Int J Clin Exp Pathol 2016;9(10):10688-10697
www.ijcep.com /ISSN:1936-2625/IJCEP0026748

Original Article

Serum lipid levels and the risk factors in the Mulao and Han ethnic groups

Ke-La Li[1], Rui-Xing Yin[1], Dai-Xun Wei[2], Wei-Xiong Lin[3], De-Zhai Yang[3], Ren-Da Lu[2], Jin-Zhen Wu[1], Wan-Ying Liu[1], Lin Zhang[1], Shang-Ling Pan[4]

[1]Department of Cardiology, Institute of Cardiovascular Diseases, The First Affiliated Hospital, Guangxi Medical University, Nanning, Guangxi, China; [2]The Disease Control and Prevention Center, Luocheng Mulao Autonomous County, Hechi, Guangxi, China; [3]Department of Molecular Genetics, Medical Scientific Research Center, Guangxi Medical University, Nanning, Guangxi, China; [4]Department of Pathophysiology, School of Premedical Science, Guangxi Medical University, Nanning, Guangxi, China

Received February 26, 2016; Accepted May 22, 2016; Epub October 1, 2016; Published October 15, 2016

Abstract: The Mulao ethnic group is an isolated minority in China. Little is known about the prevalence of dyslipidemia and its risk factors in this population. The aim of this study was to detect the difference in serum lipid levels and the risk factors in the Mulao and Han ethnic groups. A cross-sectional study of dyslipidemia was conducted in 1055 unrelated subjects of Mulao and 969 participants of Han ethnic groups. Serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and apolipoprotein (Apo) B levels were higher but the high-density lipoprotein cholesterol (HDL-C) and ApoA1 levels, and the ApoA1/ApoB ratio were lower in Mulao than in Han ($P < 0.05$-0.001). The prevalence of hypercholesterolemia and hyperlipidemia was also higher in Mulao than in Han (43.98% *vs*. 36.84% and 52.23% *vs*. 45.51%, $P < 0.01$; respectively). The effects of sex, age, body mass index (BMI), hypertension, alcohol consumption, and cigarette smoking on serum lipid levels were different between the Mulao and Han populations. The prevalence of hyperlipidemia was positively correlated with BMI, hypertension and the intakes of total energy and total fat in Han, whereas it was positively associated with age, alcohol consumption, BMI, systolic blood pressure, diastolic blood pressure, and the intakes of total energy and total fat in Mulao ($P < 0.05$-0.001). The difference in the serum lipid levels and the prevalence of hyperlipidemia between the two ethnic groups might result from the combined effects of different education level, BMI, hypertension, diet, lifestyle, and genetic background.

Keywords: Lipids, apolipoproteins, hyperlipidemia, cross-sectional studies, diet, lifestyle, risk factors

Introduction

Dyslipidemia is a common health problem in China, with rising prevalence in recent decades. It is well known that high levels of total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), and apolipoprotein (Apo) B, and low levels of high-density lipoprotein cholesterol (HDL-C) and ApoA1 are correlated with the progression of atherosclerosis and a higher incidence of coronary artery disease [1-5]. Serum lipid levels and the prevalence of dyslipidemia are determined by multiple environmental factors such as diet [6-8], lifestyle [8], physical inactivity [9, 10], and genetic factors [11]. In addition, there may be an ethnic difference in the prevalence of dyslipidemia [7, 12, 13].

There are 56 nationalities in China. Han is the largest group and Mulao is one of the 55 minorities with a population of 216,257 in 2010. Historical data can trace the history of Mulao minority back to the Jin Dynasty (AD 265-420). Mulao ethnic group is a relatively conservative and isolated minority, and the genetic relationship between Mulao ethnic group and other minorities in Guangxi was much closer than that between Mulao and Han or Uighur nationality [14]. In several previous studies, we have shown that the genetic polymorphisms of some lipid metabolism-related genes in the Mulao population were different from those in Han Chinese [15-19]. To the best of our knowledge, however, the serum lipid profiles and the prevalence of dyslipidemia in this isolated population have not been reported previ-

ously. Therefore, the present study was undertaken to compare the differences in serum lipid levels and their risk factors in the Chinese Mulao and Han populations from the same area.

Material and methods

Participants

A total of 1055 unrelated subjects of Mulao nationality and 969 participants of Han nationality were included in this study. They were randomly selected from our previous stratified randomized samples [15-19]. There were 504 males (47.77%) and 551 females (52.23%) in Mulao, and 450 men (46.44%) and 519 women (53.56%) in Han. All subjects were rural agricultural workers from the same area. The age of the subjects ranged from 15 to 93 (mean 54.89 ± 14.97) years in Mulao, and 16 to 92 (mean 54.43 ± 13.97) years in Han. Ages less than 40 years were 162 (15.36%) people in Mulao and 139 (14.34%) persons in Han; 40-49 years were 218 (20.66%) people in Mulao and 214 (22.08%) persons in Han; 50-59 years were 221 (20.95%) people in Mulao and 213 (21.98%) persons in Han; 60-69 years were 252 (23.89%) people in Mulao and 239 (24.66%) persons in Han; and 70 years and over were 202 (19.15%) people in Mulao and 164 (16.92%) persons in Han. The participants with a history or evidence of hepatic, renal, thyroid diseases, and heart attack or myocardial infarction, stroke, congestive heart failure, diabetes mellitus or fasting blood glucose ≥ 7.0 mmol/L determined by glucose meter have been excluded. They were not taking medications known to affect serum lipid levels (lipid-lowering drugs such as statins or fibrates, beta-blockers, diuretics, or hormones). The present study was approved by the Ethics Committee of the First Affiliated Hospital, Guangxi Medical University. Informed consent was obtained from all subjects.

Epidemiological survey

The survey was carried out using internationally standardized methods. Information on demographics, diet, intake of alcoholic beverages, and cigarette smoking was collected with standardized questionnaires. Dietary intake was assessed by the single 24-h dietary recall method. All subjects were requested to maintain their usual diet before testing [20]. The intakes of macronutrients from the ingredients were calculated by using the 2002 Chinese Food Composition Table [21]. Physical activity was ascertained with the use of a modified version of the Harvard Alumni Physical Activity Questionnaire [22]. The alcohol information included questions about the number of grams of rice wine, wine, beer, or liquor consumed during the preceding 12 months. Current smoking was defined as more than one cigarette per day. Participants who reported having smoked ≥ 100 cigarettes during their lifetime were classified as current smokers if they currently smoked and former smokers if they did not. Body height, weight, waist circumference were measured, and body mass index (BMI, kg/m^2) were calculated. Sitting blood pressure was measured three times with the use of a mercury sphygmomanometer after the subject rested for 5 minutes, and the average of the three measurements was used for the blood pressure levels. Systolic blood pressure was determined by the first Korotkoff sound, and diastolic blood pressure by the fifth Korotkoff sound. Body weight was measured using a portable balance scale. Height was measured using a portable steel measuring device. Waist circumference was measured with a nonstretchable measuring tape.

Biochemical analysis

Venous blood samples were obtained from all subjects after an overnight fast. Serum levels of TC, TG, HDL-C, and LDL-C were determined enzymatically using commercially available kits (RANDOX Laboratories Ltd., Ardmore, Diamond Road, Crumlin Co. Antrim, United Kingdom, BT29 4QY; or Daiichi Pure Chemicals Co., Ltd., Tokyo, Japan), respectively. Serum ApoA1 and ApoB levels were measured by an immunoturbidimetric assay (RANDOX Laboratories Ltd.). All determinations were performed with an autoanalyzer (Type 7170A; Hitachi Ltd., Tokyo, Japan) in our Clinical Science Experiment Center [15-19].

Diagnostic criteria

The normal values of serum TC, TG, HDL-C, LDL-C, ApoA1, ApoB, and the ratio of ApoA1 to ApoB in our Clinical Science Experiment Center were 3.10-5.17, 0.56-1.70, 1.16-1.42, 2.70-3.10 mmol/L, 1.20-1.60, 0.80-1.05 g/L, and 1.00-

Table 1. Comparison of the general characteristics, serum lipid levels and the prevalence of hyperlipidemia between the Mulao and Han populations

Characteristics	Mulao (*n* = 1055)	Han (*n* = 969)	*t* (x^2)	*P*
Age (years)	54.89±14.97	54.43±13.97	0.713	0.476
Male/female	504/551	450/519	0.360	0.548
Education level (years)	4.62±3.57	6.48±3.73	11.461	0.000
Physical activity (h/week)	46.63±8.62	44.82±7.68	4.971	0.000
Height (cm)	155.33±8.11	153.27±8.27	5.655	0.000
Weight (kg)	53.33±9.59	52.60±8.84	1.776	0.076
Body mass index (kg/m^2)	23.03±3.16	22.33±2.94	5.147	0.000
> 24 kg/m^2 [*n* (%)]	266 (25.21)	230 (23.74)	0.596	0.440
Waist circumference (cm)	76.95±8.91	75.08±8.01	4.950	0.000
Alcohol consumption [*n* (%)]	258 (24.46)	377 (38.91)	48.991	0.000
Cigarette smoking [*n* (%)]	271 (25.69)	302 (31.17)	7.471	0.006
Energy (kJ/day)	9023.28±402.63	8924.62±388.45	5.601	0.000
Carbohydrate (g/day)	424.23±22.68	394.61±16.32	33.467	0.000
Protein (g/day)	52.86±6.27	48.63±7.28	14.037	0.000
Total fat (g/day)	30.26±3.64	24.68±4.65	30.187	0.000
Dietary cholesterol (mg/day)	202.65±90.53	168.58±98.49	8.109	0.000
Prevalence of hypertension [*n* (%)]	421 (39.91)	314 (32.40)	12.287	0.001
Total cholesterol (TC, mmol/l)	5.06±1.15	4.92±0.97	2.947	0.003
TC > 5.17 mmol/l [*n* (%)]	464 (43.98)	357 (36.84)	10.677	0.001
Triglyceride (TG, mmol/l)	1.41±1.10	1.34±0.98	1.507	0.132
TG > 1.70 mmol/l [*n* (%)]	236 (22.37)	186 (19.20)	3.085	0.079
HDL-C (mmol/l)	1.75±0.45	1.91±0.47	7.823	0.000
HDL-C < 1.16 mmol/l [*n* (%)]	73 (6.92)	32 (3.30)	13.435	0.000
LDL-C (mmol/l)	2.98±0.88	2.73±0.79	6.704	0.000
LDL-C > 3.10 mmol/l [*n* (%)]	435 (41.23)	268 (27.66)	41.058	0.000
Apolipoprotein A1 (ApoA1, g/l)	1.33±0.40	1.42±0.22	6.196	0.000
ApoA1 < 1.20 g/l [*n* (%)]	270 (25.59)	133 (13.73)	44.604	0.000
Apolipoprotein B (ApoB, g/l)	1.01±0.58	0.91±0.22	5.044	0.000
ApoB > 1.05 g/l [*n* (%)]	230 (21.80)	220 (22.70)	0.238	0.626
ApoA1/ApoB	1.57±0.90	1.64±0.50	2.137	0.033
ApoA1/ApoB < 1.00 [*n* (%)]	193 (18.29)	39 (4.03)	101.333	0.000
Prevalence of hyperlipidemia [*n* (%)]	551 (52.23)	441 (45.51)	9.118	0.003

HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TC > 5.17 mmol/l, hypercholesterolaemia; TG > 1.70 mmol/l, hypertriacylglycerolaemia.

2.50, respectively. The individuals with TC > 5.17 mmol/L and/or TG > 1.70 mmol/L should be defined as hyperlipidemic [15-19]. Hypertension was defined as an average systolic pressure of 140 mmHg or greater and/or an average diastolic pressure of 90 mmHg or greater, and/or self-reported pharmacological treatment for hypertension within the 2 weeks prior to the interview [23]. Normal weight, overweight and obesity were defined as a BMI < 24, 24 to 28, and > 28 kg/m^2, respectively [15-19, 23].

Statistical analysis

The measurement data are presented as mean ± SD. All analyses were performed with SPSS 11.5 (SPSS Inc., Chicago, Illinois). Differences in mean values were assessed using analysis of covariance (ANCOVA) and the Student's unpaired *t* test. Sex, age, BMI, hypertension, alcohol consumption, cigarette smoking were included in the statistical models as covariates. The enumeration data were expressed as percentage. The difference of percentage was

Table 2. Effects of sex, BMI, hypertension, alcohol consumption, cigarette smoking, and age on serum lipid levels between the Mulao and Han populations

Characteristics	N	TC (mmol/L)	TG (mmol/L)	HDL-C (mmol/L)	LDL-C (mmol/L)	ApoA1 (g/L)	ApoB (g/L)	ApoA1/ ApoB
Mulao								
Male	504	5.07±1.09	1.49±1.04	1.73±0.48	2.91±0.85	1.34±0.41	1.04±0.60	1.50±0.62
Female	551	5.03±1.18	1.25±0.69^{c}	1.76±0.40	3.03±0.88^{a}	1.32±0.38	0.97±0.49^{a}	1.56±0.63
BMI ≤ 24 (kg/m^2)	789	4.99±1.13	1.27±0.80	1.80±0.45	2.92±0.86	1.35±0.41	0.96±0.52	1.60±0.64
BMI > 24 (kg/m^2)	266	5.21±1.14^{b}	1.65±1.04^{c}	1.60±0.38^{c}	3.14±0.89^{b}	1.30±0.35	1.12±0.60^{c}	1.34±0.52^{c}
Normotensive	634	4.93±1.09	1.28±0.83^{c}	1.75±0.43	2.90±0.84	1.32±0.38	0.96±0.53	1.57±0.65
Hypertensive	421	5.22±1.19^{c}	1.50±0.95	1.75±0.46	3.08±0.91^{b}	1.36±0.41	1.05±0.56^{b}	1.47±0.59^{b}
Nondrinker	797	5.02±1.14	1.31±0.79	1.72±0.42	3.02±0.86	1.30±0.38	0.99±0.54	1.51±0.61
Drinker	258	5.12±1.14	1.56±1.12^{b}	1.82±0.49^{b}	2.85±0.89^{b}	1.44±0.43^{c}	1.03±0.55	1.60±0.66^{a}
Nonsmoker	784	5.04±1.16	1.35±0.85	1.75±0.43	3.01±0.88	1.33±0.38	0.99±0.53	1.53±0.62
Smoker	271	5.05±1.09	1.42±0.99^{c}	1.74±0.48^{b}	2.88±0.85^{a}	1.35±0.42^{c}	1.02±0.57	1.53±0.65
Age < 40	162	4.57±1.03	1.22±0.86	1.72±0.49	2.60±0.76	1.24±0.44	0.90±0.57	1.62±0.70
40-49	218	4.92±1.29	1.48±1.08	1.70±0.47	2.86±0.92	1.34±0.43	1.05±0.66	1.57±0.74
50-59	221	5.28±1.10	1.48±0.93	1.74±0.42	3.18±0.89	1.38±0.36	0.99±0.43	1.53±0.55
60-69	252	5.12±1.09	1.31±0.79	1.79±0.44	3.04±0.81	1.37±0.37	1.04±0.53	1.50±0.57
≥ 70	202	5.22±1.03	1.32±0.68	1.76±0.40	3.09±0.84	1.30±0.38	0.99±0.49	1.45±0.56
F for 5 age groups	-	11.972	3.476	1.368	13.394	3.944	2.352	2.062
P for 5 age groups	-	0.000	0.008	0.243	0.000	0.003	0.052	0.084
Han								
Male	450	4.93±0.95^{x}	1.44±1.15	1.91±0.52^{z}	2.70±0.80^{z}	1.43±0.24^{z}	0.92±0.22^{z}	1.65±0.55^{z}
Female	519	4.92±0.98	1.26±0.78^{b}	1.90±0.43^{z}	2.76±0.79^{z}	1.40±0.20^{a,z}	0.90±0.22^{y}	1.64±0.45^{x}
BMI ≤ 24 (kg/m^2)	739	4.81±0.93^{y}	1.22±0.85	1.95±0.47^{z}	2.65±0.75^{z}	1.43±0.22^{z}	0.88±0.21^{z}	1.71±0.51^{z}
BMI > 24 (kg/m^2)	230	5.28±0.98^{c}	1.75±1.22^{c}	1.78±0.46^{c,z}	3.00±0.85^{c}	1.38±0.19^{a,y}	1.01±0.23^{c,y}	1.44±0.36^{c,x}
Normotensive	655	4.81±0.93^{x}	1.24±0.91	1.92±0.45^{z}	2.68±0.75^{z}	1.41±0.21^{z}	0.90±0.21^{y}	1.65±0.43^{x}
Hypertensive	314	5.15±1.01^{c}	1.56±1.07^{c}	1.89±0.51^{z}	2.85±0.86^{b,z}	1.42±0.24^{y}	0.95±0.24^{b,y}	1.63±0.61^{z}
Nondrinker	592	4.93±0.99	1.27±0.87	1.84±0.42^{z}	2.82±0.81^{z}	1.39±0.21^{z}	0.90±0.22^{z}	1.64±0.46^{z}
Drinker	377	4.91±0.93^{x}	1.45±1.11^{b}	2.02±0.53^{c,z}	2.60±0.74^{c,z}	1.46±0.22^{c}	0.94±0.21^{b,x}	1.65±0.55
Nonsmoker	667	4.93±0.97^{x}	1.28±0.82	1.90±0.44^{z}	2.77±0.77^{z}	1.41±0.21^{z}	0.91±0.22^{z}	1.63±0.44^{y}
Smoker	302	4.91±0.96	1.49±1.25^{b}	1.93±0.54^{z}	2.64±0.84^{a,y}	1.44±0.24^{y}	0.92±0.23^{y}	1.68±0.60^{y}
Age < 40	139	5.05±1.02^{z}	1.49±1.09^{x}	1.72±0.43	2.89±0.89^{y}	1.29±0.21	0.88±0.21	1.55±0.46
40-49	214	4.87±1.03	1.49±1.13	1.87±0.47^{z}	2.69±0.79^{x}	1.39±0.19	0.92±0.23^{y}	1.60±0.44
50-59	213	4.89±0.95^{z}	1.22±0.84^{y}	1.99±0.52^{z}	2.68±0.79^{z}	1.45±0.22^{x}	0.93±0.24	1.66±0.51^{x}
60-69	239	4.97±0.93	1.21±0.81	1.97±0.45^{z}	2.77±0.77^{z}	1.46±0.22^{y}	0.91±0.21^{z}	1.69±0.56^{z}
≥ 70	164	4.85±0.90^{z}	1.38±0.99	1.91±0.45^{z}	2.68±0.72^{z}	1.45±0.21^{z}	0.91±0.21^{x}	1.68±0.48^{z}
F for 5 age groups	-	1.131	4.143	8.543	2.221	17.874	0.945	2.729
P for 5 age groups	-	0.341	0.002	0.000	0.065	0.000	0.437	0.028

TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; ApoA1/ApoB, the ratio of apolipoprotein A1 to apolipoprotein B; BMI, body mass index; $^{a}P < 0.05$, $^{b}P < 0.01$ and $^{c}P < 0.001$ in comparison with male, BMI ≤ 24 (kg/m^2), normotensive, nondrinker, or nonsmoker of the same ethnic group; $^{x}P < 0.05$, $^{y}P < 0.01$ and $^{z}P < 0.001$ in comparison with the same subgroup of the Mulao population.

tested by the Chi-square test. In order to evaluate the risk factors for hyperlipidemia, unconditional logistic regression analysis was also performed in combined population of Mulao and Han, Mulao, and Han; respectively. The backward multiple logistic regression method was used to select the risk factors significantly associated with hyperlipidemia. Total intake of each nutrient was summed over all foods consumed. The Matlab5.0 software was used for processing these procedures by the method of multiplication of matrix [24]. A *P* value of less than 0.05 was considered statistically significant.

Table 3. Effects of sex, BMI, hypertension, alcohol consumption, cigarette smoking, and age on the prevalence of dyslipidemia between the Mulao and Han populations [*n* (%)]

Characteristics	*n*	TC > 5.17 mmol/L	TG > 1.70 mmol/L	HDL-C < 1.16 mmol/L	LDL-C > 3.10 mmol/L	Apo A1 < 1.20 g/L	Apo B > 1.05 g/L	ApoA1/ ApoB < 1.00
Mulao								
Male	504	229 (45.44)	140 (27.78)	43 (8.53)	198 (39.29)	147 (29.17)	111 (22.02)	92 (18.25)
Female	551	235 (42.65)	96 (17.42)^{c}	30 (5.44)^{a}	236 (42.83)	123 (22.32)^{a}	120 (21.78)	100 (18.15)
BMI ≤ 24 (kg/m^2)	789	328 (41.57)	138 (17.49)	45 (5.70)	298 (37.77)	192 (24.33)	148 (18.76)	131 (16.60)
BMI > 24 (kg/m^2)	266	136 (51.13)^{b}	98 (36.84)^{c}	28 (10.53)^{b}	136 (51.13)^{c}	78 (29.32)	83 (31.20)^{c}	61 (22.93)^{a}
Normotensive	634	249 (39.27)	119 (18.77)	38 (5.99)	237 (37.38)	164 (25.87)	117 (18.45)	106 (16.72)
Hypertensive	421	215 (51.07)^{c}	117 (27.79)^{c}	35 (8.31)	197 (46.79)^{b}	106 (25.19)	114 (27.08)^{c}	86 (20.43)
Nondrinker	797	343 (43.04)	159(19.95)	53 (6.65)	337 (42.28)	215 (26.98)	172 (21.58)	150 (18.82)
Drinker	258	121 (46.90)	77 (29.85)^{c}	20 (7.75)	97 (37.60)	55 (21.32)	59 (22.87)	42 (16.28)
Nonsmoker	784	344 (43.88)	171 (21.81)	47 (5.99)	335 (42.73)	191 (24.36)	172 (21.94)	142 (18.11)
Smoker	271	120 (44.28)	65 (23.99)	26 (9.59)^{a}	99 (36.53)	79 (29.15)	59 (21.77)	50 (18.45)
Age								
15-39	162	41 (25.31)	30 (18.52)	13 (8.02)	33 (20.37)	53 (32.72)	20 (12.35)	33 (20.37)
40-49	218	92 (42.20)	62 (28.44)	23 (10.55)	83 (38.07)	53 (24.31)	60 (27.52)	46 (21.10)
50-59	221	119 (53.85)	56 (25.34)	14 (6.33)	113 (51.13)	43 (19.46)	53 (23.98)	29 (13.12)
60-69	252	118 (46.83)	51 (20.24)	13 (5.16)	115 (45.63)	57 (22.62)	57 (22.62)	44 (17.46)
≥ 70	202	94 (46.54)	37 (18.32)	10 (4.95)	90 (44.56)	64 (31.68)	41 (20.30)	40 (19.80)
X^2 for 5 age subgroups	-	33.297	9.702	7.316	41.893	13.979	13.618	6.013
P for 5 age subgroups	-	0.000	0.046	0.120	0.000	0.007	0.009	0.198
Han								
Male	450	170 (37.78)^{x}	99 (22.00)^{x}	19 (4.22)^{y}	123 (27.33)^{z}	62 (13.78)^{z}	108 (24.00)	19 (4.22)^{z}
Female	519	187 (36.03)^{x}	87 (16.76)^{a}	13 (2.51)^{x}	145 (27.94)^{z}	71 (13.68)^{z}	112 (21.58)	20 (3.85)^{z}
BMI ≤ 24 (kg/m^2)	739	239 (32.34)^{z}	107 (14.48)	17 (2.30)^{z}	180 (24.36)^{z}	88 (11.91)^{z}	136 (18.40)	20 (2.71)^{z}
BMI > 24 (kg/m^2)	230	118 (51.30)^{c}	79 (34.35)^{c}	15 (6.52)^{b}	88 (38.26)^{c,y}	45 (19.57)^{b,x}	84 (36.52)^{c}	19 (8.26)^{c,z}
Normotensive	655	211 (32.21)^{y}	101 (15.42)	21 (3.21)^{y}	156 (23.82)^{z}	84 (12.82)^{z}	132 (20.15)	20 (3.05)^{z}
Hypertensive	314	146 (46.50)^{c}	85 (27.07)^{c}	11 (3.50)^{y}	112 (35.67)^{c,z}	49 (15.61)^{z}	88 (28.03)^{b}	19 (6.05)^{z}
Nondrinker	592	220 (37.16)^{x}	106 (17.91)	22 (3.72)^{x}	188 (31.76)^{z}	91 (15.37)^{z}	120 (20.27)	25 (4.22)^{z}
Drinker	377	137 (36.34)^{y}	80 (21.22)^{x}	10 (2.65)^{y}	80 (21.22)^{c,z}	42 (11.14)^{z}	100 (26.53)^{a}	14 (3.71)^{z}
Nonsmoker	667	248 (37.18)^{y}	118 (17.69)	20 (2.99)^{y}	192 (28.79)^{z}	93 (13.94)^{z}	148 (22.19)	24 (3.60)^{z}
Smoker	302	109 (36.09)^{x}	68 (22.52)	12 (3.97)^{y}	76 (25.17)^{z}	40 (13.25)^{z}	72 (23.84)	15 (4.97)^{z}
Age								
15-39	139	64 (46.041)^{z}	30 (21.58)	10 (7.19)	56 (40.29)^{z}	48 (34.53)	24 (17.27)	12 (8.63)^{y}
40-49	214	74 (34.58)	56 (26.17)	7 (3.27)^{y}	57 (26.64)^{x}	36 (16.82)	47 (21.96)	9 (4.21)^{z}
50-59	213	78 (36.62)^{z}	32 (15.02)^{y}	8 (3.76)	50 (23.47)^{z}	22 (10.33)^{y}	54 (25.35)	9 (4.23)^{z}
60-69	239	88 (36.82)^{x}	33 (13.81)	4 (1.67)^{x}	69 (28.87)^{z}	17 (7.11)^{z}	55 (23.01)	5 (2.09)^{z}
≥ 70	164	53 (32.32)^{y}	35 (21.34)	3 (1.83)	36 (21.95)^{z}	10 (6.10)^{z}	40 (24.39)	4 (2.44)^{z}
X^2 for 5 age subgroups	-	6.976	14.569	9.831	15.902	71.510	3.539	11.061
P for 5 age subgroups	-	0.137	0.006	0.043	0.003	0.000	0.472	0.026

TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ApoA1, apolipoprotein A1; ApoB, apolipoprotein B; ApoA1/ApoB, the ratio of apolipoprotein A1 to apolipoprotein B; BMI, body mass index; $^{a}P < 0.05$, $^{b}P < 0.01$ and $^{c}P < 0.001$ in comparison with male, BMI ≤ 24 (kg/m^2), normotensive, nondrinker, or nonsmoker of the same ethnic group; $^{x}P < 0.05$, $^{y}P < 0.01$ and $^{z}P < 0.001$ in comparison with the same subgroup of the Mulao population.

Results

General characteristics

The general characteristics between the two ethnic groups are shown in **Table 1**. The levels of physical activity, body height, BMI and waist circumference; the intakes of macronutrients such as total energy, carbohydrate, protein, total fat and dietary cholesterol; and the prevalence of hypertension were higher in Mulao than in Han ($P < 0.01$-0.001), whereas the lev-

Table 4. Correlation of hyperlipidemia with demography, diet and lifestyle between the Mulao and Han populations

Population	Risk factor	Regression coefficient	Standard error	Wald	*P* value	Odds ratio	95% Confidence intervel
Mulao plus Han	Ethnic group	-2.380	0.086	17.065	0.000	0.788	0.615-0.920
	Sex	0.218	0.920	5.645	0.018	1.243	0.823-1.245
	Body mass index	0.693	0.109	40.394	0.000	1.999	1.639-2.517
	Systolic blood pressure	0.309	0.120	6.618	0.010	1.363	1.116-1.951
	Diastolic blood pressure	0.385	0.127	9.220	0.002	1.470	1.199-2.038
	Total energy	0.577	0.146	10.362	0.001	1.325	1.264-2.338
	Total fat	0.554	0.133	17.913	0.000	1.678	1.121-1.742
Han	Body mass index	0.844	0.159	28.214	0.000	2.325	1.694-3.169
	Hypertension	0.601	0.142	17.900	0.000	1.824	0.809-3.712
	Total energy	0.553	0.213	7.831	0.006	1.821	1.531-2.456
	Total fat	0.475	0.188	9.042	0.003	1.673	1.216-2.288
Mulao	Age	0.099	0.050	3.949	0.047	1.104	1.037-1.315
	Alcohol consumption	0.151	0.048	10.062	0.002	1.163	0.901-1.137
	Body mass index	0.584	0.152	14.843	0.000	1.794	1.358-2.460
	Systolic blood pressure	0.790	0.280	7.935	0.005	2.203	1.166-2.329
	Diastolic blood pressure	0.522	0.232	5.061	0.024	1.685	0.985-1.959
	Total energy	0.622	0.181	12.521	0.000	1.964	1.066-2.323
	Total fat	0.571	0.199	9.442	0.003	1.868	1.325-2.783

els of education, and the percentage of subjects who consumed alcohol or smoked cigarettes were lower in Mulao than in Han (P < 0.01-0.001). There were no significant differences in age, weight, and sex structure between the two ethnic groups (P > 0.05 for all).

Serum lipid levels and the prevalence of hyperlipidemia

As shown in **Table 1**, the levels of TC, LDL-C and ApoB were higher but the levels of HDL-C, ApoA1, and the ratio of ApoA1 to ApoB were lower in Mulao than in Han (P < 0.05-0.001). There was no significant difference in TG levels between the two ethnic groups (P > 0.05). The prevalence of hypercholesterolemia, hypertriacylglycerolemia and hyperlipidemia in Mulao and Han was 43.98% *vs*. 36.84% (P < 0.01), 22.37% *vs*. 19.20% (P > 0.05), and 52.23% *vs*. 45.51% (P < 0.01); respectively. The anormal rates of HDL-C, LDL-C, ApoA1 and the ApoA1/ApoB ratio were also higher in Mulao than in Han (P < 0.001 for all).

Sex, BMI, hypertension, alcohol, smoking, and age on serum lipid levels

The effects of sex, BMI, hypertension, alcohol consumption, cigarette smoking, and age on serum lipid levels between Mulao and Han are shown in **Table 2**. For the Mulao population, the levels of TG and ApoB were higher but the levels of LDL-C were lower in males than in females (P < 0.05-0.001); the levels of TC, TG, LDL-C and ApoB were higher but the levels of HDL-C and the ratio of ApoA1 to ApoB were lower in subjects with a BMI > 24 kg/m^2 than in those with a BMI ≤ 24 kg/m^2 (P < 0.01-0.001); the levels of TC, TG, LDL-C and ApoB were higher but the ratio of ApoA1 to ApoB were lower in hypertensives than in normotensives (P < 0.01-0.001); the levels of TG, HDL-C, ApoA1, and the ratio of ApoA1 to ApoB were higher but the levels of LDL-C were lower in drinkers than in nondrinkers (P < 0.05-0.001); the levels of TG and ApoA1 were higher but the levels of HDL-C and LDL-C were lower in smokers than in nonsmokers (P < 0.05-0.001); the levels of TC, TG, LDL-C and ApoA1 were influenced by age (P < 0.01 for all), higher levels of TC and TG were found in age of 40 to 59 years, higher levels of LDL-C were found in age of ≥ 50 years, and higher levels of ApoA1 were found in age of 40 to 69 years.

For the Han population, the levels of TG and ApoA1 were higher in males than in females (P < 0.05-0.01); the levels of TC, TG, LDL-C and

ApoB were higher but the levels of HDL-C, ApoA1 and the ratio of ApoA1 to ApoB were lower in subjects with a BMI > 24 kg/m^2 than in those with a BMI ≤ 24 kg/m^2 (P < 0.05-0.001); the levels of TC, TG, LDL-C and ApoB were higher in hypertensives than in normotensives (P < 0.01-0.001); the levels of TG, HDL-C, ApoA1 and ApoB were higher but the levels of LDL-C were lower in drinkers than in nondrinkers (P < 0.01-0.001); the levels of TG were higher but the levels of LDL-C were lower in smokers than in nonsmokers (P < 0.05-0.01); the levels of TG, HDL-C, ApoA1, and the ratio of ApoA1 to ApoB were influenced by age (P < 0.05-0.001), higher levels of TG were found in age of < 50 years, higher levels of HDL-C were found in age of 50 to 69 years, higher levels of ApoA1 were found in age of ≥ 50 years, and higher ApoA1/ApoB ratio was found in age of ≥ 60 years.

The effects of sex, BMI, hypertension, alcohol consumption, cigarette smoking, and age on the prevalence of dyslipidemia between the two ethnic groups are shown in **Table 3**.

Risk factors for hyperlipidemia

Multivariate logistic regression analysis showed that the prevalence of hyperlipidemia was positively correlated with BMI, hypertension and the intakes of total energy and total fat in Han (P < 0.01 for all), whereas it was positively associated with age, alcohol consumption, BMI, systolic blood pressure, diastolic blood pressure, and the intakes of total energy and total fat in Mulao (P < 0.05-0.001, **Table 4**).

Discussion

The present study shows that the levels of TC, LDL-C and ApoB were higher but the levels of HDL-C and ApoA1, and the ratio of ApoA1 to ApoB were lower in Mulao than in Han (P < 0.05-0.001). There was no significant difference in TG levels between the two ethnic groups (P > 0.05). The prevalence of hypercholesterolemia and hyperlipidemia, and the anormal rates of HDL-C, LDL-C, ApoA1 and the ApoA1/ApoB ratio were also higher in Mulao than in Han (P < 0.01 for all). These differences in serum lipid profiles and the prevalence of dyslipidemia between the two ethnic groups might result from the combined effects of different education level, BMI, hypertension, diet, lifestyle, and genetic background.

In the present study, we showed that the educational level was significantly lower in Mulao than in Han. A lack of public awareness and understanding of dyslipidemia and its complications may contribute to the epidemic of uncontrolled dyslipidemia in the Mulao population. These results also underscore the urgent need for developing a good cholesterol education program to coordinate the efforts in detection, prevention, and treatment of dyslipidemia in the rural areas of China.

The BMI level in this study was significantly higher in Mulao than in Han. The levels of TC, TG, LDL-C and ApoB were higher and the levels of HDL-C and the ApoA1/ApoB ratio were lower in the subjects with a BMI > 24 kg/m^2 than in those with a BMI ≤ 24 kg/m^2 in both ethnic groups. The relationship between obesity and dyslipidemia is well recognized. The causes of dyslipidemia in obesity may be multifactorial and include hepatic over production of very low density lipoproteins (VLDL), decreased circulating TG lipolysis and impaired the acylation-stimulating protein (ASP)/C3adesArg pathway, and peripheral free fatty acid (FFA) trapping, increased FFA fluxes from adipocytes to the liver and other tissues and the formation of small dense LDL [25].

The association of dyslipidemia and hypertension is still not completely understood. In the present study, we showed that the prevalence of hypertension was significantly higher in Mulao than in Han. The levels of TC, TG, LDL-C and ApoB in both ethnic groups were higher in hypertensives than in normotensives (P < 0.01-0.001). These results suggest that dyslipidemia may share a similar pathophysiology with hypertension [26]. The presence of dyslipidemia and the resulting endothelial damage and dysfunction may play a crucial role in the development of hypertension [27].

The intakes of total energy, total fat and dietary cholesterol were higher in Mulao than in Han because the people of Mulao nationality like to eat animal offals which contain abundant saturated fatty acids. Multivariate logistic regression analysis also showed that the prevalence of hyperlipidemia was positively correlated with the intakes of total energy and total fat in both ethnic groups. The major dietary saturated long-chain fatty acids such as myristic acid (14:0) and palmitic acid (16:0) have been asso-

ciated with deleterious effects on blood lipid metabolism, especially due to their influence on plasma TG, TC and LDL-C concentrations [28].

Lifestyle factors such as alcohol consumption and cigarette smoking have been associated with dyslipidemia [8]. In the present study, we also showed that the levels of TG, HDL-C and ApoA1 in both ethnic groups were higher but the levels of LDL-C were lower in drinkers than in nondrinkers. The levels of TG and ApoA1 in Mulao were higher but the levels of HDL-C and LDL-C were lower in smokers than in nonsmokers, and the levels of TG in Han were higher but the levels of LDL-C were lower in smokers than in nonsmokers. These discrepancies may result from different types of beverages and cigarettes. In this study, 90% of the wine drunk by Mulao was rum or local wine (made from local V.quinquangularis, named Vitis Louchengensis W.T.Wang, ined), in which the alcohol content is low. One previous study found that liquor consumption was weakly positively associated with HDL-C in men. Beer consumption in men and wine consumption in women were also positively associated with HDL-C, but were not significant in the fully adjusted model [29]. In contrast, another study showed that increased wine consumption was more related to HDL-C levels, whereas beer and spirits were related to increased TG levels [30]. In addition, 95% of the cigarettes smoked by them were natural tobacco leaves, which the toxin content may be different from the commercial cigarettes. Tobacco smoke is a complex mixture of over 4,000 chemical constituents. There are many toxins in cigarette smoke such as nicotine, cadmium, carbon monoxide, and reactive oxygen species that might contribute to the cardiovascular toxicity [31]. In contrast, a great deal of the wine drunk by Han was rice wine, in which the alcohol content is high, and 95% of the cigarettes smoked by them were commercially available cigarettes.

In addition to the environmental factors, genetic polymorphisms might also be involved in the development of dyslipidemia. Intra-ethnic marriages have been performed in Mulao from time immemorial. Thus, the hereditary characteristics and phenotypes of some lipid metabolism-related genes in Mulao may be different from those in Han. In several previous studies, we have shown that the genetic polymorphisms of some lipid metabolism-related genes in the Mulao population were different from those in Han Chinese [15-19].

Conclusions

The present study reveals that the levels of serum TC, LDL-C and ApoB were higher but the levels of HDL-C, ApoA1, and the ratio of ApoA1 to ApoB were lower in Mulao than in Han. The prevalence of hypercholesterolemia and hyperlipidemia was also higher in Mulao than in Han. The effects of sex, age, BMI, hypertension, alcohol consumption, and cigarette smoking on serum lipid levels were different between the Mulao and Han populations. The prevalence of hyperlipidemia was positively correlated with BMI, hypertension and the intakes of total energy and total fat in Han, whereas it was positively associated with age, alcohol consumption, BMI, systolic blood pressure, diastolic blood pressure, and the intakes of total energy and total fat in Mulao. The difference in the serum lipid profiles and the prevalence of hyperlipidemia between the Mulao and Han populations might result from the combined effects of different education level, BMI, hypertension, diet, lifestyle, and genetic background.

Acknowledgements

This study was supported by the National Natural Science Foundation of China (No: 30960130).

Disclosure of conflict of interest

None.

Address correspondence to: Rui-Xing Yin, Department of Cardiology, Institute of Cardiovascular Diseases, The First Affiliated Hospital, Guangxi Medical University, 22 Shuangyong Road, Nanning 530021, Guangxi, China. Tel: +86-771-5358832; Fax: +86-771-5353342; E-mail: yinruixing@163.com

References

[1] Satoh H, Nishino T, Tomita K, Tsutsui H. Fasting triglyceride is a significant risk factor for coronary artery disease in middle-aged Japanese men: Results from a 10-year cohort study. Circ J 2006; 70: 227-231.

[2] März W, Scharnagl H, Winkler K, Tiran A, Nauck M, Boehm BO, Winkelmann BR. Low-density li-

poprotein triglycerides associated with low-grade systemic inflammation, adhesion molecules, and angiographic coronary artery disease: the Ludwigshafen Risk and Cardiovascular Health study. Circulation 2004; 110: 3068-3074.

[3] Kwiterovich PO Jr, Coresh J, Smith HH, Bachorik PS, Derby CA, Pearson TA. Comparison of the plasma levels of apolipoproteins B and A-1, and other risk factors in men and women with premature coronary artery disease. Am J Cardiol 1992; 69: 1015-1021.

[4] Boden WE. High-density lipoprotein cholesterol as an independent risk factor in cardiovascular disease: Assessing the data from Framingham to the Veterans Affairs High-Density Lipoprotein Intervention Trail. Am J Cardiol 2000; 86: 19L-22L.

[5] Sharrett AR, Ballantyne CM, Coady SA, Heiss G, Sorlie PD, Catellier D, Patsch W; Atherosclerosis Risk in Communities Study Group. Coronary heart disease prediction from lipoprotein cholesterol levels, triglycerides, lipoprotein(a), apolipoproteins A-I and B, and HDL density subfractions: The Atherosclerosis Risk in Communities (ARIC) Study. Circulation 2001; 104: 1108-1113.

[6] Shekelle RB, Shryock AM, Paul O, Lepper M, Stamler J, Liu S, Raynor WJ Jr. Diet, serum cholesterol, and death from coronary heart disease. The Western Electric study. N Engl J Med 1981; 304: 65-70.

[7] Bermudez OI, Velez-Carrasco W, Schaefer EJ, Tucker KL. Dietary and plasma lipid, lipoprotein, and apolipoprotein profiles among elderly Hispanics and non-Hispanics and their association with diabetes. Am J Clin Nutr 2002; 76: 1214-1221.

[8] Ruixing Y, Jinzhen W, Yaoheng H, Jing T, Hai W, Muyan L, Yiyang L, Dongmei F, Hanjun Y, Yuming C. Associations of diet and lifestyle with hyperlipidemia for middle-aged and elderly persons among the Guangxi Bai Ku Yao and Han populations. J Am Diet Assoc 2008; 108: 970-976.

[9] Slentz CA, Houmard JA, Johnson JL, Bateman LA, Tanner CJ, McCartney JS, Duscha BD, Kraus WE. Inactivity, exercise training and detraining, and plasma lipoproteins. STRRIDE: a randomized, controlled study of exercise intensity and amount. J Appl Physiol (1985) 2007; 103: 432-442.

[10] Smith CE, Arnett DK, Tsai MY, Lai CQ, Parnell LD, Shen J, Laclaustra M, Junyent M, Ordovás JM. Physical inactivity interacts with an endothelial lipase polymorphism to modulate high density lipoprotein cholesterol in the GOLDN study. Atherosclerosis 2009; 206: 500-504.

[11] Heller DA, de Faire U, Pedersen NL, Dahlén G, McClearn GE. Genetic and environmental influences on serum lipid levels in twins. N Engl J Med 1993; 328: 1150-1156.

[12] Srinivasan SR, Freedman DS, Webber LS, Berenson GS. Black-white differences in cholesterol levels of serum high-density lipoprotein subclasses among children: the Bogalusa Heart Study. Circulation 1987; 76: 272-279.

[13] Nethononda MR, Essop MR, Mbewu AD, Galpin JS. Coronary artery disease and risk factors in Black South Africans--a comparative study. Ethn Dis 2004; 14: 515-519.

[14] Xu L, Deng QY, Li SF, Zhou LN, Gong JC, Wei BY. Genetic analysis of Mulao nationality using 15 short tandem repeats. Zhonghua Yi Xue Yi Chuan Xue Za Zhi 2008; 25: 96-100.

[15] Li Q, Wei XL, Yin RX. Association of ATP binding cassette transporter G8 rs4148217 SNP and serum lipid levels in Mulao and Han nationalities. Lipids Health Dis 2012; 11: 46.

[16] Yan TT, Yin RX, Li Q, Huang P, Zeng XN, Huang KK, Aung LH, Wu DF, Liu CW, Pan SL. Sex-specific association of rs16996148 SNP in the NCAN/CILP2/PBX4 and serum lipid levels in the Mulao and Han populations. Lipids Health Dis 2011; 10: 248.

[17] Miao L, Yin RX, Hu XJ, Wu DF, Cao XL, Li Q, Yan TT, Aung LHH, Wu JZ, Lin WX. Association of rs2072183 SNP and serum lipid levels in the Mulao and Han populations. Lipids Health Dis 2012; 11: 61.

[18] Wang W, Yin RX, Wu DF, Aung LH, Huang P, Zeng XN, Huang KK, Lin QZ, Wu J, Guo T. Phosphodiesterase 3A rs7134375 single nucleotide polymorphism and serum lipid levels. Mol Med Rep 2014; 9: 1618-1628.

[19] Aung LH, Yin RX, Wu JZ, Wu DF, Wang W, Li H. Association between the MLX interacting protein-like, BUD13 homolog and zinc finger protein 259 gene polymorphisms and serum lipid levels. Sci Rep 2014; 4: 5565.

[20] Lyu LC, Yeh CY, Lichtenstein AH, Li Z, Ordovas JM, Schaefer EJ. Association of sex, adiposity, and diet with HDL subclasses in middle-aged Chinese. Am J Clin Nutr 2001; 74: 64-71.

[21] Yang YX, Wang GY, Pan XC. The 2002 Chinese Food Composition Table. Beijing: Medical Publishing House of Beijing University; 2002. pp. 1-395.

[22] Paffenbarger RS, Wing AL, Hyde RT. Physical activity as an index of heart attack risk in college alumni. Am J Epidemiol 1978; 108: 161-175.

[23] Yin RX, Wu JZ, Liu WY, Wu DF, Cao XL, Miao L, Htet Aung LH, Zhang L, Long XJ, Li M, Pan SL. Association of several lipid-related gene polymorphisms and blood pressure variation in the Bai Ku Yao population. Am J Hypertens 2012; 25: 927-936.

[24] Xiao JS, Wang MR. Matlab5.X and Scientific Calculation. Beijing: Tsinghua Publishing House; 2000. pp. 28-36.

[25] Klop B, Elte JW, Cabezas MC. Dyslipidemia in obesity: mechanisms and potential targets. Nutrients 2013; 5: 1218-1240.
[26] Ruixing Y, Jinzhen W, Weixiong L, Yuming C, Dezhai Y, Shangling P. The environmental and genetic evidence for the association of hyperlipidemia and hypertension. J Hypertens 2009; 27: 251-258.
[27] Halperin RO, Sesso HD, Ma J, Buring JE, Stampfer MJ, Gaziano JM. Dyslipidemia and the risk of incident hypertension in men. Hypertension 2006; 47: 45-50.
[28] Lottenberg AM, Afonso Mda S, Lavrador MS, Machado RM, Nakandakare ER. The role of dietary fatty acids in the pathology of metabolic syndrome. J Nutr Biochem 2012; 23: 1027-1040.
[29] De Jong HJ, de Goede J, Oude Griep LM, Geleijnse JM. Alcohol consumption and blood lipids in elderly coronary patients. Metabolism 2008; 57: 1286-1292.
[30] Foerster M, Marques-Vidal P, Gmel G, Daeppen JB, Cornuz J, Hayoz D, Pécoud A, Mooser V, Waeber G, Vollenweider P, Paccaud F, Rodondi N. Alcohol drinking and cardiovascular risk in a population with high mean alcohol consumption. Am J Cardiol 2009; 103: 361-368.
[31] Ruixing Y, Qi B, Tangwei L, Jiaquan L. Effects of nicotine on angiogenesis and restenosis in a rabbit model. Cardiology 2007; 107: 122-131.