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ORIGINAL RESEARCH

Plasma Fatty Acids, Not Dietary Fatty Acids, Associated with Obesity in Four Ethnic Minority Groups Unique to Southwest China: A Cross-Sectional Study

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Background: Dietary fatty acids (DFAs) and plasma fatty acids (PFAs) are linked to obesity. However, whether this association exists among ethnic minorities remains lacking. The present cross-sectional study was designed to investigate the correlation between DFAs, PFAs and obesity in four ethnic minority groups to Southwest China.

Methods: A total of 166 obese people, and 166 normal-BMI subjects matched based on their age-, sex-, and ethnicity- were recruited from four different ethnic minority groups. DFAs were obtained through food frequency questionnaires. PFAs were assayed by GC/MS method. Binary and multiple regression analyses were performed to evaluate the correlation among DFAs, PFAs and obesity. Canonical correlation analysis (CCA) was conducted to assess the relationship between DFAs and PFAs.

Results: FAs were found to be highest in the Naxi people and lowest in the Hani people. Multiple logistic regression analysis revealed that plasma C16:0 (OR = 1.310; 95% CI, 1.028–1.669) in the Hani people; plasma C20:3 n-6 (OR = 6.250; 95% CI, 1.224–31.927) and dietary C20:1 (OR = 9.231; 95% CI, 1.253–68.016) in the Wa people; plasma C18:0 (OR = 0.788; 95% CI, 0.681–0.912) in the Naxi people were seen to be independent predictive factors for obesity. CCA showed that DFAs were positively correlated with PFAs in the Naxi (r: 0.676; $P < 0.05$) and Bulang people (r: 0.897; $P < 0.05$), but there was no correlation in the Hani and Wa people.

Conclusion: In this study, PFAs but not DFAs were independently associated with obesity, and different among the four ethnic minorities.

Keywords: dietary fatty acids, plasma fatty acids, obesity, minorities, canonical correlation analysis

Introduction

Obesity has been defined as a "global public health crisis".[1] In 2020, a global meta-analysis data for 13.2 million subjects showed that the prevalence of central obesity reached 41.5%.[2] The prevalence of obesity will continue to rise, especially in developing countries. Judging from the changes in obesity among Chinese adults from 2000 to 2018, the prevalence of obesity has increased significantly, and even faster in rural areas.[3] Obesity has become a serious public health problem in China. Obesity and related chronic diseases not only damage the physical and psychological health of individuals but also challenge the public health and medical system of the country and society.[4]

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Accumulating etiology studies have indicated that obesity is an imbalance between energy intake and consumption resulting from a synergy of dietary, environmental, lifestyle, and genetic factors.[5–8] High-fat dietary intake is linked to increased risk of obesity. Dietary fatty acids (DFAs), the important part of the daily dietary fat, play a vital role in obesity,[9] in the case of excessive intake or inappropriate proportions.[10–12] Depending on the molecular structure, some DFAs show metabolic benefits, such as polyunsaturated fatty acids (PUFAs).[12–14] Accordingly, increasing dietary PUFAs intake was recommended by the American Heart Association.[15] However, this was soon overturned by Heileson JL.[16] It can be seen that there is controversy about the influence of DFAs on obesity, which may be caused by traditional dietary nutrition survey methods and intestinal absorption.[17,18] Although fatty acids (FAs) are traditionally considered to be a dietary factor, they have been demonstrated to be present in plasma. Plasma fatty acids (PFAs) have also been used in epidemiological studies and have been considered to be related to obesity.[19] Understanding the association among DFAs, PFAs and obesity is more meaningful for formulating dietary patterns to prevent obesity.

China is a multi-ethnic country with 55 ethnic minority groups, and Yunnan is a region in Southwest China that contains 24 of them. Most ethnic minorities have their unique dietary customs, social settlements, lifestyle, and genetic factors. The obesity epidemic is also different. The unique characteristics of DFAs and PFAs may be related to the obesity epidemic differences among ethnic minorities, but the evidence on this relationship remains lacking. The present study was designed to assess the correlation between DFAs, PFAs and obesity risk among four unique ethnic minority groups in Yunnan.

Materials and Methods

Study Participants

Considering the population gathering of minorities, the crowd cooperation and other factors, three prefectures in Yunnan Province was selected. One county was randomly selected from every prefecture (Lvchun County in Honghe Prefecture for the Hani people, Yulong County in Lijiang City for the Naxi people, Shuangjiang County of Lincang City for the Bulang and Wa people) as the site investigation points. In order to achieve 80% confidence, the minimum sample size for each ethnic group was calculated to be 309 ($n = C^2\sigma^2/p^2$; C = 1.96, P = 2, σ = 17.95). In order to enhance the test efficiency, the minimum sample size was increased by 50%, and the final sample size of each ethnic group was 464 people. As Figure 1 shows, 1913 residents (488 Hani people, 464 Bulang people, 488 Wa people and 473 Naxi people) who were ≥18 years old, without mental illness, completed the survey and obtained biological samples. All participants provided written informed consent. The study complies with the Declaration of Helsinki and was approved by the Ethical Review Board of the Kunming Medical University.

Of these 1913 residents, 166 individuals (22 Hani people, 30 Bulang people, 35 Wa people and 79 Naxi people) were obese, all of them were assigned as the obese group (OB). A further 166 subjects with normal body mass index (BMI) matched by age, sex, and ethnicity were recruited and assigned to the normal-BMI group (NB). OB was defined as BMI (weight/height^2) ≥28.0 kg/m^2, and NB was defined as 18.5 ≤ BMI < 24.0 kg/m^2.[20]

Basic Characteristics Collection

A questionnaire was distributed to the participants to gather data including clinical characteristics, sociodemographic details and lifestyle. The questionnaire information was collected by a face-to-face interview implemented by well-trained interviewers. Physical activity was measured as described as follows: Physical activity in the last 7 days was investigated. According to Ainsworth's summary of physical activity, all activities were weighted and converted into moderate physical activity time, and were divided into three levels: high (≥2 hours/week), medium (1–2 hours/week) and low (≤1 hour/week).

Physical Examination

For each subject, anthropometric indicators and blood pressure (BP) were measured by standard methods. Body weight and height were measured using standardized protocols with ultrasonic body fat analyzer (model: EF08). BP was measured using a standardized protocol with blood pressure monitor (OMRON, model: U30) and an appropriately

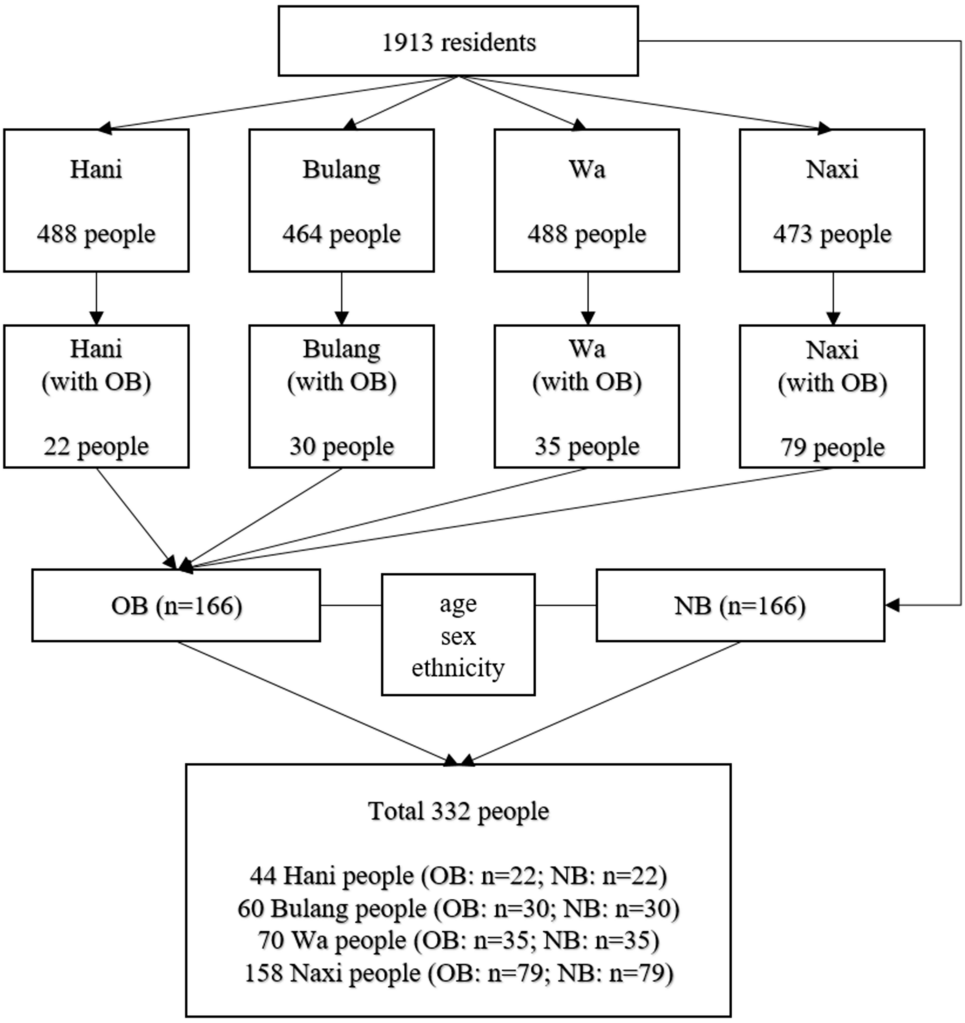

Figure 1 Inclusion process of enrolled people.
Abbreviations: OB, obesity group (BMI ≥28.0 kg/m^2); NB, normal-BMI group (18.5≤ BMI <24.0 kg/m^2).

sized cuff on the right arm after rested in a seated position for at least 10 minutes. Each BP reading was taken at 1-min intervals. The average of three readings was used for analysis.

After an overnight fast, fasting blood samples were collected after participants rested for 10 minutes in the seated position. After centrifugation at 3000 r/min for 10 minutes, each blood sample was divided into 2 1.5 mL EP tubes. All plasma samples were stored at −80 °C without freeze–thaw cycles until assay.

Fasting plasma glucose (FPG), triglycerides (TG), total cholesterol (TC), high-density lipoprotein (HDL-C), and low-density lipoprotein (LDL-C) were assayed by a biochemical autoanalyzer (Beckman Coulter AU480 Biochemical Autoanalyser, USA).

Semi-Quantitative Assessment of Dietary Fatty Acid Intake

A semi-quantified food frequency method was used to assess food intake. Based on "Chinese Food Composition 2009" (Second Edition),[21] the daily intake of total fatty acids (FAs), saturated fatty acids (SFAs), monounsaturated fatty acids (MUFAs) polyunsaturated fatty acids (PUFAs) and seventeen dietary fatty acids [lauric acid (C12:0), myristic acid (C14:0), palmitic acid (C16:0), margaric acid (C17:0), stearic acid (C18:0), arachidic acid (C20:0), docosanoic acid (C22:0), palmitoleic acid (C16:1), heptadecenoic acid (C17:1), oleic acid (C18:1), eicosaenoic acid (C20:1), erucic acid (C22:1), hexadecadienoic acid (C16:2), linoleic acid (C18:2), linolenic acid (C18:3), docosadienoic acid (C20:2), arachidonic acid (C20:4)] were calculated.

The Determine of Plasma Fatty Acids by GC/MS

According the correlation coefficient range of the standard curve (0.9991 to 0.9998), detection limit range (0.01–0.41 mg/L), intraday CV value (0.2–6.15%), daytime CV value (0.91–8.70%), recovery rate (80.06–118.34%), the modified conditions were selected for PFA methylesteration: 0.4 M KOH, 7% sulphuric acid, 65 °C and 20 minutes.[22]

The levels of thirty-seven plasma fatty acids [butyric acid (C4:0), caproic acid (C6:0), octanoic acid (C8:0), decanoic acid (C10:0), hendecanoic acid (C11:0), lauric acid (C12:0), tridecanoic acid (C13:0), myristoleic acid (C14:1 n-5), 10c-pentadecanoic acid (C15:1 n-5), 10-heptadecenoic acid (C17:1 n-7), trans linoleic acid (C18:2 n-6t), n-heneicosanoic acid (C21:0), 13,16-docosadienoic acid (C22:2 n-6), 4,7,10,13,16,19-docosahexaenoic acid (C22:6 n-3), tricosanoic acid (C23:0), Tetracosanoic acid (C24:0), nervonic acid (C24:1 n-9), myristic acid (C14:0), pentadecanoic acid (C15:0), palmitic acid (C16:0), margaric acid (C17:0), stearic acid (C18:0), arachidic acid (C20:0), docosanoic acid (C22:0), palmitoleic acid (C16:1 n-7), trans oleic acid (C18:1 n-9t), oleic acid (C18:1 n-9c), gondoic acid (C20:1 n-9), erucic acid (C22:1), linoleic acid (C18:2 n-6c), γ-linolenic acid (C18:3 n-6), α-linolenic acid (C18:3 n-3), 11,14-eicosadienoic acid (C20:2 n-6), dihomo-gamma-linolenic acid (C20:3 n-6), 11,14,17-eicosatrienoic acid (C20:3 n-3), arachidonic acid (C20:4 n-6), 5,8,11,14,17-eicosapentaenoic acid (C20:5 n-3)] were detected by gas chromatography/mass spectrometry (GC/MS) method. Chromatographic analysis was performed with a gas chromatography/mass spectrometer (GCMS-QP2010, SHIMADZU, Japan) equipped with a fused-silica (100 m × 0.2 mm ID, 0.25 μm flm) capillary column (RT-2560, SHIMADZU). The following conditions were applied for gas chromatograph: oven temperature was held at 60 °C for 5 minutes, programmed from 60 to 165 °C at a rate of 5 °C/min and held at 165 °C for 1 minute; 165 to 225 °C at a rate of 2 °C/min and held at 225 °C for 27 minutes. Run time was 84 minutes. Helium was used as the carrier gas at a flow rate of 1.3 mL/min. Flame ionization detector temperature was 60 °C, and injector temperature was 240 °C. The split ratio was set at 10:1 and 1 μL sample was injected. The following conditions were applied for mass spectrometry: ionization method was EI. Ion source temperature was 230 °C, and interface temperature was 250 °C. Solvent delay time was 3 minutes. Peak identification was based on relative retention times of 2 external standards. The area of each fatty acid peak was recorded, and PFA levels were calculated using the internal standard's area under the peak. Eventually, twenty plasma fatty acids were measured and analyzed.[22]

Statistical Analysis

The data management and statistical analyses were performed using IBM SPSS 26.0 statistical software (SPSS Inc., Chicago, USA). Data with normal distributions were expressed as the mean (standard deviation), and data with skewed distributions were expressed as the median (interquartile range). Group comparisons of anthropometric and biochemical variables were conducted by using paired-samples *t*-test or Wilcoxon signed rank test for continuous variables and chi-square test tests for categorical variables (ie, the confidence interval for a certain confidence level of the mean when the variance of the population is unknown. The *Z* is the standard normal distribution, which is the confidence interval for a certain confidence level of the mean given the population variance). Conditional logistic regression analysis was used to examine the association [odds ratio (OR)] of dietary fatty acids and plasma fatty acids with obesity. Potential confounding variables included education level, physical activity, occupation, marital status, annual family income, smoking, alcohol drinking, systolic blood pressure (SBP), diastolic blood pressure (DBP), FPG, TC, TG, LDL-C and HDL-C. In each ethnic group, compared between OB and NB group, the variables with statistically significant differences were controlled in the regression models. Relationships between dietary fatty acids set (seventeen DFA variables: C12:0, C14:0, C16:0, C17:0, C18:0, C20:0, C22:0, C16:1, C17:1, C18:1, C20:1, C22:1, C16:2, C18:2, C18:3, C20:2, C20:4) and plasma fatty acids set (twenty PFA variables: C14:0, C15:0, C16:0, C17:0, C18:0, C20:0, C22:0, C16:1 n-7, C18:1 n-9t, C18:1 n-9c, C20:1 n-9, C22:1, C18:2 n-6c, C18:3 n-6, C18:3 n-3, C20:2 n-6, C20:3 n-6, C20:3 n-3, C20:4 n-6, C20:5 n-3) were estimated using canonical correlation analysis (CCA). For all analyses, a P value < 0.05 was considered statistically significant. To evaluate the statistical power of this study, we performed a post hoc power analysis after data analysis.

Results

Baseline Characteristics of the Participants

A total of 332 individuals were surveyed, including 112 men (33.7%) and 220 women (66.3%). Median (interquartile range) age was 49 (40 to 58) years. The rate of obesity was 4.5% in the Hani people (95% CI: 2.7–6.3%), 6.5% in the Bulang people (95% CI: 3.7–8.1%), 7.2% in the Wa people (95% CI: 4.9–9.5%) and 16.7% (95% CI: 13.9–20.7%) in the Naxi people. The Bulang people have a similar lifestyle to the Wa people, but the Bulang people are more physically active. The Naxi people showed the highest level of education but the lowest level of physical activity. Basic clinical characteristics of participants in four minorities were shown in (Table 1).

Seventeen DFAs daily intake were calculated according "Chinese Food Composition 2009". Three DFAs (C22:0, C20:1, C22:1) intake were markedly higher, and dietary C16:1 intake was markedly lower in the Naxi people than in other three minorities ($P < 0.05$). The Hani people had significantly lowest intake of seven DFAs (C12:0, C17:0, C20:0, C22:0, C18:2, C18:3, C20:2) ($P < 0.05$) in four minorities. And, 82.4% DFAs showed no differences between the Bulang and the Wa people ($P > 0.05$). The comparison of DFAs among four minorities is shown in Figure 2A and B.

Twenty PFAs were detected by GC/MS. Compared among four minorities, fifteen PFAs (C15:0, C16:0, C17:0, C18:0, C20:0, C22:0, C18:1 n-9t, C20:1 n-9, C22:1, C18:2 n-6c, C18:3 n-6, C18:3 n-3, C20:2 n-6, C20:3 n-3, C20:5 n-3) showed statistically significant differences ($P < 0.05$). The ten PFAs (C20:0, C22:0, C18:1 n-9t, C20:1 n-9, C22:1, C18:2 n-6c, C18:3 n-6, C18:3 n-3, C20:3 n-3, C20:5 n-3) levels were markedly higher, and plasma C16:0 level was markedly lower in the Naxi people than in other three minorities ($P < 0.05$). Compared with other three minorities, the Hani people had significantly lower levels of three PFAs (C20:0, C20:1 n-9, C20:3 n-3, C20:5 n-3) and higher levels of two PFAs (C15:0, C17:0) ($P < 0.05$). Thirteen PFAs (C15:0, C16:0, C17:0, C20:0, C22:0, C18:1 n-9t, C20:1 n-9, C22:1, C18:2 n-6c, C18:3 n-6, C18:3 n-3, C20:3 n-3, C20:5 n-3) levers showed statistically significant differences between Hani people and Naxi people ($P < 0.05$). And 75.0% PFAs showed no differences between the Bulang and the Wa people ($P > 0.05$). The comparison of PFAs among four minorities is shown in Figure 2C and D.

The Differences in the Dietary Fatty Acids and Plasma Fatty Acids Among Four Minorities

As Table 2 showed, dietary levels of C20:1 ($Z = -2.285$; $P < 0.05$), C22:1 ($Z = -2.261$; $P < 0.05$) in the Wa people; C20:0 ($Z = -2.204$; $P < 0.05$), C20:2 ($Z = -1.980$; $P < 0.05$) in the Naxi people, were significantly higher in OB group compared

Table 1 Baseline Characteristics of Participants

Clinical Features	Hani (n = 44)	Bulang (n = 60)	Wa (n= 70)	Naxi (n = 158)
Age, years	50 (39, 58)	43 (30, 53)	46 (34, 54)	51 (45, 60)[b,c]
Male gender, n (%)	20 (45.4)	24 (40.0)	20 (28.6)[a,b]	48 (30.4)[a,b]
Low education level, n (%)	27 (61.4)	32 (53.3)	42 (60.0)	52 (32.9)[a,b,c]
Low physical activity, n (%)	7 (15.9)	2 (3.3)[a]	8 (11.4)[b]	32 (20.3)[b,c]
Peasantry, n (%)	37 (84.1)	57 (95.0)[a]	67 (94.3)[a]	147 (93.0)[a]
Smoking, n (%)	16 (36.4)	18 (30.0)[a]	19 (27.1)[a]	30 (19.0)[a,b,c]
Alcohol use, n (%)	22 (50.0)	31 (51.7)	29 (41.4)	30 (19.0)[a,b,c]
SBP, mm Hg	118.0 (110.0, 133.3)	119.7 (110.0, 136.5)*	122.7 (111.3, 132.0)	125.5 (118.3, 139.2)*
DBP, mm Hg	80.0 (75.3, 87.8)	80.0 (73.7, 85.6)*	80.0 (74.7, 85.6)	82.5 (75.9, 91.3)*
FPG, mmol/L	4.9 (4.4, 5.4)	4.7 (4.0, 5.3)	4.6 (4.2, 5.3)*	4.7 (4.2, 5.4)*
Plasma TG, mmol/L	1.4 (1.0, 1.9)	1.8 (1.2, 3.3)*,[a]	2.7 (1.6, 4.1)*,[a]	1.9 (1.5, 2.4)[a,c]
Plasma TC, mmol/L	4.8 (3.8, 5.5)	5.2 (4.4, 6.0)*,[a]	5.3 (4.7, 6.0)[a]	5.0 (4.2, 5.3)[c]
Plasma LDL-C, mmol/L	2.6 (2.1, 3.1)*	3.2 (2.6, 3.7)[a]	3.1 (2.6, 3.6)[a]	2.7 (2.1, 3.2)[b,c]
Plasma HDL-C, mmol/L	1.3 (1.1, 1.5)	1.3 (1.1, 1.5)	1.3 (1.1, 1.5)*	1.3 (1.2, 1.5)

Notes: Total n = 332. Data are presented as mean (standard deviation) for parametric variables; median (interquartile range) for non-parametric variables.*Compared with the NB group of the same minority, the difference was statistically significant. [a]Compared with Hani, the difference was statistically significant; [b]Compared with Bulang, the difference was statistically significant; [c]Compared with Wa, the difference was statistically significant.

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; FPG, fasting plasma glucose; TG, triglycerides, TC, total cholesterol; LDL-C, low-density lipoprotein; HDL-C, high-density lipoprotein.

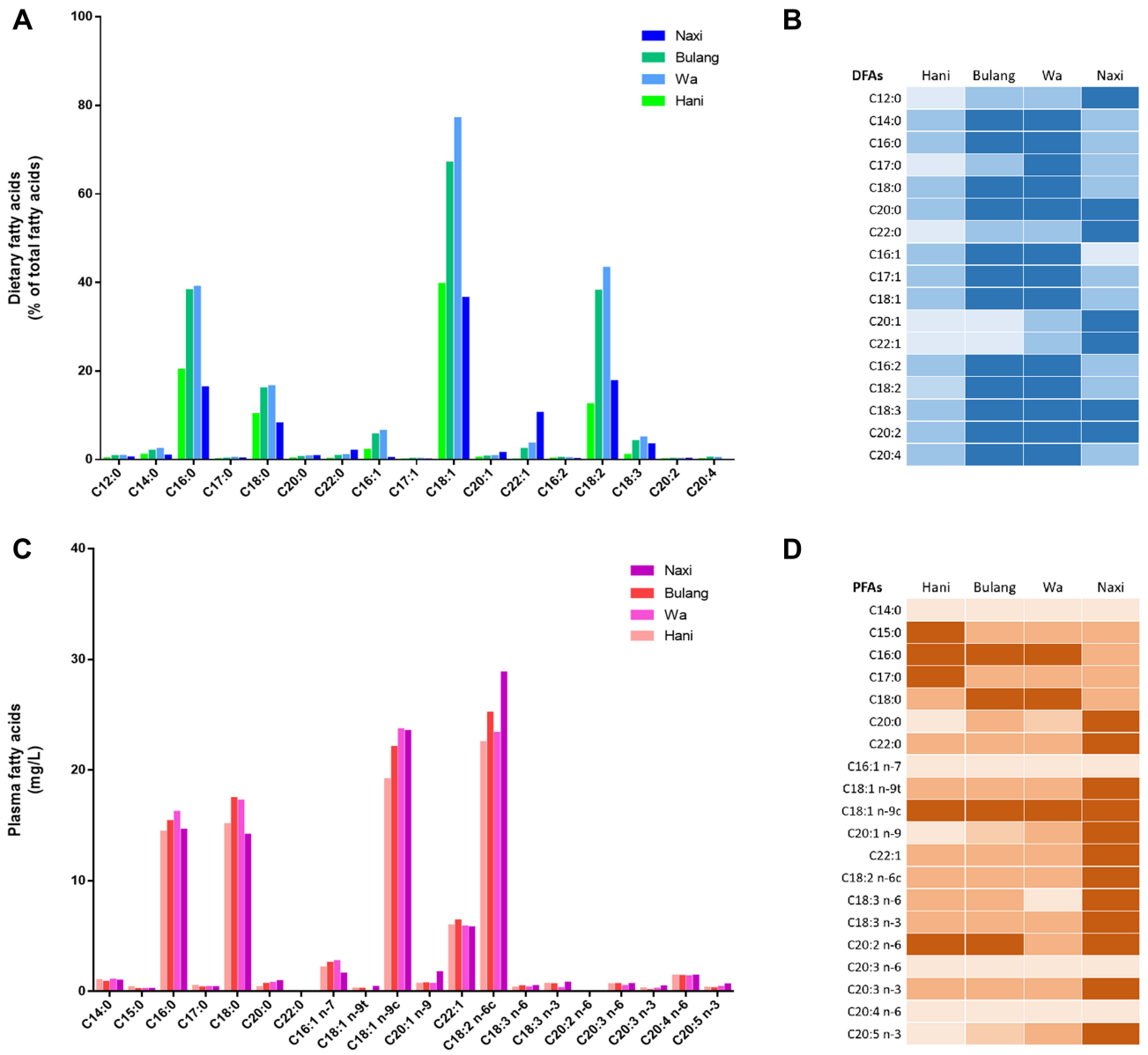

Figure 2 Comparison of fatty acids among four minorities.
Notes: (**A** and **B**), comparison of dietary fatty acids among four minorities; (**C** and **D**), comparison of plasma fatty acids among four minorities. (**B** and **D**), in the same fatty acid comparison, the darker the color, the higher the content; and the different color, the difference between the groups was statistically significant; the same color indicates no significant difference between groups. P value < 0.05 was considered statistically significant.

with NB group. Dietary levels of C16:1 ($Z = -2.607$; $P < 0.05$) and C17:1 ($Z = -3.147$; $P < 0.05$) were significantly higher in NB group compared with OB group in the Naxi people. There was no statistically significant difference in dietary fatty acids between OB and NB groups in the Hani and Bulang people ($P > 0.05$).

As Table 3 showed, among the twenty measured plasma fatty acids, Plasma levels of C16:0 ($t = -2.086$; $P < 0.05$) and C18:2 n-6c ($Z = -2.169$; $P < 0.05$) in the Hani people; C16:0 ($Z = -2.088$; $P < 0.05$), C16:1 n-7 ($Z = -2.417$; $P < 0.05$), C18:1 n-9c ($Z = -2.705$; $P < 0.05$), C20:3 n-6 ($t = -3.115$; $P < 0.05$) and C20:5 n-3 ($t = -4.107$; $P < 0.05$) in the Bulang people; C14:0 ($t = -2.293$; $P < 0.05$), C16:0 ($Z = -2.162$; $P < 0.05$), C16:1 n-7 ($t = -2.154$; $P < 0.05$), C18:1 n-9c ($Z = -2.178$; $P < 0.05$), C20:3 n-6 ($Z = -2.821$; $P < 0.05$) and C20:5 n-3 ($t = -3.265$; $P < 0.05$) in the Wa people; C14:0 ($t = -2.575$; $P < 0.05$), C17:0 ($Z = -2.189$; $P < 0.05$), C18:2 n-6c ($Z = -3.094$; $P < 0.05$), C18:3 n-6 ($t = -2.102$; $P < 0.05$), C18:3 n-3 ($Z = -2.835$; $P < 0.05$), C20:3 n-6 ($t = -5.360$; $P < 0.05$), C20:4 n-6 ($t = -2.537$; $P < 0.05$) and C20:5 n-3 ($t =$

Table 2 Dietary Fatty Acids of four Minorities (% of Total Fatty Acids)

DFAs	Hani People (n = 44)		Bulang People (n = 60)		Wa People (n = 70)		Naxi People (n = 158)	
	NB (n = 22)	OB (n = 22)	NB (n = 30)	OB (n = 30)	NB (n = 35)	OB (n = 35)	NB (n = 79)	OB (n = 79)
C12:0	0.3 (0.3)	0.4 (0.3)	0.8 (0.4, 1.6)	0.8 (0.5, 1.3)	1.0 (0.5, 1.7)	0.9 (0.4, 2.1)	0.5 (0.3, 0.8)	0.5 (0.3, 1.3)
C14:0	1.1 (0.8)	1.3 (0.5)	1.9 (1.3, 4.5)	2.3 (1.6, 3.7)	2.3 (1.5, 3.8)	2.5 (1.4, 4.6)	1.0 (0.7, 1.7)	0.9 (0.6, 1.4)
C16:0	18.8 (10.0, 24.9)	22.8 (17.0, 29.7)	35.0 (18.4, 62.2)	41.7 (30.5, 57.1)	38.8 (29.5, 63.8)	39.3 (24.0, 72.2)	16.5 (12.8, 27.5)	15.9 (10.7, 21.3)
C17:0	0.2 (0.1)	0.2 (0.2)	0.3 (0.1, 0.7)	0.2 (0.1, 0.5)	0.5 (0.3, 0.9)	0.4 (0.2, 1.0)	0.3 (0.2, 0.5)	0.3 (0.2, 0.6)
C18:0	10.0 (5.2, 13.0)	11.3 (8.4, 14.8)	15.4 (8.1, 28.7)	16.4 (13.4, 26.0)	16.7 (10.0, 25.9)	16.1 (10.9, 22.9)	8.4 (6.5, 14.5)	8.0 (5.3, 11.5)
C20:0	0.4 (0.4)	0.4 (0.2)	0.8 (0.7)	1.0 (0.9)	0.8 (0.4, 1.0)	0.7 (0.5, 1.2)	0.7 (0.5, 1.0)	0.9 (0.6, 1.6)*
C22:0	0.5 (0.8)	0.5 (0.7)	1.1 (1.1)	1.5 (1.5)	0.8 (0.5, 1.7)	1.3 (0.6, 1.7)	1.9 (1.2, 2.9)	2.3 (1.7, 3.2)
C16:1	2.4 (1.5)	2.8 (1.2)	7.9 (8.0)	7.3 (6.9)	6.6 (4.2, 9.6)	6.4 (3.7, 11.3)	0.5 (0.3, 0.8)	0.3 (0.2, 0.6)*
C17:1	0.1 (0.0)	0.1 (0.0)	0.2 (0.1, 0.7)	0.1 (0.1, 0.4)	0.2 (0.1, 0.4)	0.3 (0.2, 0.6)	0.1 (0.0, 0.1)	0.0 (0.0, 0.1)*
C18:1	35.3 (17.8, 48.2)	43.1 (30.6, 57.6)	60.4 (36.3, 111.2)	70.8 (56.5, 107.8)	78.3 (47.2, 112.8)	76.0 (45.4, 119.4)	36.9 (28.2, 56.6)	35.7 (24.8, 47.1)
C20:1	0.5 (0.5)	0.7 (0.5)	0.8 (0.6)	1.0 (0.5)	0.6 (0.5, 1.1)	1.0 (0.6, 1.5)*	1.5 (1.0, 2.4)	1.8 (1.3, 2.7)
C22:1	0.0 (0.0, 0.6)	0.2 (0.0, 2.5)	2.0 (0.2, 4.5)	3.3 (1.2, 5.7)	1.5 (0.3, 5.5)	5.1 (1.3, 8.0)*	10.3 (6.3, 16.0)	11.1 (8.9, 17.7)
C16:2	0.1 (0.1, 0.3)	0.3 (0.2, 0.4)	0.3 (0.1, 0.8)	0.5 (0.3, 0.9)	0.4 (0.2, 0.6)	0.4 (0.2, 0.6)	0.2 (0.1, 0.3)	0.2 (0.1, 0.3)
C18:2	11.6 (6.2, 19.6)	13.2 (9.1, 18.8)	48.7 (42.0)	61.9 (58.6)	41.2 (30.6, 85.9)	49.9 (31.1, 76.2)	17.5 (11.9, 26.1)	18.5 (11.9, 24.8)
C18:3	1.0 (0.5, 2.0)	1.2 (0.7, 1.7)	5.6 (4.6)	6.3 (5.3)	4.4 (3.0, 7.2)	5.6 (4.0, 8.7)	3.4 (2.4, 4.7)	4.0 (3.0, 5.5)
C20:2	0.1 (0.1)	0.1 (0.1)	0.2 (0.1, 0.5)	0.2 (0.1, 0.4)	0.2 (0.1, 0.4)	0.2 (0.1, 0.4)	0.2 (0.1, 0.3)	0.2 (0.1, 0.5)*
C20:4	0.1 (0.1)	0.1 (0.1)	0.7 (0.7)	0.6 (0.5)	0.4 (0.3, 1.0)	0.3 (0.2, 0.7)	0.1 (0.1, 0.2)	0.1 (0.1, 0.2)

Notes: Data are presented as mean (standard deviation) for parametric variables; median (interquartile range) for non-parametric variables. *A significant difference ($P < 0.05$) between NB and OB within a given people group.

Abbreviations: OB, obesity group (BMI ≥28.0 kg/m^2); NB, normal-BMI group (18.5≤ BMI <24.0 kg/m^2); DFAs, dietary fatty acids.

Table 3 Plasma Fatty Acids of four Minorities (mg/L)

PFAs	Hani People (n = 44)		Bulang People (n = 60)		Wa People (n = 70)		Naxi People (n = 158)	
	NB (n = 22)	OB (n = 22)	NB (n = 30)	OB (n = 30)	NB (n = 35)	OB (n = 35)	NB (n = 79)	OB (n = 79)
C14:0	1.0 (0.5)	1.3 (0.5)	0.6 (0.5, 1.2)	1.1 (0.8, 1.8)	0.9 (0.6, 1.3)	1.5 (0.8, 2.0)*	1.0 (0.7)	1.2 (0.5)*
C15:0	0.4 (0.4, 0.4)	0.4 (0.4, 0.4)	0.2 (0.1)	0.2 (0.1)	0.2 (0.1)	0.2 (0.1)	0.2 (0.2, 0.3)	0.3 (0.0, 0.3)
C16:0	14.0 (4.6)	17.1 (4.9)*	13.5 (12.3, 16.3)	18.1 (15.1, 22.4)*	14.3 (11.1, 18.7)	19.9 (14.2, 24.8)*	14.1 (11.9, 17.3)	15.5 (12.5, 17.8)
C17:0	0.5 (0.1)	0.5 (0.1)	0.3 (0.3, 0.4)	0.4 (0.3, 0.5)	0.4 (0.1)	0.5 (0.1)	0.4 (0.3, 0.5)	0.4 (0.4, 0.5)*
C18:0	14.8 (3.6)	16.6 (3.3)	16.9 (14.8, 19.4)	18.5 (14.9, 21.7)	18.5 (7.0)	20.2 (6.5)	15.8 (12.7, 17.9)	13.0 (11.4, 15.0)*
C20:0	0.4 (0.1)	0.4 (0.1)	0.5 (0.2, 0.5)	0.3 (0.1, 0.5)	0.4 (0.2)	0.4 (0.1)	0.5 (0.5, 0.5)	0.5 (0.5, 0.5)*
C22:0	0.3 (0.3, 0.3)	0.3 (0.3, 0.3)	0.4 (0.0, 0.5)	0.1 (0.0, 0.5)	0.3 (0.2)	0.3 (0.1)	0.5 (0.5, 0.5)	0.5 (0.5, 0.5)
C16:1 n-7	2.1 (1.1)	2.9 (1.6)	1.5 (1.2, 2.9)	3.0 (2.4, 4.3)*	2.7 (2.0)	3.8 (1.9)*	2.0 (1.4)	2.1 (1.1)
C18:1 n-9t	0.3 (0.3, 0.3)	0.3 (0.3, 0.3)	0.3 (0.2)	0.3 (0.2)	0.2 (0.2)	0.2 (0.2)	0.4 (0.4, 0.4)	0.4 (0.4, 0.5)
C18:1 n-9c	18.2 (10.0)	23.9 (11.4)	16.0 (12.1, 22.6)	28.8 (20.9, 43.9)*	19.5 (13.2, 26.7)	32.0 (20.5, 40.5)*	25.6 (15.0)	28.5 (14.5)
C20:1 n-9	0.4 (0.3, 0.4)	0.4 (0.3, 0.6)	0.7 (0.4, 0.9)	0.5 (0.3, 0.9)	0.8 (0.6, 0.9)	0.7 (0.6, 1.1)	1.0 (0.8, 1.6)	0.9 (0.8, 1.4)
C22:1	0.3 (0.0, 0.3)	0.3 (0.0, 0.4)	0.1 (0.2)	0.2 (0.2)	0.2 (0.2)	0.2 (0.1)	0.4 (0.4, 0.5)	0.4 (0.4, 0.5)
C18:2 n-6c	21.3 (6.3)	27.3 (10.2)*	22.6 (18.4, 27.1)	28.2 (23.7, 32.5)	25.8 (14.3)	27.4 (9.8)	26.7 (21.6, 33.0)	32.5 (26.1, 37.8)*
C18:3 n-6	0.7 (0.3)	0.8 (0.5)	0.6 (0.3, 0.8)	0.7 (0.4, 0.9)	0.4 (0.4)	0.4 (0.3)	0.8 (0.3)	0.9 (0.3)*
C18:3 n-3	0.7 (0.3)	0.9 (0.3)	0.7 (0.5, 1.0)	0.8 (0.5, 1.2)	0.7 (0.5, 1.1)	0.7 (0.5, 1.1)	1.5 (1.0, 2.3)	1.9 (1.4, 2.7)*
C20:2 n-6	0.7 (0.3)	0.7 (0.3)	0.6 (0.5, 0.8)	0.7 (0.6, 1.0)	0.5 (0.4, 0.7)	0.5 (0.4, 0.6)	0.7 (0.6, 0.8)	0.7 (0.6, 0.8)
C20:3 n-6	1.5 (0.3)	1.5 (0.7)	0.9 (0.8)	1.5 (0.8)*	1.2 (0.9, 1.6)	1.6 (1.2, 1.9)*	1.3 (0.5)	1.7 (0.5)*
C20:3 n-3	0.3 (0.0, 0.4)	0.4 (0.0, 0.4)	0.3 (0.0, 0.6)	0.3 (0.0, 0.6)	0.4 (0.0, 0.6)	0.4 (0.0, 0.6)	0.6 (0.6, 0.9)	0.7 (0.6, 0.9)
C20:4 n-6	6.5 (2.4)	6.5 (4.3)	4.6 (4.1)	6.6 (4.9)	5.1 (4.0, 6.9)	6.6 (5.5, 8.0)	5.5 (2.3)	6.3 (1.9)*
C20:5 n-3	0.0 (0.0, 0.6)	0.0 (0.0, 0.0)	0.1 (0.2)	0.4 (0.4)*	0.3 (0.3)	0.6 (0.3)*	0.6 (0.5)	0.9 (0.4)*

Notes: Data are presented as mean (standard deviation) for parametric variables; median (interquartile range) for non-parametric variables. *A significant difference ($P < 0.05$) between NB and OB within a given people group.

Abbreviations: OB, obesity group (BMI ≥28.0 kg/m^2); NB, normal-BMI group (18.5≤ BMI <24.0 kg/m^2); PFAs, plasma fatty acids.

−3.778; $P < 0.05$) in the Naxi people, were significantly higher in OB group compared with NB group. Plasma levels of C18:0 ($Z = -3.660$; $P < 0.05$) and C20:0 ($Z = -2.419$; $P < 0.05$) were significantly higher in NB group compared with OB group in the Naxi people.

Table 4 Conditional Logistic Regression Analysis of Obesity

	β	OR	95% CI	P
Hani				
C16:0 (PFA)	0.270	1.310	1.028–1.669	0.029
Bulang				
–	–	–	–	–
Wa				
C20:3 n-6 (PFA)	1.833	6.250	1.224–31.927	0.028
C20:1 (DFA)	2.223	9.231	1.253–68.016	0.029
Naxi				
C18:0 (PFA)	−0.238	0.788	0.681–0.912	0.001
ALL				
C20:3 n-6 (PFA)	0.925	2.523	1.350–4.716	0.004
C20:5 n-3 (PFA)	1.285	3.614	1.412–9.251	0.007

Abbreviations: PFA, plasma fatty acid; DFA, dietary fatty acid; OR, odds ratio.

Discriminant Analysis of Dietary Fatty Acids, Plasma Fatty Acids and Obesity

As shown in Tables 1 and 4. In the model that adjusted by LDL-C, plasma C16:0 (OR = 1.310; 95% CI, 1.028–1.669; $P < 0.05$; power = 0.510) was showed to be an independent predictive factor for obesity in the selected Hani population. In the model that adjusted by FPG, TG, HDL-C, plasma C20:3 n-6 (OR = 6.250; 95% CI, 1.224–31.927; $P < 0.05$; power = 0.197) and dietary C20:1 (OR = 9.231; 95% CI, 1.253–68.016; $P < 0.05$; power = 0.736) were shown to be independent predictive factors for obesity in the selected Wa population. In the model that adjusted by SBP, DBP, FPG, plasma C18:0 (OR = 0.788; 95% CI, 0.681–0.912; $P < 0.05$, power = 0.856) was shown to be an independent predictive factor for obesity in the selected Naxi population. And plasma C20:3 n-6 (OR = 2.523; 95% CI, 1.350–4.716; $P < 0.05$; power = 0.582) and plasma C20:5 n-3 (OR = 3.614; 95% CI, 1.412–9.251; $P < 0.05$; power = 0.999) were shown to be independent predictive factors for obesity in the total selected population.

The Canonical Correlation Analysis Between Dietary Fatty Acids Set and Plasma Fatty Acids Set

A canonical correlation analysis was performed on DFAs set and PFAs set, then seventeen typical correlation coefficients were obtained for each ethnic group. In the Bulang people (typical correlation coefficient: 0.974, 0.994 and 0.879; $P < 0.05$), the Naxi people (typical correlation coefficient: 0.676; $P < 0.05$) and the total selected ethnic minorities (typical correlation coefficient: 0.787, 0.628, 0.473; $P < 0.05$), PFAs set was positively correlated with DFAs set. But there was no correlation between PFAs set and DFAs set in the Hani (typical correlation coefficient: 0.993; $P > 0.05$) and the Wa people (typical correlation coefficient: 0.836; $P > 0.05$) in Figure 3.

Discussion

In the present study, plasma C16:0 in the Hani people, plasma C20:3 n-6 in the Wa people, plasma C18:0 in the Naxi people, plasma C20:3 n-6 and plasma C20:5 n-3 in the total selected ethnic minorities, were shown to be independent predictive factors for obesity. Only dietary C20:1 in the Wa people was linked to obesity. In addition, we observed a significantly positive correlation between DFAs set and PFAs set in the Bulang people, the Naxi people and the total selected ethnic minorities in Yunnan. However, the association disappeared in the Hani and the Wa people. This is the first study investigating the association of DFAs and PFAs with obesity in the four unique minority groups in Southwest China. Based on the current findings, different PFAs, but not DFAs,

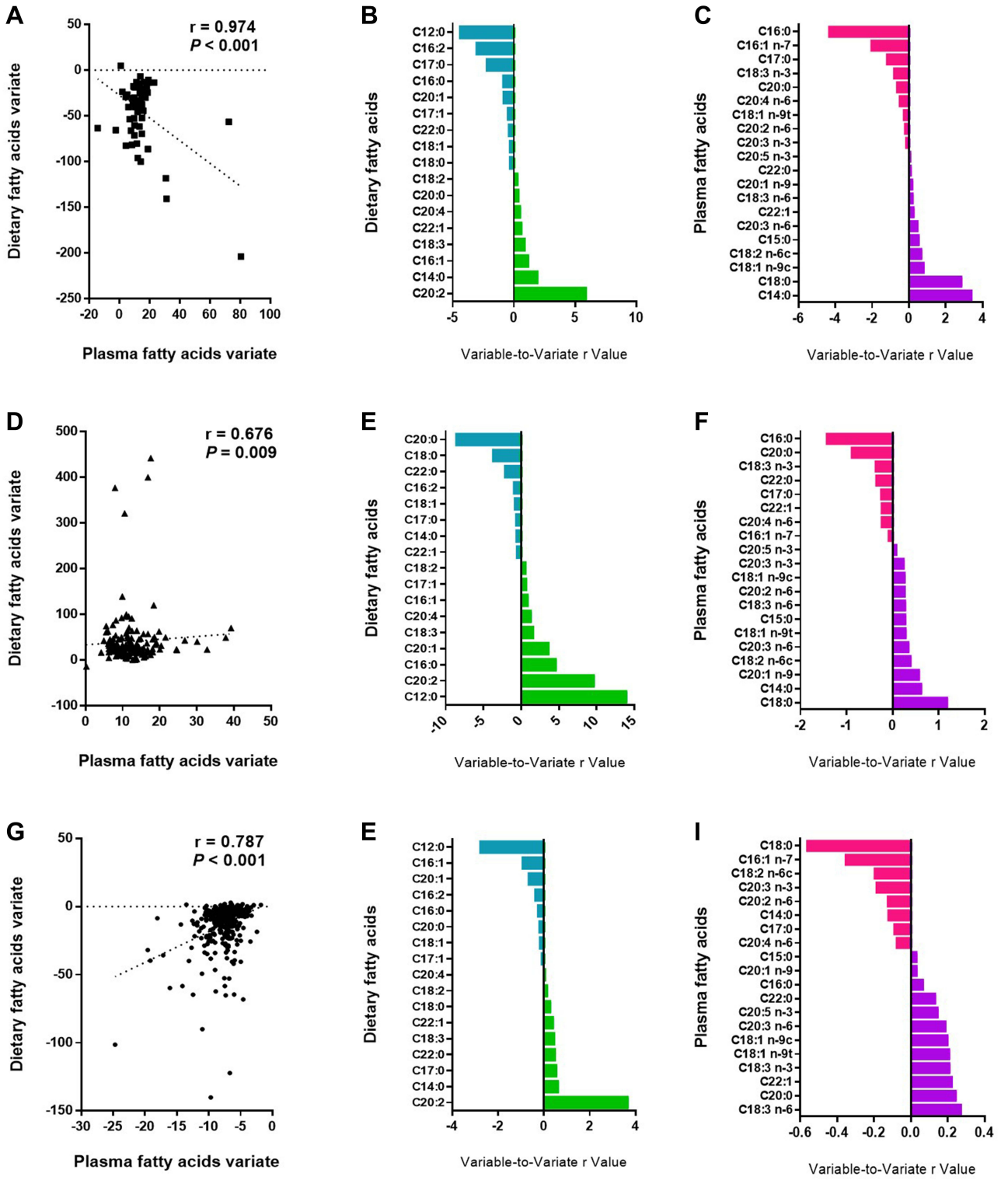

Figure 3 Canonical correlation analysis of dietary fatty acids and plasma fatty acids.

Notes: (**A**–**C**), canonical correlation analysis of dietary fatty acids and plasma fatty acids in the Bulang people; (**D**–**F**), canonical correlation analysis of dietary fatty acids and plasma fatty acids in the Naxi people; (**G**–**I**), canonical correlation analysis of dietary fatty acids and plasma fatty acids in all people. r, canonical correlation coefficient; P < 0.05 were considered significant.

linked to obesity among the four unique minority groups, and may be related to the different correlation between PFAs and DFAs in each minority group. These findings may provide theoretical bases for personalized prevention and treatment of different ethnic minorities with obesity.

Results of the our current study suggested that the PFAs are associated with obesity, which is similar to other studies.[23] In the present study, it was found that the 67% PFAs in the Naxi people were highest among the four ethnic minority groups, and the PFAs of the Hani people were significantly lower. This result may partially explain the high obesity prevalence of the Naxi people (16.7%) and the low obesity prevalence of the Hani people (4.5%). After analysis of the PFAs and obesity, it was found that the risk PFAs for obesity are different among the four ethnic minorities. Plasma C18:0 was a protective factor for obesity, and plasma C16:0, C20:3 n-6, C20:5 n-3 were risk factors for obesity. A prospective population study showed that plasma C16:0 increased serum cholesterol levels, which was positively correlated with central obesity.[24] A cell experiment has also proved that C16:0 can increase intestinal permeability, cause trigger inflammation and metabolic disorders, and is one of the important risk factors for obesity.[25] Several studies on different populations, showed that plasma C20:3 n-6 is related to obesity. A birth cohort explored that obesity is related to plasma C20:3 n-6. With each additional standard deviation of plasma C20:3 n-6 in pregnant women, BMI increased by 0.44 kg/m^2 in the offspring.[26–28] Guo evaluated the effect of dietary C20:5 n-3 on the lipid metabolism of mice with a high-fat diet and found that dietary C20:5 n-3 did not improve the obesity of the mice.[29] Li Y found that C18:0 inhibits adipocyte differentiation and lipid accumulation in 3T3-L1 cells by down-regulating adipogenic transcription factors and lipid accumulation-related genes.[30] These research results supported the conclusion of our study.

However, we found that there are few relationships between single DFA and obesity. Previous studies believed that excessive, insufficient and improper intake of dietary fatty acids could lead to obesity. Therefore, dietary fatty acid intake recommendations were formulated to prevent and treat obesity.[31] But these conclusions remain controversial.[16,32,33] Our study found that dietary fatty acids were not associated with obesity, and other research that the intake of single beneficial dietary fatty acids was not an effective treatment also supports our conclusion. Single DFA and obesity association eliminated may be related to the low absorption rate of DFAs, in addition, the dietary intake of other nutrients could have influenced the DFA intake, including fiber and sugar.

We also found the association between DFAs and PFAs are different in the four ethnic minorities, and this may partially be explained by the PFAs, but not DFAs, were independently associated with obesity. Previous studies have indicated that there are some drawbacks with studying the relationship between dietary intake and plasma concentrations in single fatty acid. The DFAs and PFAs are expected to be correlated and does not require data reduction, so we used canonical correlation analysis (CCA) to analyze the correlation between them. This analytical method increased the credibility of the results. According to the analysis results, the different relationship between DFAs and PFAs may be interpreted as follows. A high enough dietary fatty acid intake may lead to the association of DFAs and PFAs. Our results found that DFAs positively correlated with PFAs in Naxi people. This means that the higher the DFAs intake, the higher the PFAs level. In the previous study of our research group, the DFAs intake was highest and the ratio was most inappropriate in Naxi people among the four ethnic minorities.[34–36] A sufficiently high DFAs intake could change PFAs, which was consistent with some previous studies.[37,38] This correlation disappeared in the Hani people. The Hani people had the lowest DFAs intake.[6,39] This also supported our speculation. The environment may also be a factor. The Naxi people live in higher altitudes and colder areas (compared to the other three ethnic minorities, the altitude is about 1200 m higher and the temperature is about 3 °C lower). Low temperature could directly increase the proportion of unsaturated fatty acids in total PFAs.[40,41] The Bulang and Wa people share a similar environment, dietary habits (DFAs intake and PFA levels), and prevalence of obesity, but there were differences in the correlation between DFAs and PFAs. We speculated that it may be caused by genes.[41,42] In short, many factors affected the correlation between DFAs, PFAs, and obesity. PFAs may be a better indicator for epidemiological research.

A few limitations should be considered. First, our investigative subjects are limited and cannot fully reflect the characteristics of FAs in a wider range of obese minorities. Second, due to the particularity of the population, the result is difficult to be extrapolated. Third, although the sample data of the baseline survey is already representative in each ethnic group, the sample size is different in different groups due to the huge differences in obesity rates. Finally, the present study is cross-sectionally designed, which cannot be translated into a clear cause–effect inference.

Conclusions

In summary, the present study demonstrated that PFAs were independently linked to the risk of obesity. However, a weak association was found between DFAs and obesity. Therefore, whether adjusting a single DFA can effectively treat obesity in ethnic minorities would be further studied in the China Multi Ethnic Cohort (CMEC).

Data Sharing Statement

The supporting documents for this study are available from the corresponding author, Jianzhong Yin, on reasonable request.

Ethical Conduct

The study met the standards for the ethical treatment of participants and was approved by Kunming Medical University Medical Ethical Review Board.

Consent to Publication

The publication consent has been attached.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest for this work and declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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