Lipid disorders in transitional societies with particular reference to triglycerides and HDL-cholesterol

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Like Western populations, affluent urban populations in developing countries are facing the problem of dyslipidemia, an important risk factor of coronary heart disease. Our study of 453 affluent, urban Thai women revealed that the prevalences of type IIa, IIb, IV and V hyperlipoproteinemias were 32.5, 2.2, 2.4, and 0.4%, respectively. Based on a waist-over-hip circumference ratio (WHR) of > 0.8 and body mass index (BMI) of > 25.0 kg/m^2 to indicate abdominal and overall obesity, respectively, the prevalences of abdominal obesity, overall obesity, and combined abdominal and overall obesity in these women were 32.9, 5.7, and 21.2%, respectively. Both BMI and WHR in these women had significantly positive influences on their serum triglyceride (TG) and apo B levels, and significantly negative influences on their serum HDL-cholesterol levels. Only BMI had a significantly positive influence on their serum total cholesterol (TC) and LDL-cholesterol levels but a significantly negative influence on their serum apo A-I levels. A lipid-lowering effect of linoleic acid was shown in 101 dyslipidemic women receiving dietary intervention for 8 weeks, evidenced by significantly negative relationships between their serum 18:2 n-6 levels and serum TC, LDL-C, TG, and apo B levels.

Key words: dyslipidemia, heart disease, Thai women, obesity.

Dyslipidemia and coronary heart disease risk

Studies in Western populations have revealed that high serum total cholesterol (TC) levels due to elevated serum LDL-C levels are directly related to coronary heart disease (CHD) risk while serum HDL-C levels are inversely related to risk. Risk is also conferred when elevated serum triglyceride (TG) levels and low HDL-C levels occur with elevated serum TC or LDL-C levels, or serum TC/HDL-C or LDL-C/HDL-C ratio is high and serum TG is elevated.[1] Besides, hypertriglyceridemia is often associated with small dense LDL, a particularly atherogenic form of LDL.

In developing countries, populations with low socioeconomic status usually exhibit generalised hypolipidemia related to their inadequate protein-energy status. On the contrary, dyslipidemia is commonly found in affluent, urban populations.[2,3] This paper presents the prevalences of dyslipidemia and the influences of obesity and linoleate intake on serum lipoprotein levels in affluent, urban Thai women.

Population profile

In 1991, various risk factors for CHD including serum TC, LDL-C, HDL-C, triglyceride (TG), overall obesity by body mass index (BMI), abdominal obesity by waist-over-hip circumference (WHR) ratio, and serum fatty acid pattern, were assessed in 453 female Ramathibodi hospital staff (FRHS) residing in Bangkok, Thailand. Their age ranged from 19 to 61 years; the percentages of these women with 19–29, 30–39, 40–49 and 50–61 years were 21.4, 34.0, 34.9, and 9.7, respectively.[4,5]

Prevalences of dyslipidemia

Means ± SEM of serum TC, LDL-C and TG in 453 FRHS were 5.2 ± 0.1, 3.2 ± 0.1, 1.5 ± 0.1 and 1.2 ± 0.1 mmol/L, respectively. Their serum apo A-I and apo B were 148 ± 1 and 112 ± 1 mg/dL, respectively. Based on the National Cholesterol Education Program (NCEP) Expert Panel's criteria for dyslipidemia,[6] out of 453 FRHS, 258 (57%) were normolipidemia (TC < 5.17, LDL-C < 3.36, HDL-C ≥ 0.9 and TG < 2.26 mmol/L); 147 (32.5%) were type IIa hyperlipoproteinemia (TC ≥ 5.17, LDL-C ≥ 3.36 and TG < 2.26 mmol/L); 10 (2.2%) were type IIb hyperlipoproteinemia (TC ≥ 5.17, LDL-C ≥ 3.36 and TG ≥ 2.26 mmol/L); 11 (2.4%) were type IV hyperlipoproteinemia (TC < 5.17, LDL-C < 3.36, and TG ≥ 2.26–4.51 mmol/L); 2 (0.4%) were type V hyperlipoproteinemia (TG ≥ 4.52 mmol/L), 19 (4.2%) had serum TC ≥ 5.17 mmol/L only; 4 (0.9%) had serum LDL-C ≥ 3.36 mmol/L only; and 2 (0.4%) had serum HDL < 0.9 mmol/L only.

Table 1 shows that though 19 FRHS with serum TC ≥ 5.17 mmol/L only had significantly higher mean serum LDL-C levels than 258 normolipidemic FRHS, their serum LDL-C levels were still within the desirable limit whereas their significantly higher mean serum HDL-C levels reached the desirable level of ≥ 1.55 mmol/L, a negative risk factor for CHD.[6] Among the type IIa, IIb and IV hyperlipo-

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proteinemic FRHS, type IIa had the highest serum TC and LDL-C levels whereas type IIb had the highest serum apo B level, serum TC/HDL-C and LDL-C/HDL-C ratios, and the lowest serum HDL-C level.

Influence of obesity on serum lipoprotein levels

Obesity is a disease due to long-term positive energy balance caused by high energy intake and/or lack of physical activity. Those with abdominal obesity are at risk of hypertension, hyperinsulinemia, type II DM , and dyslipidemia.[5,7]

Based on the cut-off point of WHR > 0.8 to indicate abdominal obesity and BMI of < 20.0, 20.0–24.9 and > 25.0 kg/m^2 to indicate underweight, normal weight, and overall obesity, respectively, 453 FRHS were divided into six groups: 112 (24.7%) normal weight without abdominal obesity, 125 (27.6%) normal weight with abdominal obesity, 70 (15.5%) underweight without abdominal obesity, 24 (5.3%) underweight with abdominal obesity, 26 (5.7%) overall obesity without abdominal obesity, and 96 (21.2%) combined overall and abdominal obesity.[7] The validity of such categorisations to assess the extent and severity of obesity is confirmed by the differences in their mean body fat mass (BFM) determined by near-infrared absorptiometry (Table 2).[8,9]

In 208 FRHS without abdominal obesity, 26 subjects with overall obesity had significantly lower serum HDL-C levels but significantly higher TC/HDL-C and LDL-C/HDL-C ratios than 112 normal weight subjects. In 245 FRHS with abdominal obesity, 96 subjects with overall obesity had significantly lower serum HDL-C but significantly higher TC, TG, TC/HDL-C, LDL-C/HDL-C and apo B levels than 125 normal weight subjects. Besides, they also had significantly higher serum TG and apo B levels than 26 subjects with overall obesity without abdominal obesity (Table 2).

Multiple regression has also revealed that both BMI and WHR in 453 FRHS had significantly positive influences on their serum TG (log TG = – 0.72 + 0.01 BMI + 0.57 WHR, r = 0.41, P < 0.0001) and apo B levels (apo B = 2.44 + 2.13 BMI + 73.65 WHR, r = 0.39, P < 0.0001) but significantly negative influences on their serum HDL-C levels (HDL-C = 2.60 – 0.02 BMI – 0.80 WHR, r = –0.24, P < 0.00001); only BMI had significantly positive influences on their serum TC (r = 0.16, P = 0.0006), LDL-C (r = 0.14, P = 0.002), TC/HDL-C (r = 0.34, P < 0.0001) and LDL-C/HDL-C levels (r = 0.29, P < 0.0001) but significantly negative influence on serum apo A-I levels (r = –0.15, P < 0.0001).

Our findings are consistent with the adverse effects of obesity, especially abdominal obesity, on VLDL, LDL and HDL metabolism.[5] Special attention must be paid to those with elevated seurm TG, low HDL-C and high TC/HDL-C ratios because our 12-year follow-up study (1985–97) in

Table 1. Means ± SEM of serum lipid and apoprotein levels and lipid ratios in normolipidemic and dyslipidemic female Ramathibodi hospital staff

Parameter	Normal n = 258	TC ≥ 5.17 mmol/L only n = 19	Lipoprotein phenotype IIa n = 147	IIb n = 10	IV n = 11
TC (mmol/L)	4.54 ± 0.03	5.45 ± 0.01^{a1}	6.43 ± 0.06^{a1b1}	6.25 ± 0.11^{a1b1}	5.42 ± 0.16^{a1c1d2}
LDL-C (mmol/L)	2.60 ± 0.03	3.13 ± 0.04^{a1}	4.40 ± 0.06^{a1b1}	3.92 ± 0.10^{a1b1c2}	2.56 ± 0.22^{b3c1d1}
HDL-C (mmol/L)	1.45 ± 0.02	1.76 ± 0.06^{a1}	1.55 ± 0.06	1.10 ± 0.08^{a1b1c3}	1.36 ± 0.14^{b3}
TG (mmol/L)	1.06 ± 0.02	1.20 ± 0.10	1.20 ± 0.04^{a2}	2.72 ± 0.11^{a1b1c1}	3.09 ± 0.18^{a1b1c1}
TC/HDL-C (mmol/L)	3.24 ± 0.04	3.18 ± 0.14	4.43 ± 0.08^{a1b1}	5.90 ± 0.35^{a1b1c1}	4.44 ± 0.49^{a4b3d3}
LDL-C/HDL-C (mmol/L)	1.88 ± 0.04	1.84 ± 0.11	3.06 ± 0.07^{a1b1}	3.73 ± 0.28^{a1b1c3}	2.17 ± 0.35^{c2d2}
Apo A-I (mg/dL)	147.1 ± 1.9	166.6 ± 6.1^{a3}	149.4 ± 2.6^{b3}	138.3 ± 8.3^{b3}	148.4 ± 9.0
Apo B (mg/dL)	98.8 ± 1.5	104.6 ± 4.9	129.5 ± 2.2^{a1b1}	156.6 ± 8.2^{a1b1c2}	138.4 ± 8.2^{a1b2}

Significant difference from normal: ^{a1} P < 0.0005, ^{a2} P < 0.005, ^{a3} P < 0.01,^{a4} P < 0.05. Significant difference from TC ≥ 5.17 mmol/L: ^{b1} P < 0.0005, ^{b2} P < 0.005, ^{b3} P < 0.05. Significant difference from IIa: ^{c1} P < 0.0005, ^{c2} P < 0.005, ^{c3} P < 0.05. Significant difference from IIb: ^{d1} P < 0.0005, ^{d2} P < 0.005, ^{d3} P < 0.05.

Table 2. Means ± SEM of body fat mass, serum lipids, apoprotein, and lipid ratios in 453 female Ramathibodi hospital staff by waist-over-hip circumference ratio and body mass index

Parameter	WHR < 0.8.g BMI, kg/m^2 < 20.0 n = 70	20.0–24.9 n = 112	≥ 25.0 n = 26	WHR > 0.8.g BMI, kg/m^2 < 20.0 n = 24	20.0–24.9 n = 125	≥ 25.0 n = 46
BFM (kg)	15.45 ± 0.36	19.76 ± 0.24^{a1}	26.37 ± 0.48^{a1b1}	15.89 ± 0.43	20.93 ± 0.21^{a1A1}	27.22 ± 0.51^{a1b1}
TC (mmol/L)	5.18 ± 0.13	5.17 ± 0.09	5.43 ± 0.21	4.80 ± 0.16	5.22 ± 0.10^{a4}	5.53 ± 0.10^{a2b4}
LDL-C (mmol/L)	3.17 ± 0.12	3.19 ± 0.09	3.58 ± 0.21	2.78 ± 0.14^{A2}	3.21 ± 0.10^{a4}	3.42 ± 0.10^{a2}
HDL-C (mmol/L)	1.70 ± 0.12	1.51 ± 0.02	1.37 ± 0.06^{b4}	1.51 ± 0.06	1.44 ± 0.03	1.34 ± 0.03^{a4b4}
TG (mmol/L)	0.94 ± 0.04	1.04 ± 0.04	1.07 ± 0.06	1.13 ± 0.10	1.23 ± 0.06^{A2}	1.67 ± 0.08^{a1b1A1}
TC/HDL-C (mmol/L)	3.34 ± 0.12	3.50 ± 0.08	4.14 ± 0.21^{a2b3}	3.25 ± 0.12	3.78 ± 0.11^{a2A2}	4.33 ± 0.12^{a1b2}
LDL-C/HDL-C (mmol/L)	2.07 ± 0.10	2.17 ± 0.07	2.76 ± 0.20^{a2b3}	1.90 ± 0.11	2.33 ± 0.09^{a2}	2.67 ± 0.10^{a1b3}
Apo A-1 (mg/dL)	158.2 ± 4.2	147.7 ± 2.7^{a4}	140.2 ± 5.8^{a4}	160.5 ± 6.4	144.2 ± 2.1^{a3}	145.4 ± 3.3^{a4}
Apo B (mg/dL)	97.9 ± 3.0	105.7 ± 2.4^{a4}	114.6 ± 5.6^{a3}	100.8 ± 5.7	112.l ± 2.5	130.3 ± 3.2^{a1b1A2}

Within the same waist-over-hip circumference ratio (WHR), significant difference from body mass index (BMI) < 20.0 kg/m^2: ^{a1} P < 0.005, ^{a2} P < 0.005, ^{a3} P < 0.01, ^{a4} P < 0.05. Significant difference from BMI 20.0–24 9 kg/m^2: ^{b1} P < 0.0005, ^{b2} P < 0.005, ^{b3} P < 0.01, ^{b4} P < 0.05. Significant difference from WHR ≤ 0.8 with the same BMI: ^{A1} P < 0.0005, ^{A2} P < 0.05.

3313 afffluent, urban Thais aged 35–54 years showed 23 coronary artery disease deaths and significantly higher mean serum TC/HDL-C ratios, significantly lower mean serum HDL-C levels, and higher mean serum TG levels than those in 3156 survivors.[10]

Lipid-lowering effect of linoleic acid

Out of 195 dyslipidemic FRHS, 101 participated in an 8-week dietary intervention. These subjects were advised to restrict intakes of total fat and saturated fatty acids to ≤ 30 and < 10% of total calories, respectively. They were also advised to reduce cholesterol intake to < 300 mg/day and increase linoleate intake to 7–10% of total calories. After this 8-week dietary intervention, 13 FRHS became normolipidemia. The beneficial role of linoleate intake on lipoprotein metabolism in these FRHS was evidenced by significantly negative relationships between their serum 18:2 n-6 levels and their serum TC ($r = –0.12$, $P = 0.04$), LDL-C ($r = –0.15$, $P = 0.001$), TG ($r = –0.18$, $P = 0.002$) and apo B levels ($r = –0.15$, $P = 0.008$).

Conclusion

Our study has revealed that dyslipidemia is prevalent in affluent, urban Thai women. Overall and abdominal obesity are important causes of their dyslipidemia. Increased physical activity and appropriate dietary intake are important measures to be implemented in the daily lifestyles of these women in order to reduce the problem of dyslipidemia and thus, CHD.

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