS with university education had significantly lower serum LDL-C but higher serum HDL-C level than MRHS with university education.

Table 59 shows that FRHS with vocational education had significantly higher plasma fibrinogen level than FRHS with school and university education.

Table 60 shows that MRHS with university education had significantly higher serum apo B level than MRHS with school education whereas FRHS with university education had significantly higher serum apo A-I level than MRHS with university education.

Table 61 shows that MRHS with income of ≤ 3000 Baht/month had significantly higher serum HDL-C but lower serum TG levels than MRHS with income of 3001-5000 and >7000 Baht/month, respectively; FRHS with income of 5001-7000 Baht/month had significantly higher serum TG and PL.

Table 58. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and educational level

Sex	Educational levels	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC/HDL-C	LDL-C/HDL-C
←----- mmol/L -----→									
M	School	58	5.3±0.2	3.4±0.2	1.4±0.04	1.4±0.1	2.5±0.1	4.2±0.2	2.7±0.2
	Vocational	2	5.7±0.8	3.9±0.8	1.4±0.1	0.8±0.04	3.0±0.2	4.0±0.4	2.7±0.4
	University	6	5.9±0.5	4.1±0.4	1.2±0.2	1.5±0.3	2.9±0.2	5.3±0.9	3.7±0.7
F	School	221	5.3±0.1	3.3±0.1	1.4±0.04	1.3±0.5	2.7±0.04	3.9±0.1	2.5±0.1
	Vocational	23	5.0±1.0	3.1±0.2	1.4±0.1	1.1±0.1	2.9±0.2	3.6±0.2	2.3±0.1
	University	203	5.2±0.1	3.1±0.1	1.5±0.02	1.2±0.04	2.9±0.04	3.5±0.1	2.2±0.1

Comparisons were made within the same sex: significant difference from school: $p < 0.0005$, $p < 0.05$.
 C1 A2

Significant difference from males with the corresponding educational levels: $p < 0.02$, $p < 0.05$.
 a1 a2

Table 59. Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and educational level

Sex	Educational level	N	L-particle	M-particle	S-particle	Fibrinogen
<----- mg/dL ----->						
M	School	58	6.8±0.9	171.0±11.6	391.9±12.9	263.1±8.9
	Vocational	2	8.6±3.2	171.6±39.2	417.5±61.4	334.8±66.9
	University	6	8.7±3.1	190.3±50.8	435.9±36.4	236.2±20.3
F	School	221	5.2±0.4	155.9±5.6	394.1±5.7	274.1±4.7
	Vocational	23	4.3±1.3	158.2±20.1	370.4±14.7	308.8±19.3 ^a
	University	203	5.2±0.4	144.8±5.6	382.7±5.7	267.9±4.8

Comparisons were made within the same sex:

^a significant difference from school: $p<0.05$;

^b significant difference from vocational education: $p<0.05$.

Table 60. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and educational level

Sex	Educational level	N	Apoprotein A-I		Apoprotein B	
			<----- mg/dL ----->			
M	School	58	154.8 $\pm$ 5.5		120.9 $\pm$ 4.5	
	Vocational	2	143.0 $\pm$ 2.0		122.5 $\pm$ 9.5	
	University	6	125.8 $\pm$ 7.0		160.0 $\pm$ 23.4	^a
F	School	221	146.7 $\pm$ 2.1		114.5 $\pm$ 1.9	
	Vocational	23	138.6 $\pm$ 4.6		116.1 $\pm$ 7.4	
	University	203	150.9 $\pm$ 2.2	^c	108.9 $\pm$ 2.1	

Comparisons were made within the same sex:

^a
significant difference from school: $p < 0.02$.

Significant difference from males with the corresponding educational
^c
levels: $p < 0.05$.

levels than FRHS with income of <3000 Baht/month; FRHS with income of >7000 Baht/month had significantly higher serum HDL-C and PL levels but lower serum TC/HDL-C and LDL-C/HDL-C ratios than FRHS with income of 3001-5000 Baht/month and the former also had significantly higher serum PL and TG levels than FRHS with income of <3000 Baht/month and MRHS with income of >7000 Baht/month, respectively; and FRHS with income of 3001-5000 Baht/month had significantly higher serum HDL-C level than MRHS with the corresponding income.

Table 62 shows that MRHS with income of 3001-5000 Baht/month had significantly higher plasma M-particle levels than MRHS with income of <3000 Baht/month and FRHS with the same income level; FRHS with income of <3000 Baht/month had significantly lower plasma L-particle level than FRHS with income of >7000 Baht/month and significantly lower plasma fibrinogen level than FRHS with income of 3001-5000 and >7000 Baht/month; and FRHS with income of >7000 Baht/month had significantly lower plasma M-particle level than MRHS with the same income.

Table 63 shows that MRHS with income >7000 Baht/month had significantly lower serum apo A-I level than MRHS with income of <3000 and 3001-5000 Baht/month whereas MRHS with income of 5001-7000 Baht/month had significantly higher serum apo B level than MRHS with

Table 62. Means±SEM of plasma lipoprotein and fibrinogen levels in 513 EHS by sex and income

Sex	Income	N	L-particle	M-particle	S-particle	Fibrinogen
	baht/month		mg/dL			
			←-----→			
M	<3000	35	6.2±1.2	153.0±14.0	382.3±14.0	258.4±10.1
	3001-5000	13	9.6±2.4	211.3±28.2 ^{a2}	387.9±29.6	259.4±19.6
	5001-7000	14	5.3±0.8	166.8±19.6	425.1±29.6	281.1±23.9
	>7000	4	12.6±4.8	241.6±64.2	452.4±62.3	248.9±30.1
F	<3000	80	4.1±0.3	142.7±10.9	384.3±8.9	256.6±6.2
	3001-5000	110	4.6±0.3	150.9±6.7 ^B	388.8±7.9	276.9±7.3 ^{a2}
	5001-7000	96	5.5±0.6	160.8±6.9 ^D	393.5±8.9	274.9±7.9 ^{a1}
	>7000	161	5.7±0.6 ^{a1}	149.4±7.1	385.2±6.5	277.6±5.4

Comparisons were made within the same sex:

significant difference from <3000 baht/month: ^{a1} ^{a2} ^B ^D p<0.02, p<0.05.

Significant difference from males with the corresponding income level: p<0.01, p<0.05.

Table 63. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 513 RBS by sex and income

Sex	Income	N	Apoprotein A-I	Apoprotein B
	baht/month		<----- mg/dL ----->	
M	<3000	35	153.5 $\pm$ 5.6	114.6 $\pm$ 4.9
	3001-5000	13	148.9 $\pm$ 10.4	131.6 $\pm$ 13.3 ^{a2}
	5001-7000	14	159.6 $\pm$ 15.9 ^{a2,b}	138.7 $\pm$ 11.1
	>7000	4	118.5 $\pm$ 4.6	139.6 $\pm$ 22.6
F	<3000	80	143.5 $\pm$ 2.8	104.6 $\pm$ 2.9 ^{a2}
	3001-5000	110	145.1 $\pm$ 2.9	114.1 $\pm$ 2.9 ^{a1}
	5001-7000	96	150.4 $\pm$ 3.4 ^{a2,D}	115.4 $\pm$ 2.9
	>7000	161	151.4 $\pm$ 2.4	112.3 $\pm$ 2.5

Comparisons were made within the same sex:

significant difference from <3000 baht/month: ^{a1} p<0.02, ^{a2} p<0.05.

Significant difference from males with the corresponding income^D
level: p<0.05.

income of <3000 Baht/month; FRHS with income of >7000 Baht/month had significantly higher serum apo A-I level than FRHS with income of <3000 Baht/month and MRHS with the same income level; and FRHS with income of <3000 Baht/month had significantly lower serum apo B level than FRHS with income of 3001-5000 and 5001-7000 Baht/month.

Influence of smoking habit on serum lipid, apo, plasma lipoprotein and fibrinogen levels

In this study, smokers included both current and ex-smokers. Out of 43 male smokers and 9 female smokers, 18 and 3 were ex-smokers, respectively.

Table 64 shows that male smokers had significantly higher serum TC and LDL-C levels than male non-smokers; female non-smokers had significantly higher serum HDL-C and PL levels than male non-smokers; and female smokers had significantly lower serum LDL-C level than male smokers.

Table 65 shows that male smokers had significantly higher plasma S-particle level than male non-smokers.

Table 66 shows that male smokers had significantly higher serum apo A-I level than male non-smokers.

Table 67 shows that MRHS smoking <10 cigs/day had significantly higher serum TC level than male non-smokers and had significantly lower serum LDL-C level than men

Table 64. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and smoking habit

Sex	Smoking habit	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC/HDL-C	LDL-C/HDL-C
<----- mmol/L ----->									
M	Non-Smoker	23	4.9±0.2	3.0±0.2	1.2±0.1	1.3±0.2	2.4±0.1	4.3±0.3	2.7±0.2
	Smoker	43	5.7±0.2	3.6±0.2	1.4±0.1	1.4±0.1	2.6±0.1	4.4±0.3	2.8±0.2
F	Non-Smoker	438	5.3±0.1	3.3±0.04	1.5±0.02	1.5±0.03	2.8±0.03	3.8±0.5	2.3±0.04
	Smoker	9	5.0±0.2	2.7±0.3	1.5±0.1	1.6±0.4	2.8±0.2	3.6±0.4	2.0±0.3

Comparisons were made within the same sex:
al a2
significant difference from non-smoker: p<0.01, p<0.02.
A1 A2 B
Significant difference from males with the corresponding smoking habit: p<0.005, p<0.01, p<0.05.
*18 men and 3 women were ex-smokers.

Table 66. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and smoking habit

Sex	Smoking habit	N	Apoprotein A-I	Apoprotein B
			$\leftarrow$ ————— ng/dL ————— $\rightarrow$	
M	Non-Smoker	23	137.9 $\pm$ 6.5	123.6 $\pm$ 8.9
	Smoker	43	159.2 $\pm$ 6.6 ^a	125.1 $\pm$ 5.4
F	Non-Smoker	438	148.3 $\pm$ 1.5	111.9 $\pm$ 1.4
	Smoker	9	145.6 $\pm$ 7.7	114.6 $\pm$ 13.5

Comparisons were made within the same sex:

significant difference from non-smoker: ^a p<0.05.

Table 67. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 MHS by sex and intensity of smoking

Sex	Intensity of smoking	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC	LDL-C
	cigs/day								

Comparisons were made within the same sex:

al a2 a3
significant difference from 0 cig/day: p<0.0005, p<0.005, p<0.05;
b1 b2 b3

significant difference from <10 cigs/day: p<0.002, p<0.02, p<0.05.

Al A2 C
Significant difference from males with the corresponding smoking intensity: p<0.005, p<0.01, p<0.01.

smoking >20 cigs/day whereas MRHS smoking >20 cigs/day had significantly higher serum TC and LDL-C levels than MRHS smoking 10-20 cigs/day; FRHS smoking 10-20 cigs/day had significantly higher serum TG level than female non-smokers, FRHS smoking <10 cigs/day, and MRHS smoking 10-20 cigs/day but significantly lower serum PL level than female non-smokers; female non-smokers had significantly higher serum HDL-C and PL levels than male non-smokers.

Table 68 shows that MRHS smoking <10 cigs/day had significantly higher plasma S-particle level than male non-smokers whereas FRHS smoking 10-20 cigs/day had significantly higher plasma M-particle level than female non-smokers, FRHS smoking <10 cigs/day, and MRHS smoking 10-20 cigs/day.

Table 69 shows that MRHS smoking <10 cigs/day had significantly higher serum apo A-I level than male non-smokers.

Influence of alcohol consumption on serum lipids, apo, plasma lipoprotein and fibrinogen levels

Table 70 shows that MRHS drinking alcohol had significantly higher serum HDL-C level than male non-drinkers whereas female non-drinkers had significantly higher serum HDL-C level but lower serum TC/HDL-C and LDL-C/HDL-C ratios than male non-drinkers.

Table 68. Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and intensity of smoking

Sex	Intensity of smoking	N	L-particle	M-particle	S-particle	Fibrinogen
cigs/day <----- mg/dL ----->						
M	0	23	8.1±2.0	180.1±21.4	353.8±15.7	260.4±15.8
	<10	26	6.2±0.8	161.0±15.9	409.9±17.4 ^{a2}	257.6±10.2
	10-20	13	7.3±1.9	186.2±27.9	422.1±35.1	274.9±24.4
	>20	4	5.8±1.0	163.2±16.1	474.9±44.9	271.1±32.2
F	0	438	5.2±0.3	156.0±3.8	388.3±3.9	272.8±3.4
	<10	7	4.5±1.5	133.1±25.3 ^{a1,b,c}	364.1±18.3	293.1±25.0
	10-20	2	4.1±0.5	435.3±82.6	329.6±63.6	259.2±6.5

Comparisons were made within the same sex:

significant difference from 0 cig/day: ^{a1} p<0.0005, ^{a2} p<0.005;

significant difference from <10 cigs/day: ^b p<0.01.

Significant difference from males with the corresponding smoking intensity: ^c p<0.02.

Table 69. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and intensity of smoking

Sex	Intensity of smoking	N	Apoprotein A-I	Apoprotein B
	cigs/day		<----- mg/dL ----->	
M	0	23	137.9 $\pm$ 6.5	123.5 $\pm$ 8.9
	<10	26	166.6 $\pm$ 8.9 ^a	124.8 $\pm$ 7.6
	10-20	13	143.9 $\pm$ 9.7	126.7 $\pm$ 9.1
	>20	4	160.5 $\pm$ 24.6	122.5 $\pm$ 8.5
F	0	438	148.3 $\pm$ 1.4	111.9 $\pm$ 1.4
	<10	7	143.8 $\pm$ 6.5	109.5 $\pm$ 15.3
	10-20	2	151.5 $\pm$ 34.5	132.4 $\pm$ 35.6

Comparisons were made within the same sex:

^a
significant difference from 0 cig/day: $p < 0.02$.

Table 70. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and alcohol consumption

Sex	Alcohol consumption	N	TC	LDL-C	HDL-C	TG	Phospholipid	mmol/L		LDL-C	HDL-C
								TC	HDL-C		
M	Non-drinker	22	5.5±0.3	3.6±0.3	1.2±0.1	1.6±0.2	2.6±0.1	4.8±0.4	3.1±0.3		
	Drinker [*]	44	5.3±0.2	3.3±0.2	1.4±0.1 ^a	1.3±0.1	2.5±0.1	4.1±0.3	2.6±0.2		
F	Non-drinker	401	5.3±0.1	3.2±0.1	1.5±0.03	1.2±0.03	2.8±0.03	3.8±0.1 ^{A2}	2.3±0.04		
	Drinker ⁺	46	5.3±0.1	3.3±0.2	1.5±0.1	1.3±0.1	2.7±1.0	3.8±0.2	2.4±0.2		

*Out of 44 male alcohol drinkers, 10 drank alcohol daily and 34 drank alcohol occasionally.
†Out of 46 female alcohol drinkers, 2 drank alcohol daily and 44 drank alcohol occasionally.

Comparisons were made within the same sex: significant difference from non-drinker: p<0.05.
Significant difference from males with the corresponding alcohol consumption: p<0.002, p<0.01.

Table 71 shows that female non-drinkers had significantly lower plasma M-particle level than male non-drinkers; female drinkers had significantly lower plasma L-particle level but higher plasma fibrinogen level than male drinkers; and female non-drinkers had significantly lower plasma M-particle level than male non-drinkers.

Table 72 shows that male drinkers had significantly higher serum apo A-I and apo B levels than male non-drinkers whereas female non-drinkers had significantly higher serum apo A-I level but lower apo B level than male non-drinkers.

Influence of coffee consumption on serum lipids, apo, plasma lipoprotein and fibrinogen levels

Table 73 shows that MRHS drinking coffee had significantly higher serum PL level than male non-drinkers; FRHS drinking coffee had significantly higher serum TG level than female non-drinkers and had significantly higher serum HDL-C level but lower serum TC/HDL-C and LDL-C/HDL-C ratios than MRHS drinking coffee; and female non-drinkers had significantly higher serum PL level than male non-drinkers.

Table 74 shows that women drinking coffee had significantly lower plasma L-particle level than men drinking coffee.

Table 72. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and alcohol consumption in each sex

Sex	Alcohol consumption	N	Apoprotein A-I	Apoprotein B
			mg/dL	
M	Non-drinker	22	134.4 $\pm$ 5.8	140.4 $\pm$ 10.1
	Drinker	44	160.5 $\pm$ 6.6	116.7 $\pm$ 4.5
F	Non-drinker	401	149.0 $\pm$ 1.5	111.5 $\pm$ 1.5
	Drinker	46	141.4 $\pm$ 4.4	116.9 $\pm$ 4.8

Comparisons were made within the same sex:

significant difference from non-drinker: ^{a1} ^{a2} p<0.005, p<0.05.

Significant difference from males with the corresponding alcohol

consumption: ^{A1} ^{A2} p<0.02, p<0.05.

Table 73. Means±SEM of serum lipid levels, TC/HDL-C and LDL-C/HDL-C ratios in 513 RHS by sex and coffee consumption

Sex	Coffee consumption	N	TC	LDL-C	HDL-C	TG	Phospholipid	TC		LDL-C	
								TC	HDL-C	HDL-C	HDL-C
<----- mmol/L ----->											
M	Non-drinker	20	5.3±0.2	3.3±0.2	1.4±0.1	1.3±0.2	2.3±0.1	3.9±0.3	2.5±0.2		
	Drinker [*]	46	5.5±0.2	3.5±0.2	1.3±0.1	1.4±0.1	2.6±0.1 ^a	4.5±0.3	2.9±0.2		
F	Non-drinker	85	5.2±0.1	3.2±0.1	1.6±0.1	1.1±0.4	2.9±0.1 ^A	3.7±0.1	2.3±0.1		
	Drinker ⁺	362	5.3±0.1	3.3±0.1	1.5±0.02 ^{B1}	1.3±0.04 ^a	2.8±0.03	3.8±0.1 ^B	2.3±0.1		

*Out of 46 male coffee drinkers, 13 drank coffee daily and 33 drank coffee occasionally.
+Out of 362 female coffee drinkers, 151 drank coffee daily and 211 drank coffee occasionally.

Comparisons were made within the same sex: significant difference from non-drinker: p<0.05.
Significant difference from males with the corresponding coffee consumption: p<0.0005, p<0.005, p<0.02.

^a A B1 B2

Table 74. Means±SEM of plasma lipoprotein and fibrinogen levels in 513 Rhs by sex and coffee consumption

Sex	Coffee consumption	N	L-particle	M-particle	S-particle	Fibrinogen
mg/dL						
M						
	Non-drinker	20	6.2±1.6	156.7±14.1	403.1±23.2	228.3±16.6
	Drinker	46	7.4±1.0	179.8±14.7	393.9±14.1	254.4±9.5
F						
	Non-drinker	85	4.7±0.4	142.8±6.7	387.9±9.8	271.5±7.3
	Drinker	362	5.2±0.3	152.9±4.6	387.7±4.3	273.5±3.8

Significant difference from males with the corresponding coffee consumption: p<0.05.

B

Table 75 shows that women drinking coffee had significantly lower serum apo B level than men drinking coffee.

Influence of physical activity on serum lipids, apo, plasma lipoprotein and fibrinogen levels

Out of 47 MRHS who exercised, 9 exercised daily and 38 exercised occasionally whereas out of 270 FRHS who exercised, 30 exercised daily and 240 exercised occasionally.

Table 76 shows that MRHS who exercised had significantly lower serum TC and LDL-C levels than MRHS who did not exercise; FRHS who exercised had significantly lower serum TC/HDL-C and LDL-C/HDL-C ratios than FRHS who did not exercise but higher serum HDL-C and PL levels than MRHS who exercised; and FRHS who did not exercise had significantly lower serum TC and LDL-C levels as well as serum TC/HDL-C and LDL-C/HDL-C ratios than MRHS who did not exercise.

Table 77 shows that MRHS who exercised had significantly lower plasma S-particle level than MRHS who did not exercise whereas FRHS who exercised and FRHS who did not exercise had significantly lower plasma L- and S-particle levels than the corresponding groups of MRHS, respectively.

Table 75. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 513 EHS by sex and coffee consumption

Sex	Coffee consumption	N	Apoprotein A-I		Apoprotein B	
			mg/dL			
M	Non-drinker	20	160.2 $\pm$ 11.5		120.7 $\pm$ 5.5	
	Drinker	46	148.1 $\pm$ 5.1		126.3 $\pm$ 6.3	
F	Non-drinker	85	153.8 $\pm$ 3.6		111.7 $\pm$ 3.3	
	Drinker	362	146.9 $\pm$ 1.6		112.1 $\pm$ 1.6	^B

Significant difference from males with the corresponding coffee consumption: ^B p<0.05.

Table 77. Means±SEM of plasma lipoprotein and fibrinogen levels in 513 RHS by sex and exercise

Sex	Exercise	N	L-particle	M-particle	S-particle	Fibrinogen
$\leftarrow$ -----mg/dL----- $\rightarrow$						
M	No	19	6.2±0.9	175.4±16.4	460.8±26.9	272.5±15.6
	Yes	47	7.4±1.2	171.7±14.2	370.8±10.9 ^a	258.9±10.0
F	No	177	5.6±0.5	155.6±7.1	393.7±6.6 ^A	274.9±5.3
	Yes	270	4.9±0.3	148.0±4.5	383.8±4.8 ^B	271.9±4.3

Comparisons were made within the same sex: ^a

significant difference from non-exercise: $p<0.01$.

Significant difference from males with the corresponding exercise: $p<0.005$, $p<0.05$. ^A ^B

Table 78 shows that FRHS who exercised had significantly lower serum apo B level than FRHS who did not exercise whereas FRHS who did not exercise had significantly lower serum apo B level than men who did not exercise.

Study in 241 dyslipidemic RHS

FA composition of total serum lipids were determined in 241 (46.4%) out of 519 RHS.

Table 79 shows that when 241 RHS were categorized into normolipidemia and dyslipidemia. The prevalences of normolipidemia decreased with age.

Since the number of MRHS in this group was small analysis of data was only carried out in 229 FRHS.

Table 80 shows that when 229 FRHS were categorized into 113 normolipidemia and 116 dyslipidemic, normolipidemic FRHS had significantly lower age, weight, BMI, body fat in kg and WHR than dyslipidemic FRHS.

Table 81 shows that normolipidemic FRHS had significantly lower serum TC, LDL-C, TG, and PL levels, serum TC/HDL-C and LDL-C/HDL-C ratios, plasma M- and S-particle, and serum apo A-I and apo B levels than dyslipidemic FRHS.

Table 78. Means $\pm$ SEM of serum apoprotein A-I and apoprotein B levels in 513 RHS by sex and exercise

Sex	Exercise	N	Apoprotein A-I	Apoprotein B
<----- mg/dL ----->				
M	No	19	167.5 $\pm$ 12.5	136.2 $\pm$ 9.2
	Yes	47	145.4 $\pm$ 4.7	119.9 $\pm$ 5.3
F	No	177	146.9 $\pm$ 2.4	116.0 $\pm$ 2.4 ^A
	Yes	270	149.0 $\pm$ 1.8	109.4 $\pm$ 1.7 ^a

Comparisons were made within the same sex:

^a
significant difference from non-exercise: $p < 0.05$.

Significant difference from males with the corresponding exercise:

^A
 $p < 0.02$.

Table 79. Prevalences of dyslipidemia and lipoprotein phenotypes in 241 RHS by sex and age

Sex	Age	N	Normal	TC>5.2 mmol/L only	TC>5.2 & LDL-C>3.4 mmol/L	TG>2.3 mmol/L only	TC>5.2 & TG>2.3 mmol/L	HDL-C<0.9 mmol/L only						
	yr		N	%	N	%	N	%	N	%				
M	20-29	4	1	25.0	-	-	-	-	-	-				
	30-39	4	1	25.0	-	-	1	25.0	-	-				
	40-49	3	-	-	-	1	33.3	1	33.3	-				
	50-60	1	-	-	-	-	-	-	-	-				
	Total	12	2	16.7	-	6	50.0	3	25.0	-	-			
F	19-29	43	25	58.1	4	9.3	13	30.2	-	-	1	2.3		
	30-39	81	43	53.1	3	3.7	1	38.3	1	1.2	2	25.0	1	1.2
	40-49	79	39	49.4	5	6.3	26	32.9	-	-	5	6.3	4	5.1
	50-61	26	6	23.1	2	7.7	14	53.8	2	7.7	1	3.8	1	3.8
	Total	229	113	49.3	14	6.1	84	36.7	3	1.3	8	3.5	7	3.1

Table 80. Age and anthropometric parameter in 229 FRHS by dyslipidemic criteria

Parameter	Mean±SEM	
	Normolipidemia n=113	Dyslipidemia n=116
Age	36.3±0.8	40.0±0.9 ^{a2}
Height (m)	1.56±0.005	1.56±0.005
Weight (kg)	54.2±0.8	57.5±0.8 ^{a2}
BMI (kg/m ²)	22.2±0.3	23.8±0.4 ^{a1}
Body fat (% body weight)	38.1±0.4	38.9±0.4
Body fat (kg)	20.7±0.4	22.6±0.5 ^{a2}
Waist (cm)	74.0±1.0	76.0±1.1
Hip (cm)	78.8±2.9	78.2±3.1
WHR (cm)	0.80±0.01	0.83±0.01 ^{a3}

Significant difference from normolipidemic FRHS: ^{a1} p<0.0005,
^{a2} p<0.005, ^{a3} p<0.01.

Table 81. Serum lipid levels, serum lipid ratios, plasma lipoprotein and fibrinogen levels, and serum apoprotein levels in 229 FHHS by dyslipidemic criteria

Parameter	Mean±SEM	
	Normolipidemia n=113	Dyslipidemia n=116
TC (mmol/L)	4.5±0.05	6.3±0.1 ^{a1}
LDL-C (mmol/L)	2.6±0.05	4.1±0.1 ^{a1}
HDL-C (mmol/L)	1.5±0.03	1.6±0.01 ^{a1}
TG (mmol/L)	1.1±0.04	1.5±0.1 ^{a1}
Phospholipid (mmol/L)	2.7±0.1	3.1±0.1 ^{a1}
TC/HDL-C	3.2±0.1	4.4±0.1 ^{a1}
LDL-C/HDL-C	1.8±0.1	2.9±0.1 ^{a1}
L-particle (mg/dL)	5.1±0.3	6.4±0.7 ^{a1}
M-particle (mg/dL)	136.9±5.7	189.4±9.7 ^{a1}
S-particle (mg/dL)	332.9±4.3	456.5±6.9 ^{a1}
Fibrinogen (mg/dL)	264.9±5.0	275.8±6.5 ^{a2}
Apo A-I (mg/dL)	145.1±2.7	155.8±2.9 ^{a1}
Apo B (mg/dL)	95.1±2.2	130.8±2.5 ^{a1}

Significant difference from normolipidemic FHHS: ^{a1} p<0.0005, ^{a2} p<0.01.

Table 82 shows that normolipidemic FRHS had significantly lower serum 14:0, 16:0, and 20:3 n-6 levels but higher serum 18:2 n-6, 20:4 n-6 and 20:5 n-3 levels than dyslipidemic FRHS.

Effect of dietary intervention on lipid status in dyslipidemic RHS

Serum lipid, plasma lipoprotein and fibrinogen, and apoprotein levels

Twenty-seven dyslipidemic MRHS and 189 dyslipidemic FRHS (Table 24) were invited for repeating their serum lipid determination. However, only 9 dyslipidemic MRHS (33%) and 101 dyslipidemic FRHS (53%) participated in the second examination. The intervals between the first and second visits ranged from 6 to 25 wks (mean $\pm$ SD = 14 $\pm$ 6 wks). Table 83 shows that majority of dyslipidemic types in 110 RHS were type IIa and IIb hyperlipoproteinemia. At the second visit, all of these 110 RHS were instructed to change their dietary habits individually by Professor Vichai Tanphaichitr. The dietary instruction included (a) avoiding intakes of offal, skin, fatty foods, sweetmeat, sweet fruits and menus containing coconuts, (b) promoting the use of soybean oil for 2-3 table spoons in daily cooking, (c) allowing to take one egg daily in the form of fried

Table 82. Relative FA composition of total serum lipids in 229 FHHS by dyslipidemic criteria

Fatty acid	Mean±SEM	
	Normolipidemia n=113	Dyslipidemia n=116
<---- % of total fatty acids ---->		
NEFA		
14:0	0.7±0.02	0.8±0.02 ^{a2}
16:0	27.0±0.3	27.9±0.3 ^{a5}
16:1 n-7	1.2±0.04	1.3±0.03
18:0	11.5±0.1	11.9±0.1
20:0	0.5±0.03	0.5±0.02
18:1 n-9	12.4±0.2	12.1±0.2
20:3 n-9	0.4±0.01	0.4±0.01
RFA		
18:2 n-6	21.1±0.3	19.7±0.3 ^{a3}
20:3 n-6	1.6±0.05	2.1±0.1 ^{a1}
20:4 n-6	9.3±0.1	8.8±0.1 ^{a4}
18:3 n-3	1.7±0.02	1.7±0.03
20:5 n-3	0.7±0.02	0.6±0.02 ^{a2}
22:5 n-3	0.7±0.02	0.7±0.02
22:6 n-3	6.4±0.1	6.3±0.1
20:3 n-9	0.04±0.001	0.04±0.004
20:4 n-6		

Significant difference from normolipidemic FHHS: ^{a1} p<0.0005,
^{a2} p<0.002, ^{a3} p<0.005, ^{a4} p<0.01, ^{a5} p<0.02

Table 83. Prevalences of dyslipidemic types in 110 RHS on the first visit by sex and age

Sex	Age	N	Normal	TC $\geq$ 5.2 mmol/L only	TC $\geq$ 5.2, LDL-C $\geq$ 3.4 & TG $\geq$ 2.3 mmol/L	TG $\geq$ 2.3 mmol/L only	TC $\geq$ 5.2, LDL-C $\geq$ 3.4 & TG $\geq$ 2.3 mmol/L	HDL-C $\leq$ 0.9 mmol/L only	
	yr	N	%	N	%	N	%	N	%
M	20-29	3	-	-	2 66.7	-	1 33.3	-	-
	30-39	3	-	-	2 66.7	-	1 33.3	-	-
	40-49	2	-	-	1 50.0	-	1 50.0	-	-
	50-60	1	-	-	1 100.0	-	-	-	-
	Total	9	-	-	6 66.7	-	3 33.3	-	-
F	19-29	14	-	3 21.4	11 78.6	-	-	-	-
	30-39	32	-	2 6.3	26 81.3	1 3.1	2 6.3	1 3.1	3.1
	40-49	35	-	5 14.3	23 65.7	-	5 14.3	2 5.7	5.7
	50-61	20	-	2 10.0	14 70.0	2 10.0	1 5.0	1 5.0	5.0
	Total	101	-	12 11.9	74 73.3	3 3.0	8 7.9	4 4.0	4.0

egg, using soybean oil, for frying it, and (d) promoting the consumption of chicken, fish and soybean as the sources of protein. Each subject also received a brochure describing hypercholesterolemia and its dietary management written by Professor Vichai Tanphaichitr.

All of these 110 RHS returned again for their serum lipid determination. The intervals between third and second visits ranged from 7 to 23 wks (mean $\pm$ SD =12 $\pm$ 5 wks). Table 84 shows that at the second visit only 1 MRHS with type IIa hyperlipoproteinemia, aged 34 yrs, became normolipidemia.

Table 85 shows that at the third visit 2 additional MRHS and 13 FRHS became normolipidemia. Thus the responding rate to dietary intervention from the second visit in MRHS and FRHS were 25 and 13%, respectively.

Tables 86 and 87 shows that effects of dietary treatment in 110 RHS by their serum TC and TG categories in each sex and age groups, respectively.

Table 88 shows that in 9 dyslipidemic MRHS, there was no significant changes in their serum lipid levels and lipid ratios between the first and second visits whereas their serum TC and LDL-C levels at the third visit were significantly lower than those at the first visit. In 101 dyslipidemic FRHS, their serum TC/HDL-C and LDL-C/HDL-C ratios at the second visit were significantly higher than

Table 84. Prevalences of dyslipidemic types in 110 RHS on the second visit by sex and age

Sex	Age	N	Normal	TC>5.2 mmol/L only	TC>5.2, LDL-C>3.4 & TG<2.3 mmol/L	TG>2.3 mmol/L only	TC>5.2, LDL-C>3.4 & TG>2.3 mmol/L	HDL-C<0.9 mmol/L only	
yr		N	%	N	%	N	%	N	%
M	20-29	3	-	-	2 66.7	-	1 33.3	-	-
	30-39	3	33.3	-	1 33.3	-	1 33.3	-	-
	40-49	2	-	-	1 50.0	-	1 50.0	-	-
	50-60	1	-	-	1 100.0	-	-	-	-
	Total	9	11.1	-	5 55.6	-	3 33.3	-	-
F	19-29	14	-	1 7.1	13 92.9	-	-	-	-
	30-39	32	-	2 6.3	25 78.1	-	1 3.1	4 12.5	-
	40-49	35	-	4 11.4	24 68.6	-	6 17.1	1 2.9	-
	50-61	20	-	1 5.0	13 65.0	-	5 25.0	1 5.0	-
	Total	101	-	8 7.9	75 74.3	-	12 11.9	6 5.9	-

Table 85. Prevalences of dyslipidemic types in 110 RHS on the third visit by sex and age

Sex	Age	N	Normal	TC>5.2 mmol/L only	TC>5.2, LDL-C>3.4 & TG<2.3 mmol/L	TG>2.3 mmol/L only	TC>5.2, LDL-C>3.4 & TG>2.3 mmol/L	HDL-C<0.9 mmol/L only
	yr		N	%	N	%	N	%
M	20-29	3	2	66.7	-	-	-	-
	30-39	3	1	33.3	-	-	-	-
	40-49	2	-	-	-	-	-	-
	50-60	1	-	-	-	-	-	-
	Total	9	3	33.3	-	-	-	-
F	19-29	16	5	35.7	1	7.1	-	-
	30-39	32	5	15.6	1	3.1	-	-
	40-49	35	3	8.6	2	5.7	8	22.9
	50-61	20	-	-	2	10.0	1	5.0
	Total	101	13	12.9	6	5.9	10	9.9
							2	2.0

Table 86. Number and percentage of 110 dyslipidemic RBS on dietary treatment by serum total cholesterol levels in each sex and age groups

Sex	Age	N	TC<5.2 mmol/L						TC=5.2-6.2 mmol/L						TC>6.2 mmol/L					
			1st		2nd		3rd		1st		2nd		3rd		1st		2nd		3rd	
yr			n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
M	20-29	3	-	-	-	-	2	66.7	1	33.3	2	66.7	-	-	2	66.7	1	33.3	1	33.3
	30-39	3	-	-	1	33.3	1	33.3	-	-	1	33.3	-	-	3	100.0	1	33.3	2	66.7
	40-49	2	-	-	-	-	1	50.0	-	-	2	100.0	-	-	2	100.0	-	-	1	50.0
	50-60	1	-	-	-	-	-	-	-	-	-	-	1	10.0	1	100.0	1	10.0	-	-
	Total	9	-	-	1	11.1	4	44.4	1	11.1	5	55.6	1	11.1	8	88.9	3	33.3	4	44.4
F	19-29	14	1	7.1	-	-	5	35.7	9	64.3	9	64.3	5	35.7	4	28.6	5	35.7	4	28.6
	30-39	32	1	3.1	1	3.1	6	18.8	18	56.3	13	40.6	13	40.6	13	40.6	18	56.3	13	40.6
	40-49	35	-	-	-	-	3	8.6	19	54.3	17	48.6	19	54.3	16	45.7	18	51.4	13	37.1
	50-61	20	2	10.0	-	-	-	-	6	30.0	5	25.0	7	35.0	12	60.0	15	75.0	13	65.0
	Total	101	4	4.0	1	1.0	14	13.6	52	51.5	44	43.6	44	43.6	45	44.6	56	55.4	43	42.6

Table 87. Number and percentage of 110 dyslipidemic RBS on dietary treatment by serum triglyceride levels in each sex and age group

Sex	Age	N	TG<2.26 mmol/L						TG=2.26-4.51 mmol/L						TG>4.52-11.29 mmol/L					
			1st	2nd	3rd	1st	2nd	3rd	1st	2nd	3rd	1st	2nd	3rd	1st	2nd	3rd	1st	2nd	3rd
yr	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
M 20-29	3	2	66.7	2	66.7	3	100.0		1	33.3	1	33.3	-	-	-	-	-	-	-	-
30-39	3	2	66.7	2	66.7	3	100.0		1	33.3	1	33.3	-	-	-	-	-	-	-	-
40-49	2	1	50.0	1	50.0	1	50.0		-	-	1	50.0	1	50.0	1	50.0	-	-	-	-
50-60	1	1	100.0	1	100.0	1	100.0		-	-	-	-	-	-	-	-	-	-	-	-
Total	9	6	66.7	6	66.7	8	88.9		2	22.2	3	33.3	1	11.1	1	11.1	-	-	-	-
F 19-29	14	14	100.0	14	100.0	14	100.0		-	-	-	-	-	-	-	-	-	-	-	-
30-39	32	28	87.5	29	90.6	30	93.8		3	9.4	3	9.4	2	6.3	1	3.1	-	-	-	-
40-49	35	28	80.0	28	80.0	26	74.3		7	20.0	7	20.0	8	22.9	-	-	-	-	4	2.9
50-61	20	16	80.0	15	75.0	19	95.0		4	20.0	5	25.0	1	5.0	-	-	-	-	-	-
Total	101	86	85.1	86	85.1	89	88.1		14	13.9	15	14.9	11	10.9	1	1.0	-	-	-	-

Table 88. Means±SEM of serum lipid levels and TC/HDL-C and LDL-C/HDL-C ratios in 110 dyslipidemic RHS on dietary treatment

Sex	N	Visit	TC	LDL-C	HDL-C	TG	Phospholipid	TC		LDL-C	
								HDL-C	TC	HDL-C	LDL-C
(<----- mmol/L ----->)											
M	9	1 st	6.6±0.1	4.5±0.3	1.3±0.1	1.8±0.4	3.3±0.3	5.2±0.3		3.5±0.3	
		2 nd	6.0±0.2	3.9±0.2	1.3±0.1	1.8±0.3	3.0±0.2	4.8±0.3		3.1±0.2	
		3 rd	5.9±0.3	3.8±0.3	1.3±0.1	1.6±0.3	3.2±0.1	4.5±0.3		3.0±0.2	
F	101	1 st	6.3±0.1	4.1±0.1	1.6±0.1	1.5±0.1	3.0±0.1	4.4±0.1	a2	2.8±0.1	a2
		2 nd	6.3±0.1	4.1±0.1	1.5±0.03	1.5±0.1	2.9±0.1	4.6±0.1		3.1±0.1	
		3 rd	6.1±0.1	4.0±0.1	1.5±0.03	1.5±0.1	2.9±0.1	4.5±0.1		2.9±0.1	
M&F	110	1 st	6.4±0.1	4.1±0.1	1.6±0.1	1.6±0.1	2.5±0.1	4.5±0.1		3.0±0.1	
		2 nd	6.2±0.1	4.2±0.1	1.4±0.03	1.6±0.1	2.9±0.1	4.6±0.1		3.1±0.1	
		3 rd	6.0±0.1	4.0±0.1	1.4±0.03	1.5±0.1	3.0±0.1	4.4±0.1		2.9±0.1	

Significant difference from the 1 st visit of each group: p<0.005, p<0.05.
Significant difference from the 2 nd visit of each group: p<0.01, p<0.02, p<0.05.

those at the first visit whereas their serum TC and LDL-C levels at the third visit were significantly lower than those at the first and second visits; and in all 110 dyslipidemic RHS, their serum PL levels at the second and third visits were significantly higher than that at the first visit, their serum TC level at the third visit was significantly lower than those at the first and second visits, and their serum LDL-C level, TC/HDL-C and LDL-C/HDL-C ratios were significantly lower than those at the second visit.

Table 89 shows that plasma S-particle levels in 9 MRHS at the second and the third visits were significantly lower than that at the first visit; plasma fibrinogen level in 101 dyslipidemic FRHS at the first visit was significantly lower than those at the second and third visits and their plasma M-particle level at the third visit was significantly lower than those at the first and the second visits and 110 dyslipidemic RHS had significantly lower plasma M-particle level at the first visit than that at the third visit and lower plasma fibrinogen level at the first visit than those at the second and third visits.

Table 90 shows that in 101 dyslipidemic FRHS and 110 dyslipidemic RHS, their serum apo A-I levels at the second and third visits were significantly lower than those at the first visit.

Table 89. Means±SEM of serum lipoprotein and plasma fibrinogen levels in 110 dyslipidemic RHS on dietary treatment

Sex	N	Visit	L-particle	M-particle	S-particle	Fibrinogen
<----- mg/dL ----->						
M	9	1 st	9.5±3.8	201.4±36.6	491.1±14.4	250.1±24.6
		2 nd	10.1±3.2	189.8±31.7	440.2±26.5 ^{a5}	229.6±15.1
		3 rd	10.9±4.7	197.1±41.0	434.6±26.8 ^{a5}	265.2±18.0
F	101	1 st	6.2±0.8	196.3±10.6	461.3±6.9	278.6±7.1
		2 nd	6.4±0.5	178.2±8.4 ^{a2,b2}	459.6±7.0	304.2±8.8 ^{a1,b1}
		3 rd	5.6±0.4	161.5±9.2	454.0±6.1	327.6±7.2
M&F	110	1 st	6.4±0.8	196.7±10.1	463.8±6.6	276.3±6.8
		2 nd	6.7±0.6	179.2±8.2 ^{a2}	458.0±6.6	298.8±8.5 ^{a1,b1}
		3 rd	6.0±0.6	164.4±9.1	452.4±6.0	322.5±7.0

Significant difference from the 1 st visit of each group: ^{a1} p<0.0005,
^{a2} ^{a3} ^{a4} ^{a5}
^{a2} p<0.002, ^{a3} p<0.01, ^{a4} p<0.02, ^{a5} p<0.05.

Significant difference from the 2 nd visit of each group: ^{b1} p<0.02,
^{b2} p<0.05.

Table 90. Means $\pm$ SEM of serum apoprotein levels in 110 dyslipidemic EHS on dietary treatment

Sex	N	Visit	Apoprotein A-I		Apoprotein B	
			<----- mg/dL ----->			
M	9	1 st	154.6 $\pm$ 11.1		142.20 $\pm$ 9.5	
		2 nd	142.7 $\pm$ 12.04		139.41 $\pm$ 6.0	
		3 rd	132.3 $\pm$ 10.8		130.77 $\pm$ 7.3	
F	101	1 st	156.5 $\pm$ 3.3		131.4 $\pm$ 2.7	
		2 nd	143.3 $\pm$ 3.3 ^{a1}		135.6 $\pm$ 3.31	
		3 rd	142.1 $\pm$ 3.6 ^{a3}		134.9 $\pm$ 3.3	
M&F	110	1 st	156.3 $\pm$ 3.2		132.2 $\pm$ 2.6	
		2 nd	143.3 $\pm$ 3.2 ^{a1}		135.9 $\pm$ 3.1	
		3 rd	141.3 $\pm$ 3.4 ^{a2}		134.6 $\pm$ 3.1	

Significant difference from the 1 st visit of each group:

a1 a2 a3
p<0.0005, p<0.001, p<0.002.

Relative FA composition of total serum and erythrocyte lipids

Table 91 shows that serum 14:0 level and 20:3 n-9/20:4 n-6 ratio in 110 dyslipidemic RHS were significantly lower than those at the first and second visits; their serum 18:0 level at the third visit was significantly lower than that at the second visits, their serum 18:1 n-9 levels at the first visit was significantly higher than those at the second and third visits whereas opposite results were observed for serum 22:5 n-3 and 22:6 n-3; and their serum 20:3 n-6 and 22:6 n-3 levels at the third visit were significantly higher than those at the second visit whereas their serum 20:4 n-6 level at the third visit was significantly higher than that at the first visit.

Table 92 shows that erythrocyte 16:0, 18:1 n-9, and 20:3 n-6 levels in 110 dyslipidemic RHS at the third visit were significantly higher but their erythrocyte 20:5 n-3 level at the third visit was significantly lower than those at the second visit.

Table 93 shows that when 110 dyslipidemic RHS at the first visit were categorized into 16 normolipidemic and 94 dyslipidemic RHS at the third visit, their relative FA compositions of total serum lipids were not significantly different.

Table 91. Relative FA composition of total serum lipids in 110 dyslipidemic RHS on dietary treatment

Fatty acid	Mean±SEM		
	1 st	2 nd	3 rd
<———— % of total fatty acids ———>			
NEFA			
			a3,b3
14:0	0.8±0.02	0.8±0.02	0.7±0.02
16:0	28.2±0.3	28.2±0.2	27.9±0.3
16:1 n-7	1.3±0.04	1.2±0.03	1.2±0.04
			b2
18:0	11.9±0.1	12.2±0.1	11.8±0.1
20:0	0.5±0.02	0.5±0.02	0.5±0.02
		a3	a3
18:1 n-9	12.1±0.2	11.5±0.1	11.6±0.1
20:3 n-9	0.4±0.01	0.4±0.01	0.4±0.01
EFA			
18:2 n-6	19.2±0.3	19.0±0.3	19.3±0.3
			b2
20:3 n-6	2.2±0.1	2.1±0.1	2.3±0.1
			a3
20:4 n-6	8.7±0.1	8.9±0.1	9.2±0.1
18:3 n-3	1.7±0.03	1.8±0.03	1.8±0.03
20:5 n-3	0.6±0.02	0.7±0.02	0.6±0.02
		a2	a2
22:5 n-3	0.7±0.02	0.8±0.02	0.8±0.02
		a1	a1,b3
22:6 n-3	6.2±0.1	7.0±0.1	7.2±0.2
			a3,b3
20:3 n-9	0.05±0.001	0.05±0.001	0.04±0.001
20:4 n-6			

Significant difference from the 1 st visit: p<0.0005

a2 a3
p<0.005, p<0.01.

Significant difference from the 2 nd visit: p<0.0005

b2 b3
p<0.005, p<0.01.

Table 92. Relative FA composition of total erythrocyte lipids in 110 dyslipidemic RHS on dietary treatment

Fatty acid	Mean±SEM	
	2 nd	3 rd
<— % of total fatty acids—>		
NEFA		
14:0	0.5±0.01	0.5±0.01
16:0	22.4±0.2	23.1±0.1 ^{a3}
16:1 n-7	0.2±0.03	0.2±0.02
18:0	16.6±0.1	16.8±0.1
20:0	0.5±0.03	0.5±0.02 ^{a1}
18:1 n-9	11.8±0.1	12.1±0.1
20:3 n-9	0.4±0.01	0.4±0.01
EFA		
18:2 n-6	8.3±0.1	8.4±0.01 ^{a2}
20:3 n-6	1.5±0.03	1.7±0.04
20:4 n-6	14.0±0.2	14.1±0.1
18:3 n-3	1.6±0.03	1.6±0.03 ^{a2}
20:5 n-3	3.8±0.1	3.6±0.1
22:5 n-3	1.9±0.03	1.9±0.03
22:6 n-3	8.7±0.1	8.6±0.1
20:3 n-9	0.03±0.001	0.03±0.001
20:4 n-6		

Significant difference from the 2 nd visit: ^{a1} p<0.0005,
^{a2} p<0.005, ^{a3} p<0.05.

Table 93. Relative FA composition of total serum lipids in 110 dyslipidemic RHS at the third visit by dyslipidemic criteria

Fatty acid	Mean±SEM	
	Normolipidemia n=16	Dyslipidemia n=94
<— % of total fatty acids —>		
NEFA		
14:0	0.7±0.03	0.7±0.02
16:0	26.6±1.7	28.1±0.2
16:1 n-7	1.1±0.1	1.2±0.04
18:0	11.7±0.3	11.8±0.1
20:0	0.5±0.03	0.5±0.02
18:1 n-9	11.8±0.3	11.5±0.1
20:3 n-9	0.4±0.02	0.4±0.01
EFA		
18:2 n-6	20.1±0.6	19.1±0.3
20:3 n-6	2.0±0.1	2.4±0.1
20:4 n-6	9.3±0.3	9.2±0.2
18:3 n-3	1.7±0.1	1.8±0.03
20:5 n-3	0.6±0.04	0.6±0.02
22:5 n-3	0.8±0.1	0.8±0.02
22:6 n-3	6.9±0.4	7.3±0.2
20:3 n-9	0.04±0.003	0.04±0.001
20:4 n-6		

Table 94 shows that erythrocyte 18:0 level in 94 dyslipidemic RHS at the third visit was significantly lower than that in 16 normolipidemic RHS at the third visit whereas opposite result was observed for erythrocyte 18:1 n-9 level.

Table 95 shows that serum 18:1 n-9 level in 6 dyslipidemic MRHS at the third visit was significantly lower than that in 3 normolipidemic MRHS at the third visit.

Table 96 shows that there were no significant difference in relative FA composition of total erythrocyte lipids between normolipidemic and dyslipidemic MRHS.

Table 97 shows that serum 20:3 n-6 level in 88 dyslipidemic FRHS at the third visit was significantly higher than that in 13 normolipidemic FRHS at the third visit whereas Table 98 shows the same result for erythrocyte 18:1 n-9 level between the two groups.

Univariate analysis

Tables 99-114 show relationships between different independent variables and various dependent variables. These data were utilized for supporting the discussion.

Table 94. Relative FA composition of total erythrocyte lipids in 110 dyslipidemic RHS at the third visit by dyslipidemic criteria

Fatty acid	Mean±SEM	
	Normolipidemia n=16	Dyslipidemia n=94
<— % of total fatty acids —>		
NRFA		
14:0	0.45±0.03	0.5±0.01
16:0	23.3±0.4	23.1±0.1
16:1 n-7	0.2±0.04	0.2±0.02
		a2
18:0	17.5±0.3	16.7±0.1
20:0	0.5±0.1	0.5±0.01
		a1
18:1 n-9	11.4±0.3	12.2±0.1
20:3 n-9	0.4±0.03	0.4±0.01
RFA		
18:2 n-6	8.3±0.3	8.4±0.1
20:3 n-6	1.6±0.1	1.7±0.04
20:4 n-6	14.7±0.4	14.0±0.1
18:3 n-3	1.6±0.1	1.6±0.03
20:5 n-3	3.7±0.1	3.6±0.5
22:5 n-3	1.8±0.1	1.8±0.03
22:6 n-3	8.1±0.3	8.7±0.1
20:3 n-9	0.03±0.003	0.03±0.001
20:4 n-6		

Significant difference from normolipidemia: ^{a1} p<0.02, ^{a2} p<0.05.

Table 95. Relative FA composition of total serum lipids
in 9 MRHS at the third visit by dyslipidemic criteria

Fatty acid	Mean±SEM	
	Normolipidemia n=3	Dyslipidemia n=6
<— % of total fatty acids —>		
NRFA		
14:0	0.6±0.1	0.8±0.01
16:0	29.4±1.0	29.9±0.6
16:1 n-7	1.4±0.2	1.3±0.2
18:0	11.1±0.5	11.6±0.3
20:0	0.5±0.1	0.5±0.1
18:1 n-9	12.8±0.3	11.8±0.6
20:3 n-9	0.4±0.03	0.5±0.05
RFA		
18:2 n-6	19.03±1.4	19.0±1.0
20:3 n-6	2.1±0.5	2.6±0.2
20:4 n-6	8.7±0.2	9.1±0.6
18:3 n-3	1.7±0.1	1.7±0.7
20:5 n-3	0.5±0.4	0.6±0.04
22:5 n-3	0.8±0.5	0.8±0.1
22:6 n-3	5.7±0.2	6.6±0.1
20:3 n-9	0.04±0.005	0.05±0.005
20:4 n-6		

al
Significant difference from normolipidemia: p<0.05.

Table 96. Relative FA composition of total erythrocyte lipids in 9 male RHS at the third visit by dyslipidemic criteria

Fatty acid	Mean±SEM	
	Normolipidemia n=3	Dyslipidemia n=6
<---- % of total fatty acids ---->		
NEFA		
14:0	0.4±0.08	0.4±0.04
16:0	24.8±1.1	24.5±0.4
16:1 n-7	0.3±0.03	0.3±0.9
18:0	17.4±0.8	16.5±0.3
20:0	0.4±0.1	0.5±0.1
18:1 n-9	11.9±0.4	12.7±0.2
20:3 n-9	0.4±0.1	0.4±0.1
EFA		
18:2 n-6	8.4±0.6	8.2±0.8
20:3 n-6	1.6±0.5	1.6±0.1
20:4 n-6	14.3±0.5	13.4±0.4
18:3 n-3	1.7±0.4	1.5±0.1
20:5 n-3	3.3±0.4	3.8±0.2
22:5 n-3	1.6±0.2	2.0±0.1
22:6 n-3	8.1±0.5	9.0±0.6
20:3 n-9	0.03±0.01	0.03±0.004
20:4 n-6		

Table 97. Relative FA composition of total serum lipids in 101 FHHS at the third visit by dyslipidemic criteria

Fatty acid	Mean±SEM	
	Normolipidemia n=13	Dyslipidemia n=88
<— % of total fatty acids —>		
NRFA		
14:0	0.7±0.04	0.7±0.02
16:0	26.0±2.1	28.0±0.2
16:1 n-7	1.1±0.1	1.2±0.04
18:0	11.9±0.3	11.9±0.1
20:0	0.5±0.04	0.5±0.02
18:1 n-9	11.6±0.3	11.5±0.1
20:3 n-9	0.4±0.03	0.3±0.01
EFA		
18:2 n-6	20.4±0.7	19.4±0.3
20:3 n-6	1.9±0.1	2.3±0.1
20:4 n-6	9.4±0.4	9.2±0.2
18:3 n-3	1.7±0.1	1.8±0.03
20:5 n-3	0.6±0.04	0.7±0.03
22:5 n-3	0.8±0.1	0.8±0.02
22:6 n-3	7.2±0.5	7.4±0.2
20:3 n-9	0.04±0.004	0.04±0.001
20:4 n-6		

al

Significant difference from normolipidemia: p<0.01.

Table 98. Relative FA composition of total erythrocyte lipids in 101 F RBS at the third visit by dyslipidemic criteria

Fatty acid	Mean±SEM	
	Normolipidemia n=13	Dyslipidemia n=88
<--- % of total fatty acids --->		
NRFA		
14:0	0.5±0.02	0.5±0.01
16:0	23.0±3.1	23.1±0.2
16:1 n-7	0.2±0.04	0.2±0.02
18:0	17.5±0.4	16.7±0.1
20:0	0.6±0.1	0.4±0.02
18:1 n-9	11.3±0.3	12.2±0.1
20:3 n-9	0.4±0.03	0.4±0.02
RFA		
18:2 n-6	8.4±0.6	8.2±0.3
20:3 n-6	1.7±0.6	1.7±0.1
20:4 n-6	14.3±0.6	13.4±0.4
18:3 n-3	1.7±0.4	1.5±0.1
20:5 n-3	3.3±0.4	3.8±0.2
22:5 n-3	1.6±0.2	2.0±0.1
22:6 n-3	8.1±0.5	9.0±0.6
20:3 n-9	0.03±0.01	0.03±0.004
20:4 n-6		

al

Significant difference from normal: p<0.05.

Table 99. Relationship between serum TC, TG or HDL-C levels in mmol/L and other serum lipid levels, serum lipid ratios, serum apoprotein, plasma lipoprotein and fibrinogen levels in 519 RHS

x		Y=a+bx	r	df	t	p
TC	LDL-C (mmol/L)	= -1.45+0.89x	0.9389	517	62.039	<0.0005
TC	HDL-C (mmol/L)	= 1.17+0.05x	0.1221	517	2.798	<0.01
TC	Apo A-I (mg/dL)	=131.68+3.20x	0.1088	517	2.490	<0.02
TC	Apo B (mg/dL)	= 26.30+16.49x	0.5736	517	15.926	<0.0005
TG	HDL-C (mmol/L)	= 1.48-0.49x	-0.20275	517	-4.708	<0.0005
TG	Apo B (mg/dL)	=110.03+72.93x	0.4538	517	11.582	<0.0005
HDL-C	Phospholipid (mmol/L)	= 2.49+0.18x	0.1360	517	3.123	<0.002
HDL-C	Apo A-I (mg/dL)	=114.79+23.15x	0.3432	517	8.307	<0.0005
HDL-C	Apo B (mg/dL)	=127.07-9.39x	-0.1424	517	-3.271	<0.002
TC	L-particle (mg/dL)	= 2.73+0.50x	0.0925	517	2.113	<0.0005
TC	M-particle (mg/dL)	= 49.48+19.62x	0.2543	517	5.979	<0.0005
TC	S-particle (mg/dL)	= -9.08+75.39x	0.9630	517	81.288	<0.0005
TG	L-particle (mg/dL)	= 4.92+9.76x	0.3231	517	7.765	<0.0005
TG	M-particle (mg/dL)	=140.08+282.55x	0.6530	517	19.725	<0.0005
TG	S-particle (mg/dL)	=385.97+62.18x	0.1421	517	3.265	<0.0002
LDL-C	M-particle (mg/dL)	=115.90+11.37x	0.1402	517	3.221	<0.002
LDL-C	S-particle (mg/dL)	=139.40+76.31x	0.9279	517	56.606	<0.0005
LDL-C	Apo B (mg/dL)	= 59.89+16.36x	0.54171	517	14.654	<0.0005
HDL-C	L-particle (mg/dL)	= 7.33-1.34x	-0.1080	517	-2.472	<0.02
HDL-C	M-particle (mg/dL)	=208.31-37.85x	-0.2139	517	-4.981	<0.000
HDL-C	S-particle (mg/dL)	=343.95+30.73x	0.1712	517	3.952	<0.0002

Table 100. Relationship between age in yrs and serum lipid levels, serum lipid ratios, serum apoprotein, plasma lipoprotein and fibrinogen levels in 519 RRS

$Y=a+bx$			r	df	t	p
TC (mmol/L)	=	$4.30+0.03x$	0.2219	517	5.1750	<0.0005
LDL-C (mmol/L)	=	$2.59+0.02x$	0.1625	517	3.7460	<0.0005
TG (mmol/L)	=	$-0.15+5.25x$	0.2518	517	5.917	<0.0005
Phospholipid (mmol/L)	=	$2.05+0.02x$	0.2752	517	6.509	<0.0005
TC/HDL-C	=	$2.89+0.02x$	0.1898	517	4.3970	<0.0005
LDL-C/HDL-C	=	$1.75+0.01x$	0.1595	517	3.6740	<0.0005
M-particle (mg/dL)	=	$71.25+2.18x$	0.2425	517	5.684	<0.0005
S-particle (mg/dL)	=	$329.06+1.59x$	0.1746	517	4.0330	<0.0002
Fibrinogen (mg/dL)	=	$211.28+1.61x$	0.2157	517	5.024	<0.0005
Apo B (mg/dL)	=	$73.66+1.06x$	0.3159	517	7.5710	<0.0005

Table 101. Relationship between BMI in kg/m^2 and serum lipid levels, serum lipid ratios, serum apoprotein, plasma lipoprotein and fibrinogen levels in 519 BHS

	$Y=a+bx$	r	df	t	p
TC (mmol/L)	= $4.06+0.05x$	0.1786	517	4.1290	<0.0005
LDL-C (mmol/L)	= $2.23+0.05x$	0.1607	517	3.7030	<0.0005
HDL-C (mmol/L)	= $2.14-0.03x$	-0.2279	517	-5.3240	<0.0005
TG (mmol/L)	= $-0.42+0.02x$	0.3810	517	9.370	<0.0005
TC/HDL-C	= $1.12+0.12x$	0.3553	517	8.6420	<0.0005
LDL-C/HDL-C	= $0.50+0.08x$	0.3019	517	7.2010	<0.0005
Apo A-I (mg/dL)	= $179.59-1.35x$	-0.1548	517	-3.5620	<0.0005
Apo B (mg/dL)	= $43.75+3.02x$	0.3558	517	8.6560	<0.0005
L-particle (mg/dL)	= $0.49+0.21x$	0.1326	517	3.0420	<0.005
M-particle (mg/dL)	= $-15.66+7.34x$	0.3212	517	7.7100	<0.0005
S-particle (mg/dL)	= $325.09+2.77x$	0.1196	517	2.740	<0.01
Fibrinogen (mg/dL)	= $207.99+2.78x$	0.1461	517	3.3580	<0.0005

Table 102. Relationship between body fat in percent of body weight and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RMS

	$Y=a+bx$	r	df	t	p
TC (mmol/L)	= 4.17+0.03x	0.1292	517	2.9630	<0.005
LDL-C (mmol/L)	= 2.15+0.03x	0.1375	517	3.1560	<0.002
HDL-C (mmol/L)	= 2.30-0.02x	-0.2257	517	-5.267	<0.0005
TG (mmol/L)	= -0.18+6.32x	0.1849	517	4.277	<0.0005
TC/HDL-C	= 0.71+0.08x	0.3251	517	7.816	<0.0005
LDL-C/HDL-C	= 0.15+0.06x	0.2848	517	6.756	<0.0005
Apo A-I (mg/dL)	= 172.97-0.63x	-0.0968	517	-2.2110	<0.05
Apo B (mg/dL)	= 41.60+1.85x	0.2916	517	6.932	<0.0005
L-particle (mg/dL)	= -4.27+0.25x	0.2083	517	4.843	<0.0005
M-particle (mg/dL)	= -30.16+4.72x	0.2774	517	6.5640	<0.0005
Fibrinogen (mg/dL)	= 207.34+1.67x	0.1174	517	2.6870	<0.01

Table 103. Relationship between body fat in kg and serum lipid levels, serum lipid ratios, serum apoprotein levels, and plasma lipoprotein levels in 519 RHS

	$Y=a+bx$	r	df	t	p
HDL-C (mmol/L) =	$1.60-5.34x$	-0.1406	517	3.2280	<0.002
TG (mmol/L) =	$-0.20+0.01x$	0.3363	517	8.1190	<0.0005
Phospholipid (mmol/L) =	$2.87-4.46x$	-0.0865	517	-1.9740	<0.05
TC/HDL-C =	$3.35+0.02x$	0.1946	517	4.5120	<0.0005
LDL-C/HDL-C =	$2.05+0.01x$	0.1681	517	3.8760	<0.0002
Apo B (mg/dL) =	$99.57+0.54x$	0.2172	517	0.0600	<0.0005
L-particle (mg/dL) =	$4.01+0.05x$	0.1136	517	2.6010	<0.001
M-particle (mg/dL) =	$124.03+1.14x$	0.1701	517	3.9260	<0.0002

Table 104. Relationship between mid upper arm circumferences in cm and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RBS

	$Y=a+bx$	r	df	t	p
TC (mmol/L)	= 3.60+0.06x	0.1928	517	4.4690	<0.0005
LDL-C (mmol/L)	= 1.77+0.06x	0.1814	517	4.1950	<0.0005
HDL-C (mmol/L)	= 2.34-0.03x	-0.2333	517	-5.4560	<0.0005
TG (mmol/L)	= -0.53+0.02x	0.3682	517	9.0030	<0.0005
TC/HDL-C	= 0.29+0.13x	0.3632	517	8.8660	<0.0005
LDL-C/HDL-C	= -0.40+0.09x	0.3159	517	7.5730	<0.0005
Apo A-I (mg/dL)	= 187.06-1.44x	-1.504	517	3.4580	<0.001
Apo B (mg/dL)	= 21.12+3.45x	0.3690	517	9.028	<0.0005
L-particle (mg/dL)	= -151.00+0.26x	0.1464	517	3.3670	<0.001
M-particle (mg/dL)	= -59.41+7.94x	0.3167	517	7.5920	<0.0005
S-particle (mg/dL)	= 298.10+3.39x	0.1333	517	3.0580	<0.001
Fibrinogen (mg/dL)	= 186.42+3.19x	0.1530	517	3.5210	<0.001

Table 105. Relationship between mid upper arm muscle circumferences in cm and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RNS

$Y=a+bx$		r	df	t	p
TC (mmol/L)	= 3.95+0.06x	0.1576	517	3.6280	<0.0005
LDL-C (mmol/L)	= 1.97+0.06x	0.1621	517	3.7370	<0.0005
HDL-C (mmol/L)	= 2.19-0.04x	-0.1970	517	-4.5710	<0.0005
TG (mmol/L)	= -0.33+0.02x	0.2500	517	5.8720	<0.0005
TC/HDL-C	= 0.53+0.16x	0.3489	517	8.4670	<0.0005
LDL-C/HDL-C	= -2.85+0.12x	0.3084	517	7.3730	<0.0005
Apo A-I (mg/dL)	= 173.16-1.19x	-0.0989	517	-2.261	<0.5
Apo B (mg/dL)	= 40.15+3.54x	0.3017	517	7.1900	<0.0005
L-particle (mg/dL)	= -0.22+0.27x	0.1225	517	2.8080	<0.01
M-particle (mg/dL)	= -11.56+7.95x	0.32528	517	5.9410	<0.0005
S-particle (mg/dL)	= 313.86+3.62x	0.1134	517	2.5970	<0.01

Table 106. Relationship between triceps skinfold thicknesses in mm and serum lipid levels, serum lipid ratios, serum apoprotein levels, and plasma lipoprotein and fibrinogen levels in 519 RHS

$Y=a+bx$			r	df	t	p
TC (mmol/L)	=	$5.02+0.01x$	0.0934	517	2.1330	<0.05
HDL-C (mmol/L)	=	$1.58-6.45x$	-0.1034	517	-2.3640	<0.02
TG (mmol/L)	=	$-0.07+6.07x$	0.2372	517	5.5520	<0.0005
Phospholipid (mmol/L)	=	$2.56+0.01x$	0.1215	517	2.7840	<0.01
TC/HDL-C	=	$3.47+0.02x$	0.1165	517	2.6660	<0.01
LDL-C/HDL-C	=	$2.15+0.01x$	0.0953	517	2.1790	<0.05
Apo A-I (mg/dL)	=	$156.80-0.42x$	-0.1009	517	-2.306	<0.05
Apo B (mg/dL)	=	$99.21+0.73x$	0.1782	517	4.119	<0.0005
M-particle (mg/dL)	=	$118.82+1.78x$	0.1605	517	3.698	<0.0005
Fibrinogen (mg/dL)	=	$237.40+1.78x$	0.1940	517	4.498	<0.0005

Table 107. Relationship between waist or hip circumferences in cm and serum lipid levels, serum lipid ratios, and plasma lipoprotein levels in 519 RHS

x	Y=a+bx	r	df	t	p
Waist circumferences					
Phospholipid (mmol/L)	= 2.54+3.34x	0.1419	517	3.261	<0.002
TC/HDL-C	= 4.21-5.93x	-0.133	517	-3.057	<0.005
LDL-C/HDL-C	= 2.69-4.52x	-0.1234	517	-2.830	<0.005
L-particle (mg/dL)	= 6.71-0.02x	-0.09	517	-2.1930	<0.05
Hip circumferences					
TG (mmol/L)	= 0.15-1.33x	-0.1955	517	-4.534	<0.0005
TC/HDL-C	= 4.16-4.56x	-0.1228	517	-2.814	<0.01
LDL-C/HDL-C	= 2.62-3.15x	-0.1030	517	-2.3355	<0.02
Apo B (mg/dL)	= 122.63-0.12x	-0.1321	517	-3.030	<0.005
M-particle (mg/dL)	= 173.78-0.28x	0.1101	517	-2.5200	<0.05

Table 108. Relationship between WHR and serum lipid levels, serum lipid ratios, serum apoprotein, and plasma lipoprotein levels in 519 RHS

	$Y=a+bx$	r	df	t	p
HDL-C (mmol/L) =	$1.51-0.04x$	-0.1362	517	-3.1270	<0.002
TG (mmol/L) =	$-0.72+0.93x$	0.3496	517	8.485	<0.0005
Phospholipid (mmol/L) =	$2.81-0.04x$	-0.10	517	-2.39	<0.02
TC/HDL-C =	$3.58+0.18x$	0.2499	517	5.869	<0.0005
LDL-C/HDL-C =	$2.22+0.13x$	0.2124	517	4.9420	<0.0005
Apo B (mg/dL) =	$109.82+2.71x$	0.1449	517	3.332	<0.001
L-particle (mg/dL) =	$4.69+0.52x$	0.1478	517	3.3990	<0.001
M-particle (mg/dL) =	$143.52+7.27x$	0.1449	517	3.3310	<0.001

Table 109. Relationship between serum linoleate levels and serum lipid levels, serum lipid ratios, serum apoprotein, and plasma lipoprotein levels in 241 RRS

	$Y=a+bx$	r	df	t	p
TC (mmol/L)	= 7.14-0.08x	-0.2494	239	-3.9820	<0.0002
LDL-C (mmol/L)	= 4.88-0.07x	-0.2304	239	-3.6620	<0.0005
TG (mmol/L)	= 2.43-0.05x	-0.24891	239	-3.973	<0.002
TC/HDL-C	= 5.72-0.09x	-0.2591	239	-4.147	<0.0005
LDL-C/HDL-C	= 3.84-0.07x	-0.2417	239	-3.852	<0.0005
Apo B (mg/dL)	= 149.59-1.75x	-0.1866	239	-2.9370	<0.005
M-particle (mg/dL)	= 287.40-6.05x	-0.2251	239	-3.5720	<0.004
S-particle (mg/dL)	= 521.38-6.09x	-0.2314	239	-3.6780	<0.0005

*Consisting of 12 MRHS and 229 FRHS; out of 12 MRHS, 2 were normolipidemia and 10 dyslipidemia; out of 229 FRHS, 115 were normolipidemia and 126 were dyslipidemia

Table 110. Relationship between serum transferrin (TRA) levels in g/L or HBP in mg/dL and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RHS

x	Y=a+bx	r	df	t	p
TRA					
Apo A-I (mg/dL)	= 116.40+13.08x	0.15279	517	3.515	<0.0005
Apo B (mg/dL)	= 67.56+18.62x	0.22262	517	5.192	<0.0005
HBP					
TC (mmol/L)	= 4.72+0.01x	0.15100	517	3.473	<0.0005
LDL-C (mmol/L)	= 2.93+0.01x	0.09687	517	2.213	<0.05
TG (mmol/L)	= 0.73+0.01x	0.22034	517	5.136	<0.0005
PL (mmol/L)	= 2.39+0.01x	0.16647	517	3.839	<0.0001
TC/HDL-C	= 3.26+0.01x	0.13602	517	3.122	<0.002
LDL-C/HDL-C	= 2.05+0.01x	0.09966	517	2.277	<0.05
Apo B (mg/dL)	= 88.33+0.62x	0.23420	517	5.477	<0.0005
L-particle (mg/dL)	= 1.25+0.10x	0.20492	517	4.761	<0.0005
M-particle (mg/dL)	= 66.40+2.13x	0.30200	517	7.2030	<0.0005

Table III. Relationship between Hb levels in g/L and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 BHS

	$Y=a+bx$	r	df	t	p
TC (mmol/L)	= 3.60+0.13x	0.15342	517	3.530	<0.0005
LDL-C (mmol/L)	= 1.87+0.11x	0.13443	517	3.085	<0.005
TG (mmol/L)	= 0.19+0.08x	0.15505	517	3.569	<0.0005
TC/HDL-C	= 1.45+0.19x	0.19387	517	4.493	<0.0005
LDL-C/HDL-C	= 0.66+0.14x	0.17181	517	3.966	<0.0001
Apo B (mg/dL)	= 55.40+4.54x	0.18399	517	4.256	<0.0005
L-particle (mg/dL)	= -3.68+0.71x	0.15281	517	3.516	<0.0005
M-particle (mg/dL)	= -44.12+15.44x	0.23320	517	5.453	<0.0005
S-particle (mg/dL)	= 292.07+7.58x	0.11279	517	2.5810	<0.02
Fibrinogen (mg/dL)	= 199.27+5.65x	0.10140	517	2.318	<0.05

Table 112. Relationship between systolic blood pressure in mmHg and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RBS

	$Y=a+bx$	r	df	t	p
TC (mmol/L)	= 3.55+0.02x	0.18026	517	4.167	<0.0005
LDL-C (mmol/L)	= 1.97+0.01x	0.14203	517	3.262	<0.002
TG (mmol/L)	= -0.10+0.01x	0.22511	517	5.253	<0.0005
TC/HDL-C	= 1.53+0.02x	0.21395	517	4.980	<0.0005
LDL-C/HDL-C	= 0.77+0.01x	0.18328	517	4.239	<0.0005
Apo B (mg/dL)	= 31.09+0.76x	0.29788	517	7.095	<0.0005
M-particle (mg/dL)	= 0.76+1.40x	0.20544	517	4.773	<0.0005
S-particle (mg/dL)	= 271.32+1.08x	0.15624	517	3.5970	<0.0005
Fibrinogen (mg/dL)	= 205.69+0.61x	0.10536	517	2.409	<0.02

Table 113. Relationship between diastolic blood pressure in mmHg and serum lipid levels, serum lipid ratios, serum apoprotein levels, plasma lipoprotein and fibrinogen levels in 519 RHS

Y=a+bx			r	df	t	p
TC (mmol/L)	=	4.04+0.02x	0.16163	517	3.724	<0.0002
LDL-C (mmol/L)	=	2.29+0.01x	0.13464	517	3.089	<0.005
HDL-C (mmol/L)	=	1.75-4.07x	-0.08651	517	1.975	<0.05
TG (mmol/L)	=	0.02+0.02x	0.25757	517	6.061	<0.0005
TC/HDL-C	=	1.76+0.03x	0.24090	517	5.644	<0.0005
LDL-C/HDL-C	=	0.94+0.02x	0.20605	517	4.788	<0.0005
Apo B (mg/dL)	=	56.92+0.80x	0.25636	517	6.031	<0.0005
L-particle (mg/dL)	=	0.94+0.06x	0.10709	517	2.449	<0.02
M-particle (mg/dL)	=	19.74+1.87x	0.22561	517	5.266	<0.0005
S-particle (mg/dL)	=	308.67+1.13x	0.13370	517	3.0680	<0.005
Fibrinogen (mg/dL)	=	225.68+0.65x	0.09201	517	2.101	<0.05

Table 114. Relationship between FBG and serum lipid levels, lipid ratios, apoprotein levels in 519 RHS

	$Y=a+bx$	r	df	t	p
TG (mmol/L)	= 0.59+0.01x	0.13196	517	3.027	<0.005
TC/HDL-C	= 2.89+0.01x	0.10557	517	2.414	<0.02
LDL-C/HDL-C	= 1.74+0.01x	0.09008	517	2.057	<0.05
Apo B (mg/dL)	= 83.41+0.33x	0.13208	517	3.030	<0.005

CHAPTER IV

DISCUSSION

Population profile

Though all of 3,977 RHS in 1991 were invited to enroll in the study only 519 RHS (13.1%) participated the study. The participants consisted of 66 MRHS and 453 FRHS. Thus on the epidemiological point of view data of MRHS do not strongly reflect urban Thai men. Data analysis are also based on subdivision of both MRHS and FRHS into 4 age groups: 19-29, 30-39, 40-49 and 50-61 yrs. In 453 FRHS, the number of women 30-39 and 40-49 yrs old were nearly equal, ie, 154 (34.0%) and 158 (34.9%), respectively, followed by 97 women 19-29 yrs old (21.4%) and 44 women 50-61 yrs old (9.7%). It should be noted that only 4 females were 19 yrs old and 1 females was 61 yrs old. In 66 MRHS, the number of men in the younger age groups were higher than the older age groups, ie, 22 (33.3%) for 20-29 yrs old, 22 (33.3%) for 30-39 yrs old, 12 (18.2%) for 40-49 yrs old, and 10 (15.2%) for 50-60 yrs old. (Table 1). All of the participants resided in Bangkok Metropolitan area or its vicinity. Thus they can represent urban Thai population.

Serum lipid profile

It is now recognized that dyslipidemia, including elevated serum TC due to elevated serum LDL-C, elevated serum TG, and low serum HDL-C levels, is the high risk factor of CHD.^{28,29} Thus in clinical practice to obtain complete serum lipid profile, TC, HDL-C and TG must be determined whereas LDL-C can be calculated from Friedewald's formula whenever serum TG is less than 4.52 mmol/L.⁶² These were carried out in our study.

Cholesterol, TG, and PL are transported in plasma in the form of lipoproteins which contain specific apo proteins interacting with PL and free cholesterol to form the polar, outer shell of lipoprotein particles, encompassing a non-polar core containing TG and cholesterol esters. By means of preparative ultracentrifugation, 5 distinct classes of Lipoproteins can be isolated from postprandial plasma, ie, chylomicrons, VLDL, Intermediate density lipoprotein (IDL), LDL, and HDL. All of these lipoproteins contain mixture of apo, PL, free and esterified cholesterol, and TG, but in markedly different proportions.⁶⁹ Most of the cholesterol in the plasma of fasting normal subjects is carried by LDL, much smaller proportions being found in VLDL and HDL. In contrast, most of the endogenous TG is carried out by

VLDL. Chylomicrons transport dietary TG for a few hrs after each meal but are virtually absent from normal plasma after a 12 hr fast. Thus measurement of plasma or serum TC and TG gives the sum of the contributions from each lipoprotein class. Under normal circumstances the concentration of IDL in plasma is relatively low and is usually ignored but this can become a major determinant of both serum TC and TG of certain forms of dyslipidemia.⁶⁹ In normal subjects, serum TC is derived from chylomicrons <15%, VLDL 13%, LDL 70% and HDL 17% whereas the corresponding figures for serum TG are chylomicrons <5%, VLDL 55%, LDL 29%, and HDL 11%.⁶⁹ These explain why our results showing highly significant positive correlation between serum TC and serum LDL-C levels ($r=0.94$) and moderately significant positive correlate between serum TC levels and serum apo B levels ($r=0.57$), but low significant positive correlations between serum TC and serum HDL-C levels ($r=0.12$) and between serum TC and serum apo A-I levels ($r=0.11$). Wherever serum TG is isolated (Table 99). Serum HDL is decreased.⁶⁹ This is also observed in our study by significantly negative correlations between these 2 serum lipid levels ($r=-0.20$) and between serum HDL-C and serum apo B levels ($r=-0.14$). The significantly positive correlation between serum HDL-C and PL levels ($r=0.14$) (Table 99) is also consistent with

the lipid composition in HDL, ie, 38-43% and 41-42% of total lipids in HDL as TC and PL, respectively.⁶⁹

In our study, plasma lipoprotein types were also quantitated by micronephelometric method.⁶⁴ Plasma L-, M- and S-particles are equivalent to chylomicrons, VLDL and LDL, respectively. These are evident by the significantly positive correlations between serum TC or LDL-C and plasma S-particle levels ($r=0.96$ and 0.93) and between serum TG and plasma M- or L-particle levels ($r=0.65$ and 0.32). Plasma S-particle also included HDL-C evidenced by significantly positive correlation between serum HDL-C and plasma S-particle levels ($r=0.17$) but significantly negative correlation between serum HDL-C and plasma M-particle levels ($r=-0.21$) (Table 99).

Factors affecting serum lipid and lipoprotein levels

Both endogenous factors including age, gender, and inherited traits, as well as exogenous factors including body weight, dietary habits, and physical activity, can affect serum lipid levels.⁶⁹⁻⁷²

Apoproteins have three main functions. First, they help to solubilize cholesterol esters and TG by interacting with PL. Secondly, they regulate the reaction of these lipids with enzymes such as lecithin: cholesterol acyltransferase (LCAT), lipoprotein lipase, and hepatic

lipase. Thirdly, they bind to cell surface receptors and thus determine the sites of uptake and rates of degradation of other lipoprotein constituents, notably cholesterol.⁶⁹ Individual apoproteins used to be designated on the basis of their carboxy-terminal amino acids but this nomenclature has now been superseded by the alphabetical terminology introduced by Aldupovic.⁷³

Over 90% of the protein in HDL is apo A which is subdivided into apo A-I and apo A-II. The ratio of apo A-I: apo A-II is 3:1 in both HDL₂ and HDL₃. These two apoproteins have glutamine as their carboxy-terminal amino acid. The molecular weight of apo A-I is 28,300 and of apo A-II is 17,000. Apo A-II enhances the lipid binding properties of apo A-I and the latter also activates LACT.⁶⁹

Apo B is hetrogenous; apo B100 with a molecular weight of 512,000 is found mainly in chylomicrons, VLDL and LDL whereas apo B48 with a molecular weight of 241,000 is found only in chylomicrons. Apo B100 is the only apoprotein present in LDL and is a ligand for the LDL receptor but apo B48 is not.⁶⁹

Based on the aforementioned propertics of apo A-I and apo B, these two apoproteins were measured in our study, which demonstrated significantly positive correlations between serum TC and apo B levels ($r=0.57$) as well as between serum TG and apo B levels ($r=0.45$) (Table 99).

For most people, an increase in serum TC level is due to high LDL-C level caused by either or both of the following two mechanisms. First, a decrease in LDL-receptor activity leads to delayed clearance of both LDL and VLDL remnants which also results in that a greater conversion to LDL and further raises serum LDL levels. Secondly, an over production of VLDL particles by the liver which tends to raise serum lipoprotein levels along the cascade include VLDL, VLDL remnants, and LDL.⁷⁴

Several conditions including cigarette smoking, obesity, lack of exercise, androgenic and related steroids, beta-adrenergic blocking agents, and genetic factors, can cause a reduced level of serum HDL-C.^{69,74}

Elevated serum TG levels in the range of ≥ 2.26 - 5.64 mmol/L usually reflect an increase in serum VLDL only. When serum TG levels exceed 5.64 mmol/L, the serum usually contains chylomicrons in addition to VLDL and at concentrations above 11.29 mmol/L, chylomicrons typically predominates. Recent studies have also revealed that 5 common lipoprotein abnormalities accompany hypertriglyceridemia, ie, increased concentrations of chylomicron remnants, VLDL remnants and IDL, decreased serum HDL-C levels, and the presence of small dense LDL particles. These abnormalities apparently result from both metabolic alterations and physico-chemical changes secondary to

hypertriglyceridemia. All of them have been implicated in atherogenesis.^{69,74}

Age

Serum lipid concentrations and lipoprotein distribution at birth differ considerably from the patterns seen in later life. TC levels in cord blood averaged 1.65 mmol/L in two American studies, equally distributed between LDL and HDL but relatively little in VLDL evidenced by a mean serum TG of 0.41 mmol/L. The 95th percentile for serum LDL-C in cord blood in these studies was 1.05 mmol/L. A rapid rise in serum TC occurs during the 6 months of life but thereafter there is little change until puberty, serum TC and TG levels averaging 4 and 0.65 mmol/L, respectively, over this period. After the age of 15 there is a rise in serum TG and LDL-C in both sexes and a fall in serum HDL-C in boys but not in girls.⁷¹

During adult life serum TC and LDL-C levels continue to rise in both sexes with age. Mean serum LDL-C levels increase by approximately 0.34-0.45 mmol/L from 20 yrs to 60 yrs of age.⁷²

Our study has also shown that serum TC, LDL-C, TG, and PL levels increase with age evidenced by (a) significantly positive correlations between age and the

aforementioned serum lipid levels (Table 100), (b) significantly higher serum TC, LDL-C, TG, and PL levels in older age groups than younger age groups (Table 17), (c) the consistent findings on plasma M- and S- particle levels (Tables 18 and 100). Our study also agrees with our previous report in FGAT officials,⁴⁴ and (d) significantly positive correlation between age and serum apo B levels (Table 100), the apoprotein present in LDL and VLDL.

Gender

Females tend to have lower TG and VLDL-C levels and higher HDL-C levels than men from puberty onwards. Their TC and LDL-C levels are lower until the menopause but there after because higher than men of similar age, except for women taking hormone replacement therapy.⁶⁹ Females of all ages who are on estrogens and/or progestogens tend to have higher TG but lower levels of LDL-C than those not on hormones.⁷⁰

In our study, total FRHS had significantly higher serum HDL-C and PL levels than total MRHS. Total FRHS had lower serum TC, LDL-C and TG levels as well as plasma L-, M- and S-particle levels than total MRHS but the differences did not reach statistical significance (Tables 17 and 18). However, total FRHS had significantly lower serum apo B levels than MRHS (Table 19).

Obesity

It is now recognized that obesity has the adverse effects on metabolism of VLDL, LDL and HDL.⁷⁴ First, the major influence of obesity on lipoprotein metabolism is to induce an increased hepatic secretion of apo B containing lipoproteins into the circulation. This is evident by the increased in the total number of VLDL particles secreted by the liver in obesity. The most important factor reinforcing hepatic overproduction of VLDL is an increased influx of energy substrate into the liver. This takes place both postprandially and during fasting when surplus free fatty acids are released into circulation by an enlarged body pool of adipose tissue. This event is accentuated in the presence of visceral obesity because free fatty acids are released directly into the portal circulation. Whether the overproduction of VLDL particles raises serum TG level or not depends on TG clearance from circulation by lipoprotein lipase activity. Second, because of the overproduction of apo B containing lipoproteins in obesity, the conversion of VLDL to LDL is increased, which in turn, raises serum LDL-C level. However, whether hypercholesterolemia is present or not in obesity depends on the LDL clearance from circulation by the activity of LDL receptor. However, if the activity of LDL receptor is inherently low, the coexistence of obesity

can induce a striking elevation of serum LDL levels. It should also be noted that the activity of LDL receptor is suppressed in those who having high intakes of saturated fatty acids and cholesterol. Another notion is the formation of small dense LDL-particles carrying enhanced atherogenicity in hypertriglyceridemic obese subjects. Third, decreased serum HDL-C level due to hypertriglyceridemia and increased catabolism of HDL constituents by an excess of adipose tissue.

In our study grade II underweight, grade I underweight, acceptable weight, grade I obesity, and grade II obesity are defined by BMI of 16.0-18.4, 18.5-19.9, 20.0-24.9, 25.0-29.9, and 30.0-39.9 kg/m² whereas abdominal obesity is defined by WHR >1.0 in men and WHR of >0.8 in women. Besides, we had also measured TST to represent subcutaneous fat and total body fat. The adverse effects of obesity on lipoprotein metabolism were also observed in our study evidenced by (a) significantly positive correlations between BMI, body fat in percent of body weight or MUAC with the following serum or plasma parameters; TC, LDL-C, TG, apo B, L-, M-, and S-particle levels as well as significantly negative correlations between BMI, body fat in percent of body weight or MUAC with serum HDL-C or apo A-I levels (Tables 101, 102, and 104), (b) significant positive correlations between WHR

and TG, apo B, L- and M-particle levels as well as significantly negative correlations between WHR and HDL-C or PL levels (Table 108), (c) higher serum TC, LDL-C, and TG, plasma M- and S-particle, and serum apo B levels but lower serum HDL-C and apo A-I levels in subjects with higher BMI (Tables 25, 26, and 27) and (d) subjects with abdominal obesity had significantly higher serum TG, PL, and apo B as well as plasma M-particle levels but lower serum HDL level than those without abdominal obesity (Tables 37, 38, and 39).

Inadequate somatic protein status

The influence of somatic protein status on lipoprotein metabolism was also observed in this study. When subjects were categorized in to 2 groups according to MUAC of $<90\%$ and $\geq 90\%$ std to designate inadequate and adequate somatic protein status. Both MRHS and FRHS with inadequate somatic protein status had significantly lower serum TC, LDL-C, TG and apo B, as well as plasma L-particle, M-particle and S-particle levels but significantly higher serum HDL-C and apo A-I (in FRHS only) levels than those adequate somatic protein status (Tables 28, 29, and 30). These findings were supported by the significant positive correlations between MUAC and the following serum or plasma parameter: TC, LDL-C, TG, apo B,

and L-, M- and S-particle levels as well as significantly negative correlation between MUAC and serum HDL-C levels (Table 105).

Inadequate visceral protein status

Since albumin, transferrin retinol-binding protein (RBP) and VLDL are mainly synthesized by liver it is interested in assessing the influence of visceral protein status on lipoprotein metabolism. The prevalences of low serum albumin levels of <35 g/L, low serum transferrin levels of <2 g/L, and low serum RBP levels of <35 mg/L, in 453 FRHS were 6.0, 9.3, and 38.6%, respectively, whereas the corresponding figures in 66 MRHS were 1.5, 6.1, and 7.6%. FRHS with low serum RBP levels had significantly lower serum TC, LDL-C, TG, PL, and apo B, as well as plasma L- and M-particle levels but significantly higher serum HDL-C level than FRHS with normal serum RBP levels. These findings were also supported by significantly positive correlations between serum RBP levels and the following serum or plasma parameters: TC, LDL-C, TG, PL, TC/HDL-C, LDL-C/HDL-C, apo B, L- and M-particle (Table 110). FRHS with low serum transferrin levels also had significantly lower serum TC, TG and apo B levels than FRHS with normal serum transferrin levels (Table 42). These findings were also supported by significantly

positive correlations between serum transferrin levels and apo A-I or apo B (Table 110). Based on such findings we speculate that inadequate amino acid supply to liver may be the common factor limiting for the biosynthesis of transferrin, RBP, and apo B.

Anemia

In 1971, Westerman⁷⁵ reported low serum TC levels in subjects with anemia of various etiology. Based on WHO criteria,⁷⁶ males with Hb of <130 g/L and females with Hb of <120 g/L are anemic. The prevalences of anemia in 66 MRHS and 440 FRHS were 9.1 and 25.9%, respectively. Anemic FRHS had significantly lower serum TC, HDL-C, TG, PL and apo B, as well plasma L-, M- and S-particle levels than non-anemic FRHS (Tables 40, 41, and 42). These findings were supported by the significantly positive correlations between Hb levels and the following serum or plasma parameters: TC, LDL-C, TG, TC/HDL-C, LDL-C/HDL-C, apo B, L-, M- and S-particle levels (Table 111).

Blood glucose status

The WHO criteria⁷⁷ are employed for the diagnosis of diabetes mellitus when FBG is >6.7 mmol/L and 2-hr PBG is >11.1 mmol/L, and impaired glucose tolerance (IGT) when FBG is <6.7 mmol/L and 2-hr PBG is between 7.8-11.1

mmol/L. The prevalences of IGT and diabetes mellitus in 66 MRHS were 1.5 and 1.5%, respectively, whereas the corresponding figures in 453 FRHS were 11.2 and 2.2%. FRHS with IGT had significantly higher serum TC, TG, PL, and apo B, as well as plasma M-particle levels than FRHS with euglycemia whereas FRHS with diabetes mellitus had significantly higher serum TG and apo B levels than FRHS with euglycemia (Tables 46, 47, and 48).

Reaven⁷⁸ delineated the key feature of syndrome X, more commonly referred to as the metabolic syndrome, to be hyperinsulinism and insulin resistance, central obesity, hypertonia, elevation of serum TG, lowering of HDL-C and IGT or non-insulin-dependent diabetes mellitus (NIDDM). The combination of these metabolic alterations has been recently reominated as insulin resistance syndrome (IRS), emphasizing the role of insulin resistance as the underlying mechanism.⁷⁹ Several of the metabolic abnormalities in this IRS are well recognized risk factors for coronary artery disease and has been identified as the deadly quartet.⁸⁰ More recently new members have been ascribed to the metabolic syndrome. The association between hyperuricemia and lipid abnormalities on the one hand and the association between PAI-1 and TG on the other has been long recognized but only recently linked to the constellation of the metabolic syndrome.⁸¹

When we combined 51 FRHS with IGT with 10 FRHS with DM together, it is evident that the latter group had significantly higher age, BMI, body fat in kg, waist circumference, WHR (Table 49), serum TC, TG, TC/HDL-C, LDL-C/HDL-C, apo B, plasma M-particle, and FRG than the former group; their systolic and diastolic BP were higher than euglycemic FRHS but the differences did not reach statistical significance (Table 50). The significantly positive correlation between FBG with serum TG level ($r=0.13$), TC/HDL-C ($r=0.10$), LDL-C/HDL-C ($r=0.09$) and serum apo B level ($r=0.13$). These findings indicate that our FRHS with IGT and DM had same features of the metabolic syndrome, especially the abdominal obesity sutical is considered as the important component of the syndrome.⁸²

Blood pressure

Most epidemiologic studies have shown a progressive increase in cardiovascular risk with increasing blood pressure. From middle age on, systolic pressure is a stronger predictor of CHD than diastolic pressure.²⁸ The prevalences of borderline hypertension (BP 140/90-160/95 mmHg) and definite hypertension (BP $\geq 160/95$ mmHg)⁸³ in 66 MRHS were 1.5 and 3.0%, respectively, whereas the corresponding figures in 453 FRHS were 0.2 and 2.0% (Table 51). The association

between blood pressure and serum lipids has been shown in Tromso study. This association is also observed in our study evidenced by (a) significantly higher serum TC, LDL-C, and plasma S-particle levels but significantly lower serum HDL-C and apo A-I levels in subjects with definite hypertension than normotensive subjects (Tables 51, 52 and 53) and (b) significantly positive correlations between systolic or diastolic blood pressure and the following serum and plasma parameters: TC, LDL-C, TG, apo B, M- and S-particles; significantly positive correlation between diastolic blood pressure and plasma L-particle levels but negative correlation between diastolic blood pressure and serum HDL-C levels (Tables 112 and 113).

Dietary factors

Energy and cholesterol intakes and dietary energy distribution High intakes of SFAs, dietary cholesterol, and total energy raise the serum TC levels. Cholesterol-lowering diets consist of 4 principles, ie, to reduce total fat intake to $\leq 30\%$ of total calories with equal distribution of SFAs, MUFAs, and PUFAs, Secondly; to reduce cholesterol intake to < 300 mg/d; to control obesity by decreasing total energy intake and increasing physical activity; and to have protein and carbohydrate intakes of 10-20 and 50-60% of total calories.^{11,28,29} Table 5 shows

that 66 MRHS had mean protein, fat and carbohydrate intakes of 11.3, 29.7 and 58.3% of total calories whereas the corresponding figures in 447 FRHS were 11.4, 31.7, and 56.8% of total calories. The mean total SFA, MUFA, and PUFA intakes in MRHS were 5.93, 7.68, and 7.57% of total calories, respectively whereas the corresponding figures in FRHS were 5.44, 7.04, and 8.36% of total calories. The mean cholesterol intakes in MRHS and FRHS were 337 and 197 mg/d, respectively (Tables 4, 5, 6 and 7). These dietary data were consistent with their serum lipid profiles being below or in the neighbourhood of the cut-off points for dyslipidemia, i.e., serum TC of ≥ 5.2 , LDL-C of ≥ 3.4 , HDL-C of < 0.9 and TG of ≥ 2.3 mmol/L. The mean serum TC, LDL-C, HDL-C and TG in 66 MRHS were 5.4, 3.4, 1.3 and 1.4 mmol/L, respectively, whereas the corresponding figures in 453 FRHS were 5.2, 3.2, 1.5 and 1.2 mmol/L (Table 17).

Linoleic acid

The decline in CHD mortality in Australians and Americans has been closely associated, temporally, with the dietary switch from animal fats to vegetable oils.⁸⁴ Epidemiological surveys between different populations also show an inverse relationship between the CHD mortality and linoleate consumption.⁸⁵ Tanphaichitr et al^{11,86} have shown the beneficial effects of linoleate intake on serum

TC and LDL-C levels. Eight healthy men were fed diets supplying a daily means($\pm$ SEM) energy of $3,055\pm176$ kcal and containing 15, 30, and 55% of total calories derived from protein, fat and carbohydrate, respectively, for 3 wk. The protein sources were pork, egg, and rice which supplied 7.6, 2.8, and 4.6% of total calories. The fat sources were soybean oil and food structural fat which supplied 20 and 10% of total calories. Their SFAs, 18:1 n-9, 18:2 n-6, 20:4 n-6, and 18:3 n-3 intakes were 5.6, 8.9, 11.3, 0.09, and 1.4% of total calories. Their mean ($\pm$ SEM) daily cholesterol intake was 925 ± 56 mg (303 mg/1,000 kcal). After taking soybean oil for 3 wk, there were significant increase in serum 18:2 n-6 level and significant decrease in serum 18:1 n-9 level with concomitant reduction in serum TC and LDL-C levels. The mean decrease in serum TC and LDL-C levels during taking soybean oil from the baseline values were 10.1 and 15.5%, respectively. In another study, 8 healthy men were fed diets containing 20% of total calories from soybean oil without cholesterol intake (vegetarian diet) and with a mean ($\pm$ SEM) daily cholesterol intake of 556 ± 20 mg (ovovegetarian diet), each diet for 10 days. The mean decreases in their serum TC and LDL-C levels during taking vegetarian diet from the baseline values were 22.8 and 28.9%, respectively, whereas those during taking

ovovegetarian diet were 20.0 and 25.5%. Their studies have shown that total fat and linoleate intakes of 30 and 10% of total calories can lower serum TC and LDL-C levels but this effect depends on the amount of cholesterol intake. The prevailing evidence suggests that the decrease in serum LDL-C induced by 18:2 n-6 is a result of modified secretion of cholesteryl ester and increased LDL receptor function.⁸⁷ Tanphaichitr et al⁸⁶ have also shown that there was no significant change in serum HDL-C level after consuming soybean oil for 3 wks. This is consistent with the current view that the lowering effect of linoleate-rich diets on serum HDL-C level appears to be a short term phenomenon, and occurs only with very large increase in 18:2 n-6 intake.⁸⁴

The influence of linoleate intake on serum lipid levels is also observed in our study. Relative FA composition of total serum lipids were determined in 12 out of 66 MRHS and 229 out of 453 FRHS. Only 2 out of 12 MRHS and 113 out of 229 FRHS were normolipidemia. Thus there were adequate number of normolipidemic and dyslipidemic subjects in FRHS for statistical comparison. Dyslipidemic FRHS had significantly higher serum TC, LDL-C, TG, PL, apo A-I, apo B, and plasma M- and S-particle levels than normolipidemic FRHS (Table 81). Dyslipidemic FRHS also had significantly higher age, BMI,

body fat in kg, and WHR than normolipidemic FRHS (Table 80). Besides dyslipidemic FRHS had significantly lower serum 18:2 n-6, 20:4 n-6 and 20:5 n-3 but higher 20:3 n-6 levels than normolipidemic FRHS (Table 82). These findings indicate that not only body composition but also linoleate intake determined their serum lipid levels. The role of linoleic acid in regulating lipoprotein metabolism in these FRHS is evidenced by significantly negative correlations between serum 18:2 n-6 levels and the following serum or plasma parameters: serum TC, LDL-C, TG, TC/HDL-C, LDL-C/HDL-C, apo B, M- and S-particle levels (Table 110).

Alcohol Regular consumption of alcohol leads to appreciable increase in HDL-C, an effect which independent of all other variables.⁶⁹ People drinking 2 units per day in the Cooperative Lipoprotein Phenotyping Study had HDL-C levels which were 0.33 mmol/L higher than non-drinkers.⁸⁸ The increase in HDL involves both HDL₂ and HDL₃ and may reflect an alcohol induced increase in lipoprotein lipase. Out of 66 MRHS, 44 were alcohol drinkers who had significantly higher serum HDL-C and apo A-I but lower apo b level than non-drinkers. Out of 401 FRHS, 46 were alcohol drinkers but they did not have any difference in serum lipoprotein profile from non-drinkers (Tables 70, 71, and 72). These findings were most likely due to the

difference in their drinking pattern. Out of 44 male alcohol drinkers, 10 drank alcohol daily and 34 drank alcohol occasionally whereas out of 46 female alcohol drinkers, 2 drank alcohol daily and 44 drank alcohol occasionally. However, the influence of sex on serum HDL-C, plasma M-particle, serum apo A-I and apo B levels in non-drinkers was abolished in alcohol drinkers.

Coffee A number of surveys have shown that ingestion of large quantities of coffee, especially boiled coffee, is associated with a rise in serum TC. This effect is not observed when equivalent quantities of tea are drunk, nor with instant coffee, and is therefore unlikely to be due to caffeine.⁶⁹

Out of 66 MRHS, 13 drank coffee daily and 33 drank coffee occasionally, whereas out of 447 FRHS, 151 drank coffee daily and 211 drank coffee occasionally. Most of them drank instant coffee. Only female coffee drinkers had significantly higher serum TG level than non-drinkers (Table 73). This may be rather related to the effect of sugar intake present in coffee consumed on TG synthesis than coffee itself.

Tobacco

Cigarette smoking has been suggested to increase the heart rate and increased heart rate has been shown to

increase atherogenic serum lipid fractions.⁸⁹ The prevalences of cigarette smokers in 66 MRHS and 447 FRHS were 65.2 and 2.0%, respectively. Male smokers had significantly higher serum TC, LDL-C, apo A-I, and plasma S-particle levels than male non-smokers (Tables 64, 65 and 66). When 43 male smokers were categorized into 3 groups according to the intensity of smoking, it was observed that 26 (60.5%) smoked <10 cigarettes/d, 13(30.2%) smoked 10-20 cigarettes/d, and 4(9.3%) smoked >20 cigarettes/d. The effects of smoking on lipoprotein metabolism were detected even in those who smoked <10 cigarettes/d (Tables 67, 68, and 69).

Physical activity

Aerobic exercise, such as jogging, when performed with sufficient intensity and frequency, results in appreciable reductions in fasting TG, TC and LDL-C, and marked rises in HDL-C. Such differences are even more evident in marathon runners than in joggers. The extent of these changes correlates with the degree of fitness achieved and is independent of other influences such as body weight and cigarette consumption. However, dietary factors, including alcohol, may play a role in determining the extent of the rise in HDL-C in runners.⁶⁹ The amount of exercise required to influence lipoprotein levels is

considerable, equivalent to running 10 miles/wk for 6-12 months.⁹⁰ Out of 66 MRHS, 9(13.6%) exercised daily and 38 (37.6%) exercised occasionally, whereas out of 447 FRHS, 30 (3.3%) exercised daily and 240 (53.7%) exercised occasionally. Forty-seven MRHS who exercise had significantly lower serum TC, LDL-C and plasma S-particle levels than 19 MRHS who did not exercised whereas 270 FRHS who exercised had significantly lower serum TC/HDL-C, LDL-C/HDL-C, and apo B than 177 FRHS who did not exercise (Tables 76, 77, and 78). These findings indicate the benefits of exercise on serum lipoprotein profile.

Levels of education and income

Since all of the subjects participating in this study were RHS their salaries were determined by their education levels. Out of 66 MRHS, 58(87.9%), 2(3.0%), and 6(9.1%) achieved school, vocational, and university levels whereas out of 447 FRHS, 221 (49.4%), 23(5.2%), and 203(45.4%) achieved school, vocation, and university levels. Those who earned >7,000 Baht/month were RHS achieving university level, those who earned ≤3,000-5,000 Baht/month were subjects achieving school or vocational levels, and those who earned 5,001-7,000 Baht/month were subjects achieving vocational or university levels (Tables 58, 59, 60, 61, 62, and 63). FRHS achieving university

level had significantly lower serum LDL-C, TC/HDL-C, and LDL-C/HDL-C but higher PL than FRHS achieving school level (Table 58). FRHS earning >7,000 Baht/month had significantly higher serum HDL-C but lower serum TC/HDL-C and LDL-C/HDL-C than FRHS earning 3,001-5,000 Baht/month (Table 61). This may imply that those who have high education level are more conscious to have a healthy life style including dietary factors and physical activity, which lead them to have an appropriate lipoprotein profile.

Corneal arcus

The presence of a corneal arcus before the age of 60 is often a sign of hypercholesterolemia. It reflects deposition of lipoprotein lipids within the cornea and is virtually irreversible.⁶⁹ Rosenman et al⁹¹ showed in their study involving over 3,000 men that the presence of corneal arcus was associated with a significantly increased risk of CHD even after adjusting for serum TC and smoking.

The prevalences of corneal arcus in 66 MRHS and 453 FRHS were 18.2 and 10.20%, respectively. Forty-six FRHS with corneal arcus had significantly higher serum TC, LDL-C, HDL-C, PL, apo B and plasma S-particle levels than 107 FRHS without corneal arcus (Tables 54, 55, and 56).

Since corneal arcus is one of the physical signs present in familial hypercholesterolemia.⁶⁹ This implies the high prevalence of heterozygous familial hypercholesterolemia in Thai population 56:500 in RHS. Their serum TC of ≥ 5.2 , LDL-C of ≥ 3.4 , and TG < 2.3 mmol/L (Table 54) indicate type IIa hyperlipoproteinemia which is consistent with familial hypercholesterolemia. However, other factors also influenced their serum lipid levels including age and BMI (Table 57).

Prevalences of dyslipidemia and lipoprotein phenotypes

Dyslipidemia including hypercholesterolemia due to elevated LDL-C, low HDL-C, and hypertriglyceridemia especially in the presence of low HDL-C is now recognized as the high risk factor of CHD. Two guidelines, European and U.S., have been currently publicized for the diagnosis of dyslipidemia.^{28,29} Table 115 summarizes the guidelines developed by these 2 groups of experts. In general, the differences between the 2 guidelines are minor. The U.S. guidelines were employed in this study. Table 116 shown the details of NCEP Expert Panel's criteria for dyslipidemia.²⁹

The mean serum TC, LDL-C, HDL-C and TG levels in 66 MRHS were 5.4, 3.4, 1.3 and 1.4 mmol/L, respectively, whereas the corresponding figures in 453 FRHS were 5.2,

Table 115. Guidelines for the diagnosis of dyslipidemia

Serum lipid	Cut-off limit	
	EAS [*]	NCEP ⁺
	<----- mmol/L ----->	
TC	≥5.2	≥5.2
LDL-C	≥3.5	≥3.4
HDL-C	<0.9 in men <1.1 in women	<0.9
TG	≥2.3	≥2.3

^{*}European Atherosclerosis Society²⁸.

⁺National Cholesterol. Education Program Expert panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults.²⁹

Table 116. NCEP Expert Panel's Criteria for dyslipidemia²⁹

Serum lipid	Risk		
	Desirable	Borderline-higher	High
TC, mmol/L mg/dL	<5.17 <200	5.17-6.19 200-239	≥6.20 ≥240
LDL-C	<3.36 <130	3.36-4.12 130-159	≥4.13 ≥160
HDL-C, mmol/L mg/dL	≥1.55 ≥60	0.90-1.54 35-59	<0.90 <35
TG mmol/L mg/dL	<2.26 <200	2.26-4.51 200-399	4.53-11.29 400-1000

*Negative risk factor

3.2, 1.5, and 1.2 mmol/L (Table 17). These findings indicate that serum lipid profile in MRHS was in the borderline-high risk TC and LDL-C whereas those in FRHS was in the desirable serum lipid levels.

However, when NCEP Expert Panel's criteria for dyslipidemia was applied in our subjects, it is evident that the major dyslipidemia in RHS was hypercholesterolemia due to elevated serum LDL-C levels. The prevalences of borderline-high risk and higher-risk serum TC in 66 MRHS were 6.1 and 27.3%, respectively, whereas the corresponding figures in 453 FRHS were 21.6 and 18.6% (Table 20). The prevalences of borderline-high risk and high risk serum LDL-C in 66 MRHS were 7.6 and 25.8%, respectively, whereas the corresponding figures in 453 FRHS were 15.0 and 19.9% (Table 21). However, 27.3% of 66 MRHS and 38.2% of 453 FRHS had serum HDL-C of ≥ 1.55 mmol/L (Table 22). The prevalences of borderline-high risk and high risk serum TG in 66 MRHS were 13.6 and 1.5% whereas the corresponding figures in 453 FRHS were 4.4 and 0.4%. None of the subjects had serum TG levels of ≥ 11.30 mmol/L (Table 23).

In this study, the lipoprotein phenotyping was defined by the World Health Organization classification of hyperlipoproteinemia⁶⁹ (Table 24). The lipoprotein phenotypes in both MRHS and FRHS ranked in the following

order: type IIa, IIb and IV hyperlipoproteinemia (Table 24).

Effects of dietary intervention on serum lipid status in dyslipoproteinemic subjects

The benefits of dietary intervention in 110 RHS were evidenced by (a) the significant lower serum TC and LDL-C levels as well as serum TC/HDL-C and LDL-C/HDL-C ratios at the third visit than those at the second visit⁷ (Tables 88 and 89); the net decreases of their serum TC and LDL-C levels at the third visit from the second visit were 3.22 and 4.76%, respectively, and (b) out of 109 dyslipidemic RHS at second visit, 15 became normolipidemic; the responding rate was 13.76%. However, there were no significant changes in serum 18:2 n-6 levels in 110 dyslipidemic RHS throughout the study (Table 91). This implies that the whole group did not increase the linoleate consumption. As a matter of fact, most of them admitted that they bought readily cooked foods back home for their supper and ate lunch at the hospital. Oils used for cooking these foods are usually lard, coconut oil or palmolein which are not rich in linoleic acids. It should be noted that mean serum 18:2 n-6 level in these 110 dyslipidemic RHS was lower than that in 113 normolipidemic FRHS (Tables 82 and 91). When these 110

dyslipidemic RHS was categorized into 16 normolipidemia and 94 dyslipidemic RHS according to their serum lipid levels at the third visit the mean serum linoleate level in 16 normolipidemic RHS was higher than that in 94 dyslipidemic RHS at the third visit but the difference did not reach statistical significance (Table 93). There was no significant difference in their erythrocyte linoleate levels neither (Table 94). These findings suggest that the changes in their serum TC and LDL-C levels may be due to other dietary factors, such as increase in lean meat intake instead of fatty meat which will lead to reduced intakes of cholesterol, total fat and SFAs.

Plasma fibrinogen

Since elevated plasma fibrinogen levels have consistently emerged as an independent risk factor for CHD, plasma fibrinogen was determined in this study.²⁸ Factors which increased plasma fibrinogen were age (Tables 18 and 100), BMI (Tables 26 and 101), body fat in percent of body weight (Table 102), MUAC (Tables 29 and 104), UAMC (Table 32), TST (Tables 35 and 106), WHR (Table 38), Hb levels (Tables 44 and 111), and systolic and diastolic blood pressure (Tables 112 and 113).

Mean plasma fibrinogen level in 113 normolipidemic FRHS was lower than that in 116 dyslipidemic FRHS but the

difference did not reach statistical significance (Table 81). It is interesting to note that during the follow-up study in 110 dyslipidemic RHS their plasma fibrinogen level at the second visit was significantly higher than that at the first visit and their plasma fibrinogen level at the third visit was also significantly higher than those at the first and second visits.

The mean plasma fibrinogen levels in 66 MRHS and 453 FRHS were 262.8 and 272.7 mg/dL. Base on $\text{mean} \pm 2 \text{ SD}$, the lower and upper normal limits of plasma fibrinogen levels in men and women were 125-401 and 132-413 mg/dL, respectively. Based on these limits, out of 66 MRHS, there were 3(4.5%) who had plasma fibrinogen below and above the out-off limits whereas the corresponding figures in 453 FRHS were 1(0.2%) and 23(5.1%).

CHAPTER V

SUMMARY

The purposes of the study are

1. To assess the influences of age, gender, body composition, visceral protein status, anemia, blood glucose status, blood pressure, dietary factors, smoking habit, levels of education and income and inherited traits on serum lipoprotein and plasma fibrinogen levels in RHS
2. to determine the prevalences of dyslipoproteinemia in RHS and
3. to assess the effects of dietary intervention on lipid status in RHS with dyslipoproteinemia.

The 591 participants consisted of 66 MRHS and 453 FRHS with the ages ranging from 19 to 61 yrs. The results are summarized below.

Factors influence serum lipid and lipoprotein levels

Age

Serum TC, LDL-C, TG and PL levels in RHS increased with age evidenced by significantly positive correlations between age and these serum lipid and apo B levels as well as significantly higher serum TC, LDL-C, TG, PL levels, plasma M- and S-particle levels in older age groups than younger age groups.

Gender

Total FRHS had significantly higher serum HDL-C and PL levels than total MRHS but total FRHS had lower serum TC, LDL-C, and TG levels as well as plasma L-, M- and S-particle levels than total MRHS. Total FRHS had significantly lower serum apo B levels than MRHS.

Obesity

There were significantly positive correlations between BMI, body fat in percent of body weight or MUAC with the following serum or plasma parameters: TC, LDL-C, TG, apo B, L-, M-, and S-particle levels but significantly negative correlations between the afores and anthropometric parameters and serum HDL-C and apo A-I levels.

Abdominal obesity is defined by WHR. There were significantly positive correlations between WHR and serum TG, apo B, plasma L- and M-particle levels but significantly negative correlations between WHR and HDL-C or PL levels.

Subjects with higher BMI had higher serum TC, LDL-C, TG, plasma M-, S-particle and serum apo B levels but lower serum HDL-C and apo A-I levels. Subjects with abdominal obesity had significantly higher serum TG, PL

and apo B as well as plasma M-particle levels but lower serum HDL-C level than those without abdominal obesity.

Inadequate somatic protein status

Both MRHS and FRHS with inadequate somatic protein status (MUAC <90% std) had significantly lower serum TC, LDL-C, TG and apo B, as well as plasma L-, M- and S-particle levels but significantly higher serum HDL-C and apo A-I levels than those adequate somatic protein status.

Inadequate visceral protein status

The prevalences of low serum albumin (<35 g/L), low serum transferrin (<2 g/L), and low serum RBP (<35 mg/dL) levels in 453 FRHS were 6.0, 9.3 and 38.6%, respectively, whereas the corresponding figures in 66 MRHS were 1.5, 6.1 and 7.6%. FRHS with low serum RBP levels had significantly lower serum TC, LDL-C, TG, PL and apo B as well as plasma L- and M-particle levels but significantly higher serum HDL-C level than FRHS with normal serum RBP levels. FRHS with low serum transferrin levels also had significantly lower serum TC, TG and apo B levels than FRHS with normal serum transferrin levels.

Anemia

The prevalences of anemia in 66 MRHS (Hb <130 g/L) and 440 FRHS (Hb <120 g/L) were 9.1 and 25.9%

respectively. Anemic FRHS had significantly lower serum TC, HDL-C, TG, PL and apo B, as well as plasma L-, M- and S-particle levels than non-anemic FRHS.

Blood glucose status

The prevalences of IGT and diabetes mellitus in 66 MRHS were 1.5 and 1.5% respectively, whereas the corresponding figure in 453 FRHS were 11.2 and 2.2%. FRHS with IGT had significantly higher serum TC, TG, PL and apo B, as well as plasma M-particle levels than FRHS with euglycemia whereas FRHS with DM had significantly higher serum TG and apo B levels than FRHS with euglycemia.

Blood pressure

The prevalences of borderline hypertension (BP $\geq 140/90$ - $< 160/95$ mmHg) and definite hypertension (BP $\geq 160/95$ mmHg) in 66 MRHS were 1.5 and 3.0% whereas the corresponding figures in 453 FRHS were 0.2 and 2.0%. Subjects with definite hypertension had significantly higher serum TC, LDL-C and plasma S-particle levels but significantly lower serum HDL-C and apo A-I levels than normotensive subjects.

Dietary factors

Energy and cholesterol intakes and dietary energy distribution Total MRHS had mean protein, fat and

carbohydrate intakes of 11.3, 29.7 and 58.3 of total calories whereas those in 447 FRHS were 11.4, 31.7 and 56.8% of total calories. The mean cholesterol intakes in MRHS and FRHS were 337 and 197 mg/d, respectively.

Linoleic acid Relative FA composition of total serum lipids were determined in 12 out of 66 MRHS and 229 out of 453 FRHS. Dyslipidemic FRHS had significantly higher serum TC, LDL-C, TG, PL, apo A-I, apo B and plasma M- and S-particle levels than normolipidemic FRHS. Besides dyslipidemic FRHS had significantly lower serum 18:2 n-6, 20:4 n-6 and 20:5 n-3 but higher 20:3 n-6 levels than normolipidemic FRHS. These findings indicate the linoleate role in regulating lipoprotein metabolism. There were significantly negative correlations between serum linoleate levels and serum TC, LDL-C, TG, apo B, plasma M- and S-particle levels.

Corneal arcus

The prevalences of corneal arcus in 66 MRHS and 453 FRHS were 18.2 and 10.20%, respectively. This suggests the high prevalence of heterozygous familial hypercholesterolemia in Thai population, ie, 56: 500 in RHS. Subjects with corneal arcus had significantly higher serum TC and LDL-C levels than those without corneal arcus.

Prevalences of dyslipidemia and lipoprotein phenotypes

The mean serum lipid profiles in MRHS were in the borderline-high risk TC and LDL-C whereas those in FRHS was in the desirable serum lipid levels. However, the prevalences of type IIa, IIb and IV hyperlipoproteinemias in 66 MRHS with were 22.7, 6.1, and 1.5% respectively, whereas the corresponding figures in 453 FRHS were 36, 2.6, and 0.9%.

Alcohol Out of 66 MRHS, 44 were alcohol drinkers. They had significantly higher serum HDL-C and apo A-I but lower apo B levels than 22 non-drinkers.

Coffee Only female coffee drinkers had significantly higher serum TG level than non drinkers.

Tobacco

Male smokers had significantly higher serum TC, LDL-C, apo A-I and plasma S-particle levels than male non-smokers.

Physical activity

Forty-seven MRHS who exercised had significantly lower serum TC, LDL-C and plasma S-particle levels than 19

MRHS who did not exercised whereas 270 FRHS who exercised had significantly lower serum TC/HDL-C and LDL-C/HDL-C ratios as well as serum apo B level than 177 FRHS who did not exercise.

Levels of education and income

RHS who had high education level had an appropriate serum lipoprotein profile. Their may be due to their awareness of having a healthy life style including good dietary habits and adequate physical activity.

Effects of dietary intervention on serum lipid status in dyslipoproteinemia subjects

The net decreases of 110 RHS in serum TC and LDL-C levels at the third visit from the second visit were 3.22 and 4.76% respectively. Out of 109 dyslipidemic RHS at the second visit, 15 became normolipidemia at the third visit; the responding rate was 13.76%. Though those becoming normolipidemia at the third visit had higher mean serum linoleate level than those with persistent dyslipidemia at the third visit, the difference did not reach statistical significance. Thus there might be other dietary factors lowering their serum TC and LDL-C levels.

Plasma fibrinogen

The normal limits of plasma fibrinogen levels based on their mean $\pm$ 2SD in 66 MRHS and 453 FRHS were 125-401 and 132-413 mg/dL, respectively. The prevalences of elevated plasma fibrinogen levels in 66 MRHS and 453 FRHS were 4.5 and 5.1%, respectively. Factors which increased plasma fibrinogen levels were age, BMI, body fat in percent of body weight, MUAC, UAMC, TST, WHR, Hb levels, and systolic and diastolic blood pressure.

REFERENCES

1. Alfin-Slater RB, Aftergood L. Fats, essential fatty acids and ascorbic acid. J Am Diet Assoc 1974; 64: 168-70.
2. Alfin-Slater RB, Kritchevsky D. Nutrition and the adult. New York: Plenum Press, 1980.
3. Howard RB, Herbold NH. Nutrition in clinical care. New York: McGraw Hill Book Company, 1978.
4. Tanphaichitr V. Dyslipidemia and CHD. Intern Med 1987; 3: 188.
5. Tanphaichitr V. Assessment of essential fatty acid status. In: Lin HJ, Swaminathan R. and Robershaw AM eds. Proceedings of the Fourth Asian-Pacific Congress of Clinical Biochemistry.
6. Pakpeankitvatana R, Tanphaichitr V. Essential fatty acid and lipid status in Thai adults. Intern Med. 1985; 1: 25-2.
7. Tanphaichitr V. Human alpha-linolenate deficiency. Intern Med 1989; 5: 147-8.
8. Guthrie HA. Introduction Nutrition. St Louis: The C.V. Mosby Company, 1979.
9. Brown MS, Goldstein JL. Familial hypercholesterolemia: a genetic defect in the LDL receptor. New Engl J Med 1976; 294: 1386-90.

10. WHO MONICA PROJECT. WHO MONICA project: Objectives and design. *Int J Epidemiol* 1989; 18: 3(Suppl 1):S29-S34.
11. Tanphaichitr V; Leelahagul P, Pakpeankitvatana R. Dietary prescription of protein and fat. In Kim WH, Lee YC, Lee KY, Ju JS, Kim SH, eds. *Proceedings of the 14th International Congress of Nutrition. Vol I.* Seoul: Ewha Womans University, 1989: 553-6.
12. Multiple Risk Factor Intervention Trial Research Group. Multiple risk factor intervention trial: risk factor changes and mortality results. *JAMA* 1982; 248: 1465-77.
13. WHO MONICA PROJECT. WHO MONICA project: risk factors. *Int J Epidemiol.* 1989; 18: 3 (Suppl 1): S46-S55.
14. D'Agostino RB, Kannel WB, Belanger A, et al. Trends in CHD and risk factors at age 55-64 in the Framingham Study. *Int J Epidemiol* 1989; 18: 3(Suppl 1): 67-S72.
15. Pearson TA. Coronary arteriography in the study of the epidemiology of coronary artery disease. *Epidemiol Rev* 1984; 6: 140-66.
16. Russell R. The pathogenesis of atherosclerosis-an update. *New Engl J Med* 1986; 20: 488-500.
17. Blumgart HL. Blood lipids and atherosclerosis. *Am J Med* 1951; 11: 271-3.

18. Swank RL, Grimsgaard A. Multiple sclerosis: the lipid relationship. *Am J Clin Nutr* 1988; 48: 1387-93.
19. Kannel WB. Update on the role of cigarette smoking in coronary artery disease. *Am Heart J* 1981; 101: 319-28.
20. Neaton JD, Wentworth D. Serum cholesterol, blood pressure, cigarette smoking, and death from CHD. *Arch Intern Med* 1992; 152: 56-64.
21. Bruke GL, Sprafka JM, Folsom AR, et al. Trends in CHD mortality, morbidity and risk factor levels from 1960 to 1986: The Minnesota Heart Survey. *Int J Epidemiol* 1989; 18: 3(Suppl 1): S73-S81.
22. Stehbens WE. The epidemiological relationship of hypercholesterolemia, hypertension, diabetes mellitus and obesity to CHD and atherogenesis. *J Clin Epidemiol* 1990; 43: 733-41.
23. Gofman JW, Lindgren F. The role of lipids and lipoproteins in atherosclerosis. *Science* 1950; 111: 166-86.
24. Zemel PC, Sowers JR. Relation between lipids and atherosclerosis: epidemiologic evidence and clinical implications. *Am J Cardiol*. 1990; 66: 71-121.

25. Freedman DS, Gruchow HW, Anderson J, et al. Relation of triglyceride levels to coronary artery disease: The Milwaukee Cardiovascular Data Registry. *Am J Epidemiol*. 1988; 127: 1118-30.
26. Austin MA. Plasma triglyceride as a risk factor for CHD. The epidemiological evidence and beyond. *Am J Epidemiol* 1989; 129: 249-59.
27. Grundy SM, Yegh GL. Two different views of the relationship of hypertriglyceridemia of CHD. *Arch Intern Med* 1992; 152: 28-34.
28. Recommendations of the European Atherosclerosis Society prepared by the International Task Force for Prevention of Coronary Heart Disease. Prevention of coronary heart disease: scientific background and new clinical guidelines. *Nutr Met Card Dis* 1992; 2: 113-56.
29. Expert Panel on Detection, Evaluation, and Treatment of High blood Cholesterol in Adults. Summary of the second report of the National Cholesterol Education Program (NCEP) Expert and on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel II). *JAMA* 1993; 269: 3015-23.

30. Fiebach NH, Hebert PR, Stampfer MJ, et al. A prospective study of high blood pressure and cardiovascular disease in women. *Am J Epidemiol* 1989; 130: 646-54.
31. Castelli WP. Categorical issues in therapy for CHD. *Cardiol Prac* 1985; 42: 743-50.
32. Seltzer CC. Framingham Study Data and "Established Wisdom" about cigarette smoking and CHD. *J Clin Epidemiol* 1989; 42: 743-50.
33. Kannel WB, D'Agostino RB, Belanger JA. Fibrinogen, cigarette smoking, and risk of cardiovascular disease: insights from the Framingham Study. *Am Heart J* 1987; 113: 1006-10.
34. Keys A, Fidanza F. Serum cholesterol and relative body weight of coronary patients in different populations. *Circulation* 1960; 22: 1091-106.
35. Osterman J, Lin T. Serum cholesterol profiles during treatment of obese outpatients with a very low calorie diet. Effect of initial cholesterol levels. *Int J Obes* 1992; 16: 49-58.
36. Kromhout D. Body weight, diet, and serum cholesterol in 871 middle-aged men during 10 years of follow-up (the Zutphen Study). *Am J Clin Nutr* 1983; 38: 591-8.

37. Kannel WB, McGee DL. Diabetes and cardiovascular disease. The Framingham Study. JAMA 1979; 241: 2035-8.
38. Reaven GM, Hoffman BB. A role for insulin in the aetiology and course of hypertension. Lancet. 1987; 22: 435-7.
39. Morris JN, Everitt MG. Vigorous exercise in leisure-time : protection against CHD. Lancet 1980; 2: 1207-10.
40. Report of a WHO Study Group. Diet, nutrition, and the prevention of chronic diseases. WHO Tech Rep Ser 1990; 797: 9-203.
41. Shaper AG. Alcohol and ischemic heart disease in middle aged British men. Brit Med J 1987; 294: 733-7.
42. Ginsberg H, Olefsky J, Farquhar JW, et al. Moderate ethanol ingestion and plasma triglyceride levels. A study in normal and hypertriglyceridemic persons. Ann Intern Med 1974; 80: 143-9.
43. Rissanen AM. Familial occurrence of CHD: effect of age at diagnosis. Am J Cardiol 1979; 44: 60-6.

44. Pakpeankitvatana R, Tanphaichitr V, Lochaya S, Viruenchavee N, Tamwiwat C, Watthana-Kasetr S. Clinical and epidemiological studies on hyperlipidemia in urban Thais. In: Yasumoto K, Itokawa Y, Koshi H, Sanno Y, eds. Proceedings of the Fifth Asian Congress of Nutrition. Tokyo: Center of Academic Publication Japan. 1988: 430-3.
45. Tanphaichitr V, Kulapongse S, Pakpeankitvatana R, Leelahagul P, Tamwiwat C, Lochaya S. Prevalence of Obesity and its associated risks in urban Thais. In: Oomura Y, Tarui S, Inoue S, Shimazu T, eds. Progress in Obesity Research 1990. London: John Libbey, 1990: 649-53.
46. Division of Nutrition, Department of health. Thai food composition table. Bangkok: Ministry of Public Health, 1984.
47. Food Composition table for use in East Asia. USA: U.S. Department of Health, Education and Welfare, 1972.
48. Science and Education Administration, United States Department of Agriculture. Agriculture handbook No. 8-1: composition of foods, dairy and egg products, raw, processed, prepared. Washington DC: US Government Printing Office, 1976.

49. Science and Education Administration, United States Department of Agriculture. Agriculture handbook No. 8-4: composition of foods, fat and oils, raw, processed, prepared. Washington DC: US Government Printing Office, 1979.
50. Science and Education Administration, United States Department of Agriculture. Agriculture handbook No. 8-10: composition of foods, pork products, raw, processed, prepared. Washington DC: US Government Printing Office, 1979.
51. Science and Education Administration, United States Department of Agriculture. Agriculture handbook No. 8-11: composition of foods, vegetables and vegetable products, raw, processed, prepared. Washington DC: US Government Printing Office, 1984.
52. Jelliffe DB. The assessment of the national status of the community. Geneva: World health organization. 1966.
53. Grant JP, Custer PB, Thurlow J. Current techniques of nutritional assessment Surg Clin N Amer 1981; 61: 437-63.
54. Bjorntorp P. Regional patterns of fat distribution. Ann Intern Med 1985; 103: 994-5.
55. Thomas AE, McKay DA, Cutlip MB. A nomograph method for assessing body weight. Am J Clin Nutr 1976; 29: 302-4.

56. Connway JM, Norris KH, Bodwell CE. A new approve for the determination of body composition: infeared inheritance. *Am J Clin Nutr* 1985; 40: 1123-30.
57. Monotest cholesterol. Mannheim: Boehringer Mannheim GmbH Diagnostica, 1990.
58. Test-combination free cholesterol. Mannheim: Boehringer Mannheim GmbH Diagnostica, 1987.
59. Peridochrom triglycerides. Mannheim : Boehringer Mannheim GmbH Diagnostica, 1982.
60. Burstein M. Determination of HDL-cholesterol. *Lipid Res* 1970; 11: 583-7.
61. Lopes-Virella MF, Stone P, Ellis S, Colwel JA. Cholesterol determination in high-density lipoproteins separated by three different methods. *Clin Chem* 1977; 23: 882-4.
62. Friedewald WT, Levy RI, Frederickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972; 18: 499-502.
63. Turbiquant. Marburg: Behringwerke AG, 1991.
64. The Thorp micro-nephelometer MK IV. Cheshire: Scientific Furnishings Limited, 1979.
65. Bartlett GR. Phosphorus assay in column chromatography. *J Biol Chem* 1959; 234: 466-8.

66. Rose HG, Oklander M. Improved procedure for the extraction of lipids from human erythrocyte. *J Lipid Res* 1965; 6: 428-31.
67. Muskiet FAJ, Van Doormaal JJ, Martini IA, et al. Capillary gas chromatographic profiling of total long chain fatty acids and cholesterol in biological materials. *J Chromatog* 1983; 278: 231-44.
68. Nie NH, Hull CH, Jenkins JG, et al. Statistical package for the social sciences, 2nd ed. New York: McGraw-Hill, 1975.
69. Thompson G. Hand book of hyperlipidemia. London: Current Science Ltd, 1989.
70. Heiss G, Tamir I, Davis CE, Tyroler HA, et al. Lipoprotein cholesterol distributions in selected North American populations. The Lipid Research Clinics Program Prevalence Study. *Circulation* 1980; 61: 302-15.
71. Tamir J, Heiss G, Glueck CJ, Christensen B, Switrovich P, Rijkind BM. Lipid and lipoprotein distributions in white children ages 6-19 years. The Lipid Research Clinics. Program Prevalence Study. *J Clin Dis* 1981; 34: 27-39.

72. Connor SL, Connor WE, Sexton G, Calvin L, Bacon S. The effects of age, body weight and family relationships on plasma lipoproteins and lipids in men, women and children of randomly selected families. *Circulation* 1982; 65: 1290-8.
73. Alaupovic P. Apolipoproteins and lipoproteins. *Atherosclerosis*. 1971; 13: 141.
74. Grundy SM. Cholesterol and atherosclerosis : diagnosis and treatment. Philadelphia: JB Lippincott Co, 1990.
75. Westerman M. Hypocholesterolemia and anemia. *Br J Haemat* 1971; 31: 87-94.
76. World Health Organization. Nutritional anemias: WHO Technical Report Series No. 405, 1968.
77. World Health Organization. Diabetes mellitus. WHO Technical Report Series No. 694, 1983.
78. Reaven GM. Role of insulin resistance in human disease. *Diabetes*. 1988; 37: 1595-607.
79. Hafner SM, Valdez RA, Hazuda HP, Mitchell BD, Morales PA, Stern MP. Prospective analysis of the insulin resistance syndrome (syndrom X). *Diabetes* 1992; 41: 45-22.
80. Kaplan NM. The deadly quartet. *Arch Intern Med* 1989; 149: 1514-20.

81. Taskinen M-R. Strategies for the diagnosis of metabolic syndrome. *Curr Opin Lipidol* 1993; 4: 434-43.
82. Despres' J-P. Abdominal obesity as important component of insulin-resistance syndrome. *Nutrition* 1993; 9: 452-9.
83. Bonaa KH, Thelle DS. Association between blood pressure and serum lipids in a population: the Tromso study. *Circulation* 1991; 83: 1305-14.
84. Nestel PJ. Optimizing dietary fatty acids to prevent coronary heart disease. In: Kim WY, Lee YC, Lee KY, Ju JS, Kim SH, eds. *Proceedings of the 14th International Congress of Nutrition*. Seoul: Ewha Womans University, 1990:427-30.
85. Wood DA, Riemersma RA, Butler S, et al. Linoleic and eicosapentaenoic acids in adipose tissue and platelets and risk of coronary heart disease. *Lancet* 1987; 1: 177-83.
86. Tanphaichitr V, Leelahagul P, Pakpeankitvatana R. Nutritional factors lowering serum total cholesterol level. *Intern Med* 1991; 7: 32-4.
87. Thornbarg JT, Rudel LL. How do polyunsaturated fatty acids lower lipids? *Curr Opin Lipidol* 1992; 3: 17-21.



88. Castelli WP, Doyle JT, Gordon T, et al. Alcohol and blood lipids. *Lancet* 1977; 2: 153-5.
89. Bonaa KH, Arnesen E. Association between heart rate and atherogenic blood lipid fractions in a population: The Tromso study. *Circulation* 1992; 86: 394-405.
90. Williams PT, Wood Pd, Haskell WL, Varnizan K. The effects of running mileage and duration on plasma lipoprotein levels. *JAMA* 1982; 247: 2674-9.
91. Rosenman RH, Brand RJ, Sholtz RI, Jenkins CD. Relation of corneal arcus to cardiovascular risk factors and the incidence of coronary disease. *N Engl J Med* 1974; 291: 1322-4.