

Original Article

Serum lipid profiles, the prevalence of dyslipidemia and the risk factors in two isolated Chinese minorities

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Abstract: Both Jing and Mulao nationalities are the isolated minorities in China. Little is known about the prevalence of dyslipidemia between the two ethnic groups. Therefore, the aim of this study was to compare the differences in serum lipid profiles, the prevalence of dyslipidemia and their risk factors between the Jing and Mulao populations. A cross-sectional study of dyslipidemia was conducted in Dongxing city, Guangxi, China, during Dec 2011 and Jan 2012. A total of 1254 subjects of Jing and 1251 participants of Mulao were surveyed by a stratified randomized sampling. Information on demography, diet and lifestyle was collected with standardized questionnaire. Serum lipid levels were detected using the commercially available kits. The levels of low-density lipoprotein cholesterol (LDL-C), apolipoprotein (Apo) A1, and the ratio of ApoA1 to ApoB were lower but the levels of ApoB were higher in Jing than in Mulao ($P < 0.001$ for all). The prevalence of hypertriglyceridemia (32.38% vs. 24.38%), high ApoB (35.25% vs. 15.35%) and low ApoA1/ApoB ratio (22.65% vs. 16.87%) was higher and low high-density lipoprotein cholesterol (0.48% vs. 2.16%), high LDL-C (17.54% vs. 40.53%) and low ApoA1 (5.98% vs. 11.43%) was lower in Jing than in Mulao ($P < 0.001$ for all). The risk factors for serum lipid parameters and hyperlipidemia were different between the two ethnic groups. Serum lipid profiles, the prevalence of dyslipidemia and their risk factors are different between the Jing and Mulao populations. These differences may result from the combined effects of different diet, lifestyle, and genetic factors.

Keywords: Lipids, apolipoproteins, dyslipidemia, prevalence, risk factors

Introduction

Dyslipidemia is one of the most important risk factors for coronary artery disease (CAD). With the improvement of people's living standard and the change of lifestyle, the prevalence of dyslipidemia in China has been increased significantly according to the Chinese Residents of Nutrition and Health Survey in 2002 [1]. The results showed that the prevalence of dyslipidemia had been 18.6%, and there were about 160 million peoples with dyslipidemia. Dyslipidemia, one of the components of the metabolic syndrome, is closely related with obesity, type 2 diabetes, hypertension, CAD,

and stroke [2]. The prevalence of dyslipidemia may be different among diverse racial/ethnic groups [3].

There are fifty-six nationalities in China. Both Jing and Mulao nationalities are the peculiar and isolated minorities in Guangxi. Jing nationality, the only oceanic nation in China, is a very small minority of coastal fisheries with population of 22,517 who live in compact communities primarily in the three islands of Wanwei, Wutou and Shanxin in Dongxing city, Guangxi, near the Sino-Vietnamese border. About 1511, their ancestors emigrated from Vietnam to China and first settled on the three uninhabited

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lands. Mulao is one of the mountain minorities with population of 207,352 according to the China's fifth national census in 2000. Approximately ninety percent of Mulao people are dwelling in the Luocheng Mulao Autonomous County, Guangxi. Historical data can trace the history of this ethnic minority back to the Jin Dynasty (AD 265-420). The special customs and culture including their intra-ethnic marriages, dietary habits, lifestyle and genetic background are different between the two minorities, but little is known about the differences in serum lipid profiles and their risk factors between the two ethnic groups. Therefore, the aim of this study was to compare the serum lipid profiles, the prevalence of dyslipidemia and their risk factors between the Jing and Mulao populations.

Material and methods

Study population

The participants of this study were randomly selected from our previous stratified randomized samples. Previous stratified randomized sampling in Jing nationality was conducted in Dongxing city, Guangxi, China, during Dec 2011 and Jan 2012. First, teams (resident communities; inhabitants) were selected randomly from the three islands (villages). Then, sexes and age subgroups in each team were separated for the survey. Finally, the sampled resident sections were determined from the local population registers. A total of 1800 subjects were asked to participate in the study and 1674 subjects actually participated. The response rate was 93%. A total of 52 persons (3.1%) with a history and/or evidence of other diseases were excluded from the samples. The sampling of the Mulao population was also done by the same method. The present study included 1254 subjects of Jing (accounted for 5.57% of the total Jing population) and 1251 participants of Mulao. The subjects of Jing nationality consisted of 678 (54.07%) males and 576 (45.93%) females, aged from 35 to 92 years, with a mean age of 57.64 ± 13.10 years. The participants of Mulao nationality consisted of 680 (54.37%) males and 571 (45.64%) females, aged from 35 to 93 years, with a mean age of 57.53 ± 13.05 years. 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 and the Human Subjects Institutional Review Board of Guangxi Medical University. Informed consent was obtained from all subjects. Written informed consent was obtained from each participant.

Epidemiological survey

The survey was carried out using internationally standardized methods [4]. The information on demographics, medical history, dietary habits, lifestyle, education level, and physical activity was obtained with a standard questionnaire. The 24 h dietary recall method was used to determine the dietary intake of each subject. Detailed descriptions of all foods, beverages, and supplements consumed during the 24 h period before the interview, including the quantity, cooking method, and brand names, were recorded by a well trained physician. The intakes of macronutrients from the ingredients were determined using the 2002 Chinese Food Composition Table [5]. The physical examination included the measurements of several parameters such as body height, weight, waist circumference, etc., and body mass index (BMI) was calculated as weight (kg) divided by height (m) squared. Sitting blood pressure was measured three times using a mercury sphygmomanometer after the subject had rested for 5 min, and the average of the three measurements was used as the blood pressure level. Systolic blood pressure was determined by the first Korotkoff sound, and diastolic blood pressure by the fifth Korotkoff sound.

Determination of serum lipid parameters

A blood sample was obtained after an overnight fast. The levels of total cholesterol (TC) and triglyceride (TG) were determined enzymatically using commercially available kits (RANDOX Laboratories Ltd., Ardmore, Antrim, UK). The levels of high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) were detected using standard enzymatic methods (Daiichi Pure Chemicals Co., Ltd., Tokyo, Japan). Serum apolipoprotein A1 (ApoA1)

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Table 1. General characteristics, serum lipid profiles, and the prevalence of dyslipidemia between the Jing and Mulao populations

| Parameter | Jing (n = 1254) | Mulao (n = 1251) | t (x ²) | P |
|--------------------------------------|-----------------|------------------|---------------------|---------|
| Age (years) | 57.64±13.10 | 57.53±13.05 | 0.014 | 0.989 |
| Male/female | 678/576 | 680/571 | 0.021 | 0.884 |
| Education level (years) | 4.81±3.46 | 4.62±3.97 | 1.277 | 0.202 |
| Physical activity (h/week) | 42.47±8.08 | 45.31±8.89 | -8.367 | < 0.001 |
| Height (cm) | 158.30±7.95 | 153.86±7.81 | 8.656 | < 0.001 |
| Weight (kg) | 58.80±10.05 | 53.69±9.49 | 13.066 | < 0.001 |
| Body mass index (kg/m ²) | 23.39±3.17 | 22.12±3.16 | 10.005 | < 0.001 |
| > 24 kg/m ² [n (%)] | 467 (37.24) | 322 (21.17) | 38.395 | < 0.001 |
| Waist circumference (cm) | 80.35±9.35 | 76.01±8.85 | 11.940 | < 0.001 |
| Alcohol consumption [n (%)] | 201 (16.03) | 354 (28.00) | 54.655 | < 0.001 |
| Cigarette smoking [n (%)] | 235 (18.64) | 345 (27.58) | 27.492 | < 0.001 |
| Energy (kcal/day) | 2112.36±92.50 | 2181.32±96.51 | -18.275 | < 0.001 |
| Carbohydrate (g/day) | 396.08±21.72 | 418.35±23.79 | -24.468 | < 0.001 |
| Protein (g/day) | 51.76±7.45 | 49.33±7.17 | 8.317 | < 0.001 |
| Total fat (g/day) | 28.11±5.76 | 29.38±4.52 | -6.138 | < 0.001 |
| Dietary cholesterol (mg/day) | 199.24±97.38 | 203.79±99.31 | 1.159 | 0.247 |
| Total dietary fiber (g/day) | 8.33±3.48 | 9.88±4.26 | 9.974 | < 0.001 |
| Systolic blood pressure (mmHg) | 131.37±21.42 | 132.79±23.19 | -1.593 | 0.111 |
| Diastolic blood pressure (mmHg) | 80.05±10.57 | 82.35±12.22 | -5.036 | < 0.001 |
| Pulse pressure (mmHg) | 51.16±17.39 | 50.44±17.19 | 1.035 | 0.301 |
| Prevalence of hypertension [n (%)] | 468 (37.32) | 534 (42.69) | 7.511 | 0.006 |
| Total cholesterol (TC, mmol/L) | 5.15±0.89 | 5.21±1.29 | -1.427 | 0.154 |
| TC > 5.17 mmol/L [n (%)] | 569 (45.37) | 614 (49.08) | 3.451 | 0.063 |
| Triglyceride (TG, mmol/L) | 1.67±0.89 | 1.62±2.20 | 0.722 | 0.471 |
| TG > 1.70 mmol/L [n (%)] | 406 (32.38) | 305 (24.38) | 19.697 | < 0.001 |
| HDL-C (mmol/L) | 1.79±0.44 | 1.77±0.44 | 1.136 | 0.256 |
| HDL-C < 0.91 mmol/L [n (%)] | 6 (0.48) | 27 (2.16) | 13.593 | < 0.001 |
| LDL-C (mmol/L) | 2.84±0.43 | 3.04±0.91 | -7.213 | < 0.001 |
| LDL-C > 3.20 mmol/L [n (%)] | 220 (17.54) | 507 (40.53) | 160.597 | < 0.001 |
| Apolipoprotein A1 (ApoA1, g/L) | 1.30±0.23 | 1.36±0.40 | -4.621 | < 0.001 |
| ApoA1 < 1.00 g/L [n (%)] | 75 (5.98) | 143 (11.43) | 24.412 | < 0.001 |
| Apolipoprotein B (ApoB, g/L) | 1.07±0.25 | 1.00±0.55 | 4.178 | < 0.001 |
| ApoB > 1.14 g/L [n (%)] | 442 (35.25) | 192 (15.35) | 131.185 | < 0.001 |
| ApoA1/ApoB | 1.28±0.37 | 1.56±0.69 | -12.649 | < 0.001 |
| ApoA1/ApoB < 1.00 [n (%)] | 284 (22.65) | 211 (16.87) | 13.200 | < 0.001 |
| Prevalence of hyperlipidemia [n (%)] | 759 (60.53) | 719 (57.47) | 2.412 | 0.120 |

HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Hypercholesterolaemia, TC > 5.17 mmol/L; Hypertriglyceridaemia, TG > 1.70 mmol/L; Hyperlipidemia, TC > 5.17 mmol/L and/or TG > 1.70 mmol/L.

and ApoB levels were measured by an immunoturbidimetric assay (RANDOX Laboratories Ltd.).

Diagnostic criteria

The normal range of serum TC, TG, HDL-C, LDL-C, ApoA1, ApoB levels, and the ApoA1/ApoB ratio was 3.10-5.17, 0.56-1.70, 0.91-1.81,

2.70-3.20 mmol/L, 1.00-1.76, 0.63-1.14 g/L, and 1.00-2.50; respectively. The subjects with TC > 5.17 mmol/L and/or TG > 1.70 mmol/L were defined as hyperlipidemia [5].

Statistical analyses

Quantitative variables were represented as mean ± standard deviation (serum TG levels

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Table 2. Serum lipid levels according to sex, BMI, blood pressure, drinking, smoking and age between the Jing and Mulao populations

| Parameter | n | TC (mmol/L) | TG (mmol/L) | HDL-C (mmol/L) | LDL-C (mmol/L) | ApoA1 (g/L) | ApoB (g/L) | ApoA1/ApoB |
|-------------------------------|------|--------------------------|-----------------------------|------------------------|---------------------------|---------------------------|---------------------------|---------------------------|
| Jing | | | | | | | | |
| Male | 678 | 5.08±0.80 | 1.50 (1.16) | 1.74±0.45 | 2.81±0.37 | 1.29±0.23 | 1.08±0.23 | 1.27±0.40 |
| Female | 576 | 5.23±0.98** | 1.40 (1.14) | 1.84±0.42*** | 2.86±0.48 | 1.31±0.24 | 1.07±0.26 | 1.29±0.34 |
| BMI ≤ 24 (kg/m ²) | 787 | 5.08±0.87 | 1.33 (1.06) | 1.87±0.44 | 2.81±0.41 | 1.33±0.21 | 1.03±0.24 | 1.36±0.39 |
| BMI > 24 (kg/m ²) | 467 | 5.26±0.91** | 1.66 (1.29)*** | 1.64±0.40*** | 2.88±0.46* | 1.26±0.27*** | 1.14±0.25*** | 1.15±0.31*** |
| Normotensive | 786 | 5.11±0.90 | 1.36 (1.08) | 1.81±0.47 | 2.82±0.44 | 1.31±0.24 | 1.06±0.24 | 1.30±0.39 |
| Hypertensive | 468 | 5.21±0.85 | 1.53 (1.22)*** | 1.74±0.39** | 2.87±0.40* | 1.29±0.22 | 1.09±0.25 | 1.25±0.34* |
| Nondrinker | 1053 | 5.13±0.92 | 1.44 (1.14) | 1.76±0.43 | 2.84±0.44 | 1.30±0.24 | 1.08±0.25 | 1.26±0.35 |
| Drinker | 201 | 5.26±0.71 | 1.49 (1.16) | 1.92±0.46*** | 2.84±0.34 | 1.34±0.22** | 1.06±0.25 | 1.36±0.45** |
| Nonsmoker | 1019 | 5.15±0.90 | 1.44 (1.12) | 1.79±0.41 | 2.84±0.44 | 1.30±0.23 | 1.08±0.25 | 1.27±0.34 |
| Smoker | 235 | 5.13±0.85 | 1.44 (1.17) | 1.78±0.55 | 2.83±0.38 | 1.30±0.23 | 1.06±0.23 | 1.32±0.50 |
| Age ≤ 40 year | 130 | 4.56±0.80 | 1.23 (1.00) | 1.63±0.40 | 2.58±0.41 | 1.24±0.19 | 0.99±0.25 | 1.35±0.46 |
| 40~year | 307 | 4.96±0.78 | 1.35 (1.10) | 1.78±0.45 | 2.79±0.40 | 1.29±0.20 | 1.05±0.22 | 1.29±0.36 |
| 50~year | 297 | 5.33±0.91 | 1.43 (1.14) | 1.81±0.43 | 2.93±0.46 | 1.32±0.24 | 1.10±0.24 | 1.24±0.30 |
| 60~year | 263 | 5.36±0.89 | 1.55 (1.19) | 1.82±0.43 | 2.92±0.39 | 1.30±0.21 | 1.07±0.23 | 1.27±0.37 |
| 70~year | 224 | 5.20±0.86 | 1.49 (1.21) | 1.80±0.47 | 2.83±0.40 | 1.35±0.33 | 1.10±0.27 | 1.30±0.42 |
| ≥ 80 year | 33 | 5.56±0.81 | 1.41 (1.29) | 1.83±0.43 | 2.96±0.34 | 1.28±0.24 | 1.12±0.27 | 1.12±0.27 |
| F for 6 age subgroups | - | 23.044 | 1.184 | 3.970 | 16.970 | 3.813 | 5.951 | 2.878 |
| P for 6 age subgroups | - | < 0.001 | 0.315 | < 0.001 | < 0.001 | 0.002 | < 0.001 | 0.014 |
| Mulao | | | | | | | | |
| Male | 680 | 5.31±1.34 ^c | 1.18 (0.84) ^c | 1.76±0.46 | 3.02±0.88 ^c | 1.38±0.42 ^c | 1.05±0.60 | 1.52±0.67 ^c |
| Female | 571 | 5.10±1.20** ^a | 1.08 (0.84)*** ^c | 1.77±0.42 ^c | 3.08±0.94 ^c | 1.34±0.37 | 0.95±0.47*** ^c | 1.60±0.72 ^c |
| BMI ≤ 24 (kg/m ²) | 929 | 5.13±1.06 | 1.06 (0.82) ^c | 1.87±0.44 | 3.00±0.85 ^c | 1.38±0.41 ^b | 0.96±0.48 ^c | 1.62±0.72 ^c |
| BMI > 24 (kg/m ²) | 322 | 5.44±1.77** | 1.45 (0.95)*** ^c | 1.61±0.39*** | 3.18±1.06** ^c | 1.31±0.37** ^a | 1.13±0.69*** | 1.38±0.57*** ^c |
| Normotensive | 717 | 5.17±1.32 | 1.05 (0.81) ^c | 1.78±0.42 | 3.02±0.85 ^c | 1.37±0.38 ^c | 0.96±0.48 ^c | 1.30±0.39 |
| Hypertensive | 534 | 5.26±1.23 | 1.27 (0.88)*** ^c | 1.74±0.39 | 3.09±0.99 ^c | 1.36±0.42 ^b | 1.05±0.62** | 1.50±0.69 ^c |
| Nondrinker | 897 | 5.15±1.15 | 1.12 (0.85) ^c | 1.73±0.41 ^a | 3.10±0.90 ^c | 1.32±0.37 | 1.00±0.54 ^c | 1.53±0.67 ^c |
| Drinker | 354 | 5.38±1.63** | 1.14 (0.82) ^c | 1.87±0.50*** | 2.90±0.94*** ^c | 1.47±0.44*** ^b | 1.02±0.56 ^c | 1.65±0.72*** ^c |
| Nonsmoker | 906 | 5.18±1.32 | 1.13 (0.84) ^c | 1.76±0.42 ^a | 3.06±0.91 ^c | 1.36±0.39 | 1.00±0.55 ^a | 1.56±0.71 ^c |
| Smoker | 345 | 5.29±1.20 | 1.10 (0.83) ^c | 1.78±0.55 | 3.01±0.91 | 1.38±0.41 | 1.00±0.53 | 1.57±0.63 ^c |
| Age ≤ 40 year | 141 | 5.13±1.23 ^c | 1.13 (0.82) | 1.77±0.47 ^b | 2.96±0.99 ^c | 1.43±0.36 ^c | 1.00±0.68 | 1.69±0.67 ^c |
| 40~year | 298 | 5.09±1.81 | 1.07 (0.78) ^c | 1.69±0.44 ^b | 2.92±1.00 ^a | 1.31±0.45 | 1.02±0.65 | 1.57±0.91 ^c |
| 50~year | 303 | 5.37±1.02 | 1.17 (0.83) ^c | 1.79±0.40 | 3.22±0.84 ^c | 1.41±0.35 ^c | 0.96±0.33 ^c | 1.59±0.57 ^c |
| 60~year | 251 | 5.26±1.08 | 1.11 (0.85) ^c | 1.82±0.48 | 3.03±0.85 | 1.38±0.40 ^b | 1.07±0.58 | 1.48±0.60 ^c |
| 70~year | 227 | 5.21±1.03 | 1.12 (0.88) ^c | 1.79±0.41 | 3.08±0.89 ^c | 1.33±0.39 | 0.99±0.51 ^b | 1.50±0.63 ^c |
| ≥ 80 year | 31 | 4.09±0.87 ^b | 0.94 (0.80) ^c | 1.67±0.56 | 2.78±0.64 | 0.86±0.27 | 0.86±0.27 ^c | 1.58±0.45 ^c |
| F for 6 age subgroups | - | 5.054 | 2.047 | 3.181 | 4.431 | 3.697 | 1.655 | 2.125 |
| P for 6 age subgroups | - | < 0.001 | 0.070 | 0.007 | < 0.001 | 0.003 | 0.143 | 0.060 |

TC, total cholesterol; TG, triglyceride; 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. *P < 0.05, **P < 0.01 and ***P < 0.001 in comparison with male, BMI ≤ 24 kg/m², normotensive, nondrinking, or nonsmoker of the same ethnic group; ^aP < 0.05, ^bP < 0.01 and ^cP < 0.001 in comparison with the same subgroup of Jing. The values of TG were presented as median (inter-quartile range).

were represented as medians and inter-quartile ranges). Comparisons of mean values of general characteristics between Jing and Mulao were performed by Student's unpaired *t*-test. Comparisons of serum lipid levels between the two ethnic groups or two subgroups were tested by analysis of covariance (ANCOVA). Sex, age, weight, body height, BMI, alcohol consumption, cigarette smoking, blood pressure,

and age were adjusted for the statistical analysis. Qualitative variables were expressed as percentages, the difference between the two ethnic groups was tested by the Chi-square test. The association of serum lipid levels and several environmental factors was tested by multivariable linear regression analysis. To evaluate the association of hyperlipidemia and several environmental factors, unconditional

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logistic regression analysis was also performed in a combined population of Jing and Mulao, Jing, and Mulao; respectively. A *P* value of less than 0.05 was considered statistically significant.

Results

Population characteristics

The general characteristics of Jing and Mulao are presented in **Table 1**. The levels of body height, weight, BMI, waist circumference and the intake of protein were higher in Jing than in Mulao (*P* < 0.001 for all), whereas the intensity of physical activity, the intakes of energy, carbohydrate, total fat, total dietary fiber, the levels of diastolic blood pressure and the prevalence of hypertension, and the rates of drinking and smoking were lower in Jing than in Mulao (*P* < 0.01 for all).

Serum lipid levels and the prevalence of dyslipidemia

The levels of LDL-C, ApoA1, and the ratio of ApoA1 to ApoB were lower but the levels of ApoB were higher in Jing than in Mulao (*P* < 0.001 for all). The abnormal rates of HDL-C, LDL-C and ApoA1 were lower and the abnormal rates of TG, ApoB and ApoA1/ApoB ratio were higher in Jing than in Mulao (*P* < 0.001 for all; **Table 1**). The prevalence of hypercholesterolemia, hypertriglyceridemia and hyperlipidemia between Jing and Mulao was 45.37% vs. 49.08% (*P* > 0.05), 32.38% vs. 24.38% (*P* < 0.001) and 60.53% vs. 57.47% (*P* > 0.05); respectively.

Sex, BMI, blood pressure, alcohol, smoking, age and serum lipid levels

For the Jing population, the levels of TC and HDL-C were higher in females than in males; TC, TG, LDL-C and ApoB were higher and HDL-C, ApoA1 and the ratio of ApoA1 to ApoB were lower in subjects with a BMI > 24 kg/m² than those with a BMI ≤ 24 kg/m²; TG and LDL-C were higher and HDL-C and the ratio of ApoA1 to ApoB were lower in hypertensives than in normotensives; and HDL-C, ApoA1 and the ratio of ApoA1 to ApoB were higher in drinkers than in nondrinkers (*P* < 0.05-0.001; **Table 2**).

For the Mulao population, the levels of TC, TG and ApoB were lower and the ratio of ApoA1 to

ApoB was higher in females than in males; TC, TG, LDL-C and ApoB were higher and HDL-C, ApoA1 and the ratio of ApoA1 to ApoB were lower in subjects with a BMI > 24 kg/m² than those with a BMI ≤ 24 kg/m²; TG, ApoB and the ratio of ApoA1 to ApoB were higher in hypertensives than in normotensives; and TC, HDL-C, ApoA1 and the ratio of ApoA1 to ApoB were higher and LDL-C was lower in drinkers than in nondrinkers (*P* < 0.05-0.001; **Table 2**).

The levels of TC (female and ≥ 80 year), TG (all subgroups but not ≤ 40 year), HDL-C (female, nondrinker, nonsmoker, and 40~ year) and ApoB (female, BMI ≤ 24 kg/m², normotensive, nondrinker, drinker, nonsmoker, 50~, 70~, and ≥ 80 year) were lower and the levels of TC (male and ≤ 40 year), HDL-C (≤ 40 year), LDL-C (all subgroups but not 60~ and ≥ 80 year), ApoA1 (male, BMI ≤ 24 kg/m², BMI > 24 kg/m², normotensive, hypertensive, drinker, ≤ 40, 50~, and 60~ year), and the ratio of ApoA1 to ApoB (all subgroups but not normotensive) were higher in Mulao than in Jing corresponding subgroups (*P* < 0.05-0.001; **Table 2**).

Risk factors for serum lipid parameters

The risk factors for serum lipid parameters between the two ethnic groups are listed in **Table 3**. Serum lipid parameters were correlated with sex, age, alcohol consumption, cigarette smoking, height, weight, BMI, waist circumference, and blood pressure in both ethnic groups (*P* < 0.05-0.001), but the risk factors were different between the Jing and Mulao populations.

Risk factors for hyperlipidemia

The prevalence of hyperlipidemia was associated with age, waist circumference, BMI, and the intakes of total energy and total dietary fiber in Jing, whereas it was correlated with age, sex, cigarette smoking, systolic blood pressure, and the intakes of total energy, total fat and total dietary fiber in Mulao (*P* < 0.05-0.001; **Table 4**).

Discussion

The present study showed that the serum lipid profiles and the prevalence of dyslipidemia were different between the Jing and Mulao populations. The levels of LDL-C, ApoA1, and the ratio of ApoA1 to ApoB were lower but the levels

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Table 3. Related risk factors for serum lipid parameters between the Jing and Mulao populations

| Lipid | Risk factor | Unstandardized coefficient | Std. error | Standardized coefficient | t | P |
|----------------|--------------------------|----------------------------|------------|--------------------------|--------|---------|
| Jing and Mulao | | | | | | |
| TC | Age | 0.007 | 0.002 | 0.087 | 4.200 | < 0.001 |
| | Alcohol consumption | 0.117 | 0.037 | 0.066 | 3.112 | 0.002 |
| | Weight | -0.011 | 0.004 | -0.102 | -2.663 | 0.008 |
| | Diastolic blood pressure | 0.005 | 0.002 | 0.047 | 2.300 | 0.022 |
| | Body mass index | 0.060 | 0.013 | 0.174 | 4.664 | < 0.001 |
| TG | Age | -0.010 | 0.003 | 0.082 | -3.716 | < 0.001 |
| | Height | -0.022 | 0.006 | -0.106 | -3.847 | < 0.001 |
| | Gender | -0.354 | 0.096 | -0.105 | -3.693 | < 0.001 |
| | Waist circumference | 0.041 | 0.004 | 0.227 | 10.818 | < 0.001 |
| | Systolic blood pressure | 0.005 | 0.002 | 0.062 | 2.933 | 0.003 |
| HDL-C | Alcohol consumption | 0.152 | 0.014 | 0.217 | 10.695 | < 0.001 |
| | Weight | -0.007 | 0.002 | -0.171 | -4.194 | < 0.001 |
| | Ethnic group | -0.094 | 0.017 | -0.107 | -5.434 | < 0.001 |
| | Gender | 0.096 | 0.023 | 0.109 | 4.161 | < 0.001 |
| | Height | 0.005 | 0.002 | 0.092 | 3.040 | < 0.001 |
| LDL-C | Waist circumference | -0.010 | 0.002 | -0.217 | -0.620 | < 0.001 |
| | Weight | -0.007 | 0.003 | -0.104 | -2.882 | 0.004 |
| | Age | 0.003 | 0.001 | 0.047 | 2.290 | 0.022 |
| | Ethnic group | 0.211 | 0.030 | 0.147 | 7.315 | < 0.001 |
| ApoA1 | Body mass index | 0.039 | 0.008 | 0.174 | 4.958 | < 0.001 |
| | Alcohol consumption | 0.080 | 0.010 | 0.152 | 7.768 | < 0.001 |
| | Systolic blood pressure | 0.001 | 0.000 | 0.093 | 3.612 | < 0.001 |
| | Diastolic blood pressure | -0.002 | 0.001 | -0.058 | -2.191 | 0.029 |
| | Waist circumference | -0.005 | 0.001 | -0.142 | -6.799 | < 0.001 |
| ApoB | Ethnic group | 0.040 | 0.013 | 0.060 | 2.974 | 0.003 |
| | Systolic blood pressure | 0.003 | 0.000 | 0.149 | 5.747 | < 0.001 |
| | Ethnic group | -0.035 | 0.017 | -0.042 | -2.045 | 0.041 |
| | Gender | -0.051 | 0.018 | -0.060 | -2.783 | 0.005 |
| | Waist circumference | 0.007 | 0.001 | 0.157 | 7.475 | < 0.001 |
| ApoA1/ApoB | Diastolic blood pressure | -0.003 | 0.001 | -0.079 | -2.556 | 0.011 |
| | Age | -0.003 | 0.001 | -0.068 | -3.330 | 0.001 |
| | Alcohol consumption | 0.105 | 0.019 | 0.115 | 5.624 | < 0.001 |
| | Ethnic group | 0.217 | 0.022 | 0.189 | 9.646 | < 0.001 |
| | Gender | 0.075 | 0.030 | 0.065 | 2.478 | 0.013 |
| | Height | 0.004 | 0.002 | 0.060 | 1.967 | 0.049 |
| | Weight | -0.005 | 0.002 | -0.093 | -2.270 | 0.023 |
| | Waist circumference | -0.011 | 0.002 | -0.178 | -5.213 | < 0.001 |
| Jing | | | | | | |
| TC | Gender | 0.277 | 0.056 | 0.155 | 4.972 | < 0.001 |
| | Age | 0.019 | 0.002 | 0.287 | 9.682 | < 0.001 |
| | Cigarette smoking | 0.061 | 0.035 | 0.076 | 2.441 | 0.015 |
| | Alcohol consumption | 0.106 | 0.034 | 0.091 | 3.138 | 0.002 |
| | Systolic blood pressure | -0.008 | 0.002 | -0.197 | -5.324 | < 0.001 |
| | Diastolic blood pressure | 0.017 | 0.003 | 0.202 | 5.830 | < 0.001 |
| | Body mass index | 0.047 | 0.014 | 0.169 | 3.321 | 0.001 |
| TG | Gender | -2.243 | 0.065 | -0.138 | -3.737 | < 0.001 |

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|------------|--------------------------|--------|-------|--------|---------|---------|
| | Age | -0.004 | 0.002 | -0.060 | -2.091 | 0.037 |
| | Cigarette smoking | 0.154 | 0.023 | 0.196 | 6.703 | < 0.001 |
| | Height | -0.033 | 0.005 | -0.293 | -7.208 | < 0.001 |
| | Waist circumference | 0.045 | 0.005 | 0.4482 | 8.819 | < 0.001 |
| | Diastolic blood pressure | 0.008 | 0.002 | 0.099 | 3.701- | < 0.001 |
| HDL-C | Gender | 0.145 | 0.024 | 0.164 | 6.109 | < 0.001 |
| | Age | 0.004 | 0.001 | 0.110 | 4.005 | < 0.001 |
| | Alcohol consumption | 0.145 | 0.015 | 0.251 | 9.409 | < 0.001 |
| | Waist circumference | -0.018 | 0.001 | -0.372 | -13.999 | < 0.001 |
| | Diastolic blood pressure | 0.004 | 0.001 | 0.090 | 2.756 | 0.006 |
| | Systolic blood pressure | -0.002 | 0.001 | -0.090 | 2.756 | 0.006 |
| LDL-C | Gender | 0.085 | 0.026 | 0.099 | 3.223 | 0.001 |
| | Age | 0.006 | 0.001 | 0.186 | 6.111 | < 0.001 |
| | Cigarette smoking | 0.031 | 0.012 | 0.082 | 2.590 | 0.010 |
| | Diastolic blood pressure | 0.005 | 0.001 | 0.134 | 3.790 | < 0.001 |
| ApoA1 | Alcohol consumption | 0.051 | 0.009 | 0.167 | 5.928 | < 0.001 |
| | Age | 0.001 | 0.001 | 0.064 | 2.256 | 0.024 |
| | Weight | -0.018 | 0.008 | -0.782 | -2.413 | 0.016 |
| ApoB | Age | 0.003 | 0.001 | 0.141 | 4.917 | < 0.001 |
| | Height | -0.004 | 0.001 | -0.117 | -2.873 | 0.004 |
| | Weight | 0.004 | 0.002 | 0.177 | 2.585 | 0.010 |
| | Waist circumference | 0.003 | 0.002 | 0.130 | 2.270 | 0.023 |
| ApoA1/ApoB | Age | -0.002 | 0.001 | -0.078 | -2.802 | 0.005 |
| | Alcohol consumption | 0.063 | 0.013 | 0.129 | -4.380 | < 0.001 |
| | Height | 0.004 | 0.002 | 0.091 | 2.234 | 0.026 |
| | Weight | -0.011 | 0.002 | -0.291 | -4.380 | < 0.001 |
| Mulao | | | | | | |
| TC | Gender | -0.165 | 0.074 | -0.064 | -2.233 | 0.026 |
| | Waist circumference | 0.016 | 0.004 | 0.107 | 3.649 | < 0.001 |
| | Systolic blood pressure | 0.006 | 0.002 | 0.115 | 2.957 | 0.003 |
| TG | Age | -0.011 | 0.005 | -0.066 | -2.173 | 0.030 |
| | Alcohol consumption | 0.725 | 0.144 | 0.149 | 5.022 | < 0.001 |
| | Height | -0.124 | 0.055 | -0.444 | -2.243 | 0.025 |
| | Weight | 0.176 | 0.079 | 0.760 | 2.229 | 0.026 |
| | Waist circumference | 0.043 | 0.011 | 0.173 | 4.068 | < 0.001 |
| | Systolic blood pressure | 0.009 | 0.004 | 0.099 | 2.648 | 0.014 |
| | Body mass index | -0.406 | 0.191 | -0.585 | -2.131 | 0.033 |
| HDL-C | Cigarette smoking | -0.023 | 0.011 | -0.065 | -2.117 | 0.034 |
| | Alcohol consumption | 0.205 | 0.031 | 0.208 | 6.675 | < 0.001 |
| | Height | 0.005 | 0.002 | 0.082 | 2.235 | 0.026 |
| | Weight | -0.011 | 0.002 | -0.241 | -4.891 | < 0.001 |
| | Waist circumference | -0.006 | 0.002 | -0.116 | -2.786 | 0.005 |
| LDL-C | Alcohol consumption | -2.222 | 0.056 | -0.110 | -3.939 | < 0.001 |
| | Body mass index | 0.040 | 0.008 | 0.137 | 4.845 | < 0.001 |
| ApoA1 | Age | -0.003 | 0.001 | -0.084 | -2.837 | 0.005 |
| | Alcohol consumption | 0.157 | 0.025 | 0.178 | 6.388 | < 0.001 |
| | Waist circumference | -0.005 | 0.001 | -0.116 | -4.067 | < 0.001 |
| | Systolic blood pressure | 0.002 | 0.001 | 0.144 | 3.577 | < 0.001 |
| | Diastolic blood pressure | -0.003 | 0.001 | -0.102 | -2.612 | 0.009 |

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|------------|--------------------------|--------|-------|--------|--------|---------|
| ApoB | Gender | -0.129 | 0.037 | -0.118 | -3.456 | 0.001 |
| | Age | -0.004 | 0.001 | -0.090 | -3.014 | 0.003 |
| | Alcohol consumption | -0.088 | 0.041 | -0.073 | -2.165 | 0.031 |
| | Waist circumference | 0.008 | 0.002 | 0.124 | 4.283 | < 0.001 |
| | Systolic blood pressure | 0.006 | 0.001 | 0.268 | 6.622 | < 0.001 |
| | Diastolic blood pressure | -0.008 | 0.002 | -0.177 | -4.545 | < 0.001 |
| ApoA1/ApoB | Gender | 0.140 | 0.047 | 0.101 | 2.982 | 0.003 |
| | Age | -0.004 | 0.001 | -0.076 | -2.724 | 0.007 |
| | Alcohol consumption | 0.233 | 0.051 | 0.152 | 4.555 | 0.001 |
| | Waist circumference | -0.016 | 0.002 | -0.205 | -7.320 | < 0.001 |

TC, total cholesterol; TG, triglyceride; 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.

Table 4. Related risk factors for hyperlipidemia between the Jing and Mulao populations

| Population | Risk factor | Regression coefficient | Standard error | Wald | P value | Odds ratio | 95% Confidence interval |
|----------------|--------------------------|------------------------|----------------|--------|---------|------------|-------------------------|
| Jing and Mulao | Age | 0.077 | 0.031 | 6.171 | 0.013 | 1.080 | 1.016-1.148 |
| | Waist circumference | 0.607 | 0.137 | 19.534 | < 0.001 | 1.834 | 1.402-2.400 |
| | Body mass index | 0.253 | 0.108 | 5.450 | 0.020 | 1.288 | 1.041-1.593 |
| | Alcohol consumption | 0.285 | 0.101 | 7.899 | 0.005 | 1.330 | 1.090-1.622 |
| | Diastolic blood pressure | 0.242 | 0.097 | 6.196 | 0.013 | 1.274 | 1.053-1.541 |
| | Total energy | 0.553 | 0.164 | 10.234 | 0.001 | 1.372 | 1.196-2.428 |
| | Total fat | 0.546 | 0.138 | 13.131 | < 0.001 | 1.571 | 1.137-2.346 |
| | Total dietary fiber | -0.244 | 0.103 | 5.418 | 0.021 | 1.278 | 1.055-1.671 |
| Jing | Age | 0.252 | 0.046 | 29.675 | < 0.001 | 1.286 | 1.175-1.408 |
| | Waist circumference | 0.783 | 0.187 | 17.585 | < 0.001 | 2.188 | 1.518-3.156 |
| | Body mass index | 0.321 | 0.158 | 4.103 | 0.043 | 1.378 | 1.010-1.880 |
| | Total energy | 0.431 | 0.193 | 7.842 | 0.005 | 1.713 | 1.138-1.998 |
| | Total dietary fiber | -0.258 | 0.086 | 6.222 | 0.011 | 1.435 | 1.042-1.976 |
| Mulao | Age | -0.120 | 0.045 | 7.067 | 0.008 | 0.887 | 0.811-0.969 |
| | Sex | -0.643 | 0.144 | 19.820 | < 0.001 | 0.526 | 0.396-0.698 |
| | Cigarette smoking | -0.430 | 0.161 | 7.170 | 0.007 | 0.651 | 0.475-0.891 |
| | Systolic blood pressure | 0.407 | 0.128 | 10.163 | 0.001 | 1.502 | 1.170-1.928 |
| | Total energy | 0.601 | 0.174 | 11.546 | < 0.001 | 1.868 | 1.047-2.353 |
| | Total fat | 0.564 | 0.191 | 9.468 | 0.002 | 1.637 | 1.206-2.273 |
| | Total dietary fiber | -0.286 | 0.110 | 6.459 | 0.009 | 1.715 | 1.148-2.336 |

Ethnic group (Jing = 0; Mulao = 1), gender (male = 0; female = 1), age (< 40 = 1; 40~ = 2; 50~ = 3; 60~ = 4; 70~ = 5; and > 80 = 6), BMI ($\leq 24 \text{ kg/m}^2 = 0$; $> 24 \text{ kg/m}^2 = 1$), waist circumference [$< 90 \text{ (male)}/85 \text{ (female) cm} = 0$; $\geq 90 \text{ (male)}/85 \text{ (female) cm} = 1$], systolic blood pressure ($< 140 \text{ mmHg} = 0$; $\geq 140 \text{ mmHg} = 1$), diastolic blood pressure ($< 90 \text{ mmHg} = 0$; $\geq 90 \text{ mmHg} = 1$), hypertension (normotensive = 0; hypertensive = 1), alcohol consumption (nondrinker = 0; drinker = 1), cigarette smoking (nonsmoker = 0; smoker = 1).

of ApoB were higher in Jing than in Mulao. The prevalence of hypertriglyceridemia, high ApoB and low ApoA1/ApoB ratio was higher and low HDL-C, high LDL-C and low ApoA1 was lower in Jing than in Mulao. Multivariable linear regression and unconditional logistic regression analyses revealed that the risk factors for serum

lipid parameters and hyperlipidemia were also different between the two ethnic groups. It is well recognized that dyslipidemia is a complex trait caused by multiple environmental and genetic factors and their interactions [5-7]. The differences in serum lipid profiles and the prevalence of dyslipidemia between the Jing and

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Mulao populations may result from different dietary habits, lifestyle and genetic background.

Jing nationality is the only oceanic ethnic minority in China. They make living by fishing, and the economic condition is relatively good. They mainly eat fish and shrimp and like to use fish sauce as a kind of seasoning which is one of the local characteristics and also known as the "catfish sauce". Marine fish contains a lot of polyunsaturated fatty acids (PUFAs) that are mainly omega-3 fatty acids which main ingredient is eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). A number of studies showed that omega-3 fatty acids can reduce the generation of TG in liver and increase the excretion of TG and lower serum TG levels. It also can lower TC, LDL-C levels and increase HDL-C levels [8]. In addition, numerous studies reported that omega-3 fatty acids could improve insulin resistance [9], hypertension and hyperglycemia [10]. It is benefit for people eating fish rich in PUFAs for long-term to reduce LDL-C levels while reducing sclerosis of arterial congee appearance to reduce the death of coronary heart disease, acute myocardial infarction [11]. American Heart Association recommends coronary heart disease patients to acquire omega-3 PUFAs 1 g daily, and people without coronary heart disease eat fish, especially fatty fish at least twice a week.

The great majority of the Mulao people live in the mountain area. Rice and corn are the staple food all year. With the improvement of their living standard and the change of lifestyle, the intake of animal (pig) fat was more in Mulao than in Jing nationalities. The people of Mulao nationality like to eat cold foods along with acidic and spicy dishes, so bean soy sauce and pickled vegetables are among their most popular dishes. Some research showed that soluble dietary fiber absorbed a large number of bile acid, preventing cholesterol into blood and increasing its excretion, thereby increasing the conversion rate of cholesterol to lower serum cholesterol levels [12]. They also like to use animal oil to cook foods and eat fat meat, animal offal, brain ridge and pith which contain abundant saturated fatty acid. For nearly 50 years it has been widely accepted that high-fat diets, particularly those that contain large quantities of saturated fatty acids, raise blood cholesterol

concentrations and predispose individuals to cardiovascular disease [13]. Moreover, these high calorie diets that contain high saturated fatty acid, high fat and high cholesterol may stimulate the synthesis of blood cholesterol and elevate blood lipid concentrations.

The relationship between obesity and dyslipidemia has been clearly documented. The typical dyslipidemia of obesity consists of increased TG and free fatty acids, decreased HDL-C with HDL dysfunction and normal or slightly increased LDL-C with increased small dense LDL [14]. The concentrations of serum ApoB are also often increased, partly due to the hepatic overproduction of ApoB containing lipoproteins [15]. In the present study, we showed that the mean values of BMI and waist circumference were higher in Jing than in Mulao, the levels of ApoB and the prevalence of hypertriglyceridemia were also higher in Jing than in Mulao. Hypertriglyceridemia in obesity may be the major cause of the other lipid abnormalities since it will lead to delayed clearance of the TG-rich lipoproteins [16] and formation of small dense LDL [16, 17]. Lipolysis of TG-rich lipoproteins is further impaired in obesity by reduced mRNA expression levels of lipoprotein lipase (LPL) in adipose tissue [18] and reduced LPL activity in skeletal muscle [19]. Hypertriglyceridemia further induces an increased exchange of cholesterol esters and TG between VLDL and HDL and LDL by cholesterylester-transfer-protein. In addition, hepatic lipase removes TG and phospholipids from LDL for the final formation of TG-depleted small dense LDL.

Several observational studies and meta-analyses have consistently shown that moderate drinking when taken on a regular basis can increase TG, HDL-C and lower LDL-C levels, and protect against cardiovascular disease and death [20], but heavy drinking could reduce HDL-C level and constitute a severe risk condition [21]. In the current study, we showed that the percentages of subjects who consumed alcohol were higher in Mulao than in Jing, but the people of Mulao like to drink their homemade "Chongyang wine", a kind of glutinous rice wine. Its alcohol content is low, but rich in a variety of amino acids, glucose, vitamins and minerals. In addition, there are many wild grapes, *V. quinquangularis* (*Vitis louchengensis* W.T. Wang, ined), these fruits are used to make

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wine by local people. Conversely, the people of Jing drink rice wine on the market, with a higher content of alcohol. The effect of different kinds of alcoholic beverage on the lipid profiles is not well known, but some investigators have suggested that certain types of alcohol, e.g., wine, may be more protective than others. Ruidavets et al. [22] found that wine was positively associated with HDL-C, and beer was positively associated with HDL-C in men and with TG in men and women. Choudhury et al. [23] also showed serum TG levels were significantly lower in those who drank beer. The effects of alcohol intake on LDL-C appear to vary by specific patient types or patterns of alcohol intake, and perhaps by population and sex. A study in older Italian subjects (65-84 years old) found that alcohol intake increased serum LDL-C levels [24]. Another study in Turks also found increases in LDL-C, as well as in ApoB and TG, with alcohol in men, while women decreased TG and no change in LDL-C or ApoB with alcohol [25].

Some studies have proven that cigarette smoking has negative influences on the lipid profiles, but the results are inconsistent. Rao et al. [26] showed that cigarettes smoking raised LDL-C and TC levels but lowered HDL-C levels. In the current study, we showed that the percentages of subjects who smoked cigarettes were higher in Mulao than in Jing, but there were no significant differences in serum lipid parameters between smokers and nonsmokers in the both ethnic groups. These results suggested that cigarette smoking may not be independent risk factors for serum lipid levels in these populations.

Epidemiological studies have provided abundant evidence that plasma/serum lipid levels are closely related to sex and age [27]. Our study also showed that sex and age distribution could influence serum lipid levels in both ethnic groups. The levels of TC and HDL-C in Jing, and LDL-C and the ratio of ApoA1 to ApoB in Mulao were higher in females than in males. The levels of TC, TG and ApoB in Mulao were lower in females than in males. Sex differences in serum lipid profiles might result from different general characteristics such as BMI, waist circumference, alcohol consumption, dietary habits, exercise, and sex hormones. A meta-analysis demonstrated that testosterone decreased

TC, TG, and HDL-C levels, but increased LDL-C levels [28]. Estrogen is negatively correlated with serum TC, TG, and LDL-C levels, and positively correlated with HDL-C levels. Postmenopausal estrogen levels drop and subsequent changes in serum lipid levels could lead to cardiovascular events rising [29].

Both dyslipidemia and hypertension are the components of the metabolic syndrome. Blood pressure and plasma/serum lipid levels have been consistently related in several previous studies [30]. In the present study, we showed that the levels of TG and LDL-C in Jing were higher and HDL-C and the ratio of ApoA1 to ApoB were lower; and the levels of TG, ApoB and the ratio of ApoA1 to ApoB in Mulao were higher in hypertensives than in normotensives. These findings suggest that dyslipidemia and hypertension may have many common risk factors.

Serum lipid levels are also influenced by genetic factors [5, 7]. Several genetic polymorphisms were related to serum lipid levels [5, 7]. The genetic background of Jing and Mulao is different. Therefore, we guess that some lipid-related genetic polymorphisms may be different between the two ethnic groups, but it still needs to be determined.

Study limitations

There are several limitations in this study. First, the sample size is small. Second, a 24 h dietary recall may be inaccurate when diets are highly variable. Third, it is well known that both estrogen and menopause can influence serum lipid levels. In the present study, however, we did not distinguish the premenopausal and postmenopausal women in the both ethnic groups because of the relatively small samples. Finally, it is clearly established that serum lipid levels are regulated by multiple environmental and genetic factors, and their interactions [5-7]. Although we have detected the effect of several risk factors on serum lipid levels in this study, there are still many unmeasured environmental and genetic factors and their interactions.

Conclusions

Serum lipid profiles, the prevalence of dyslipidemia, and their risk factors were different between the Jing and Mulao populations. These

differences between the two minorities may result from the combined effects of different diets, lifestyle as well as genetic background.

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Disclosure of conflict of interest

None.

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