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

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Abstract

Background: Dietary fatty acids (DFAs) and plasma fatty acids (PFAs) are linked with obesity. However, whether this association exists amongst ethnic minorities remains lacking. The present case-control study was designed to investigate the correlation between DFAs, PFAs and obesity in 4 ethnic minority groups to Southwest China.

Method: A total of 166 obese people, and 166 normal-BMI subjects were recruited from 4 different ethnic minority groups. The subjects were also matched based on their age-, sex-, and ethnicity. 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; $P < 0.05$) in the Hani people, plasma C18:0 (OR = 0.499; 95% CI, 0.343 - 0.727; $P < 0.05$) in the Naxi people, plasma C20:3 n-6 (OR = 6.250; 95% CI, 1.224 - 31.927; $P < 0.05$) in the Wa 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 4 ethnic minorities.

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].

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^[9]. Dietary fatty acids (DFAs), the important part of the daily dietary fat, play a vital role in obesity, 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)^[13–15]. Accordingly, increasing dietary PUFAs intake was recommended by the American Heart Association^[16]. However this was soon overturned by Heilesen JL^[17,18]. 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^[19, 20]. 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^[21]. 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 4 unique ethnic minority groups in Yunnan.

Methods

Participants

Considering the population gathering of minorities, the crowd cooperation and other factors, 3 prefectures in Yunnan Province are selected. One county is 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. 1913 residents (488 Hani people, 473 Naxi people, 464 Bulang people, and 488 Wa people) who were ≥ 18 years old, without mental illness, completed the survey and obtained biological samples. All participants provided written informed consent, and the study was approved by the institutional review board of the Kunming Medical University.

Of these 166 individuals (22 Hani people, 79 Naxi people, 30 Bulang people, and 35 Wa people) were obese, all of them were selected and assigned as the obese-BMI group (OB). Another 166 age-, sex-, and ethnicity-matched normal-body mass index (normal-BMI) subjects were recruited and assigned as the normal-BMI group (NB) (Fig. 1). OB was defined as $\text{BMI} (\text{weight}/\text{height}^2) \geq 28.0 \text{ kg}/\text{m}^2$, and NB was defined as $18.5 \leq \text{BMI} < 24.0 \text{ kg}/\text{m}^2$. (Fig. 1)

Data collection

A questionnaire was distributed to the participants to gather data including clinical characteristics, sociodemographic details, lifestyle and medical history. A semi-quantified food frequency method will be used to assess food intake, and calculate 20 dietary fatty acids, Total fatty acids (FAs), Saturated fatty acids (SFAs), Monounsaturated fatty acids (MUFAs), Polyunsaturated fatty acids (PUFAs), 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)] intake based on "Chinese Food Composition Table 2009" (Second Edition).

For each subjects, anthropometric indicators (height, body weight, waist and hip circumference) and blood pressure (BP) were measured by standard methods. After an overnight fast, fasting blood samples were collected after participants rested for 10 minutes in the seated position. All plasma samples were stored at -80°C without freeze-thaw cycles until assay. Fasting plasma glucose (FPG), total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL-C), and low-density lipoprotein (LDL-C) were assayed by a biochemical autoanalyzer (Beckman Coulter AU480 Biochemical Autoanalyser, USA).

The levels of 37 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-Pentadecenoic 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-Docosaheptaenoic 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), Dihomogamma 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. According the correlation coefficient range of the standard curve (0.9991 to 0.9998), detection limit range (0.01 to 0.41 mg/L), intraday CV value (0.27 to 6.15%), daytime CV value (0.91 to 8.70%), recovery rate (80.06 to 118.34%), the modified conditions were selected for PFA methylesteration: 7% sulphuric acid, 65 °C and 20 min.

Chromatographic analysis was performed with a gas chromatography/mass spectrometer (GCMS-QP2010, SHIMADZU, Japan) equipped with a fused-silica (100 m $\times$ 0.2 mm ID, 0.25 μ m film) capillary column (RT-2560, SHIMADZU). The following conditions were applied for gas chromatograph: oven temperature was held at 60 °C for 5 min, programmed from 60 to 165°C at a rate of 5°C/min and held at 165 °C for 1 min; 165 to 225°C at a rate of 2°C/min and held at 225°C for 27 min. Run time was 84 min. 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 μ L 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 min. 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.

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 (SD)), 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 ranks test for continuous variables and chi-square test tests for categorical variables. 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, including 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 were controlled for in the regression models. Relationships between dietary fatty acids set (17 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 (20 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.

Results

Baseline characteristics

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, 5.9% in the Bulang people, 7.2% in the Wa people and 17.8% in the Naxi people. Basic clinical characteristics of participants in 4 minorities were shown in (Table 1).

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)
Male gender, n (%)	20 (45.4)	24 (40.0)	20 (28.6)	48 (30.4)
Low education lever, n (%)	24 (54.5)	27 (45.0)	28 (40.0)	24 (15.2)
Low physical activity, n (%)	6 (13.6)	2 (3.3)	8 (11.4)	29 (18.4)
Peasantry, n (%)	33 (75.0)	57 (95.0)	66 (94.3)	146 (92.4)
Married, n (%)	37 (84.1)	49 (81.7)	63 (90.0)	140 (88.6)
Smoking, n (%)	16 (36.4)	18 (30.0)	18 (25.7)	30 (19.0)
Alcohol use, n (%)	19 (43.2)	12 (20.0)	9 (12.9)	13 (8.2)
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)	2.7 (1.6, 4.1)	1.9 (1.5, 2.4)
Plasma TC, mmol/L	4.8 (3.8, 5.5)	5.2 (4.4, 6.0)	5.3 (4.7, 6.0)	5.0 (4.2, 5.3)
Plasma LDL-C, mmol/L	2.6 (2.1, 3.1)	3.2 (2.6, 3.7)	3.1 (2.6, 3.6)	2.7 (2.1, 3.2)
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)
Total DFA, mg/L	95.2 (62.8, 133.7)	53.2 (38.6, 82.3)	55.2 (40.2, 73.4)	112.3 (81.2, 161.4)
Total PFA, mg/L	92.3 (68.0, 109.2)	95.3 (79.3, 130.3)	94.9 (75.6, 139.6)	102.5 (84.7, 123.2)
Total n = 332. 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; DFA, dietary fatty acid; PFA, plasma fatty acid.				

The total DFAs, SFAs, MUFAs, PUFAs and 16 DFAs intakes were calculated according “Chinese Food Composition Table 2009”. PUFAs and 3 DFAs (C22:0, C20:1, C22:1) intakes were markedly higher, and dietary C16:1 intake was markedly lower in the Naxi people than in other 3 minorities ($P < 0.05$). The Hani people had significantly lowest intake of 7 DFAs (C12:0, C17:0, C20:0, C22:0, C18:2, C18:3, C20:2) ($P <$

0.05) in 4 minorities. 94% DFAs showed no differences between the Bulang and the Wa people. The comparison of DFAs (C16:0, C18:0, C20:3) among 4 minorities were showed in Fig. 2 (A).

20 PFAs were detected by GC/MS. Compared among 4 minorities, 15 PFAs(C15:0, C16:0, C17:0, C18:0, C18:1 n-9t, C18:2 n-6c, C20:0, C18:3 n-6γ, C20:1, C18:3 n-3α, C20:2 n-6, C22:0, C20:3 n-3, C22:1, C20:5 n-3) showed statistically significant differences ($P < 0.05$). The total PFA level and 10 PFAs (C18:1 n-9, C18:2 n-6c, C20:0, C18:3 n-6γ, C20:1 n-9, C18:3 n-3α, C22:0, C20:3 n-3, C22:1, C20:5 n-3) levels were markedly higher, and plasma C16:0 level was markedly lower in the Naxi people than in other 3 minorities ($P < 0.05$). Compared with other 3 minorities, the Hani people had significantly higher levels of 3 PFAs (C20:0, C20:1 n-9, C20:3 n-3, C20:5 n-3) and lower levels of 2 PFAs (C15:0, C17:0) ($P < 0.05$). 11 PFAs (C15:0, C16:0, C17:0, C18:1 n-9t, C18:2 n-6c, C20:0, C18:3 n-6, C18:3 n-3, C22:0, C22:1, C20:5 n-3) levels showed statistically significant differences between Hani people and Naxi people ($P < 0.05$). 5 PFAs (C20:0, C18:3 n-6, C20:1 n-9, C20:2 n-6, C20:5 n-3) levels showed statistically significant differences between the Bulang people and the Wa people ($P < 0.05$). The comparison of PFAs (C16:0, C18:0, C20:3 n-6) among 4 minorities were showed in Fig. 2 (B).

The case-control results of dietary fatty acids and plasma fatty acids in 4 minorities

The case-control results of DFAs and PFAs in 4 minorities were showed in (Table 2).

Table 2
Dietary fatty acids and plasma fatty acids of 4 ethnic minorities

FA	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)
Dietary fatty acids (DFA) (% of total fatty acids)								
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)
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)
C20:2	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)
C20:3	-	0.0 (0.0, 0.0)	0.0 (0.0, 0.1)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.00, 0.0)	-	-
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 (0.0, 0.2)	0.1 (0.1, 0.2)
C20:5	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.1)
Plasma fatty acids (PFA) (mg/L)								
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)
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) *
C20:2 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)

Normally distributed data is described by M (SD), skewed distribution data is described by M (P25, P75). *, $P < 0.05$.

FA	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)
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) *
Normally distributed data is described by M (SD), skewed distribution data is described by M (P25, P75). *, $P < 0.05$.								

Dietary levels of C17:1 ($t = -2.831$; $P < 0.05$), C18:1 ($t = -2.978$; $P < 0.05$), C20:3 ($Z = -2.361$; $P < 0.05$) in the Wa people; C15:0 ($Z = -2.048$; $P < 0.05$), 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 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 ($P \geq 0.05$).

Among the 20 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 ($t = -3.115$; $P < 0.05$), C20:5 n-3 ($t = -4.10$; $P < 0.057$), total FA ($Z = -2.314$; $P < 0.05$) 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 ($Z = -2.821$; $P < 0.05$), 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 ($t = -2.1020$; $P < 0.05$), C18:3 n-3 ($Z = -2.835$; $P < 0.05$), C20:3 n-6 ($t = -5.360$; $P < 0.05$), C20:4 n-6 ($t = -2.537$; $P < 0.05$), C20:5 n-3 ($t = -3.778$; $P < 0.05$) and total FA ($Z = -2.150$; $P < 0.05$) 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.

Discriminant analysis of dietary fatty acids, plasma fatty acids and obesity

In model that adjusted for ethnicity, age, gender, education level, physical activity, occupation, marital status, annual family income, smoking, alcohol drinking, SBP, DBP, FPG, TC, TG, LDL-C and HDL-C, plasma C16:0 (OR = 1.310; 95% CI, 1.028–1.669; $P < 0.05$) in the selected Hani population; plasma C20:3 n-6 (OR = 6.250; 95% CI, 1.224–31.927; $P < 0.05$) and dietary C20:1 (OR = 8.879; 95%CI, 1.126–69.989; $P < 0.05$) in the selected Wa population; DBP (OR = 1.054; 95% CI, 1.006–1.103; $P < 0.05$) and plasma C18:0 (OR = 0.499; 95% CI, 0.343–0.727; $P < 0.05$) in the selected Naxi population; SBP (OR = 1.045; 95%CI, 1.022–1.068; $P < 0.05$), plasma C20:3 n-6 (OR = 2.523; 95%CI, 1.350–4.716; $P < 0.05$) and plasma C20:5 n-3 (OR = 3.614; 95%CI, 1.412–9.251; $P < 0.05$) in the total selected population, were showed to be independent predictive factors for obesity. (Table 3)

Table 3
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*
Naxi				
C18:0 (PFA)	-0.695	0.499	0.343–0.727	< 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*
PFA, plasma fatty acid; DFA, dietary fatty acid; OR, odds ratio. The model was adjusted by ethnicity, age, gender, education lever, physical activity, occupation, marital status, annual family income, smoking, alcohol drinking, SBP, DBP, FPG, TC, TG, LDL-C and HDL-C. *, P < 0.05.				

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 17 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; $P > 0.05$). (Fig. 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 4 unique minority groups in Southwest China. Based on the current findings, different PFAs, but not DFAs, linked to obesity among the 4 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^[22]. In the present study, it was found that the 67% PFAs in the Naxi people were highest among the 4 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 (17.8%) 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 4 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^[23]. 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^[24]. 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 0.44kg/m² in the offspring^[25-27]. Guo XF 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^[28]. 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^[29]. 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^[30]. But these conclusions remain controversial^[16-18]. 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 4 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 analyse (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 4 ethnic minorities^[31, 32]. A sufficiently high DFAs intake could change PFAs, which was consistent with some previous studies^[33, 34]. This correlation disappeared in the Hani people. The Hani people had the lowest DFAs intake^[6, 35]. 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 3 ethnic minorities, the altitude is about 1200m higher and the temperature is about 3°C lower). Low temperature could directly increase the proportion of unsaturated fatty acids in total PFAs^[35, 36]. 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^[37, 38]. 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, the present study is cross-sectionally designed, which cannot be translated into a clear cause–effect inference.

In summary, the present study demonstrated that PFAs were independently linked to the risk of obesity. But a weak association was found between DFAs and obesity. Therefore, whether adjusting a single DFA can effectively treat obesity in these 4 ethnic minorities requires further research.

Abbreviations

List of abbreviations	
DFAs	Dietary Fatty Acids
PFAs	Plasma Fatty Acids
FAs	Fatty Acids
BMI	Body Mass Index
CCA	Canonical Correlation Analysis
PUFAs	Polyunsaturated Fatty Acids
OB	Obese-BMI
NB	Normal-BMI
SFAs	Saturated Fatty Acids
MUFAs	Monounsaturated Fatty Acids
BP	Blood Pressure
FPG	Fasting Plasma Glucose
TC	Total Cholesterol
TG	Triglycerides
HDL-C	High-Density Lipoprotein-Cholesterol
LDL-C	Low-Density Lipoprotein- Cholesterol
DBP	Diastolic Blood Pressure
SBP	Systolic Blood Pressure

Declarations

Author contribution.

1. JH and Yr L patient data were analyzed and interpreted
2. Db Z participate in plasma fatty acid detection
3. Others participated in the main writing of the manuscript

All authors read and approved the final manuscript

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Ethics approval and consent to participate

The research does not report on or involve the use of any animal or human data or tissue, not applicable the ethics approval and consent to participate. The publication consent has been attached.

Availability of data

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests

Consent to publication

The publication consent has been attached

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Figures

Figure 1

Inclusion prosses of enrolled people

OB, obeses ($\text{BMI} \geq 28.0 \text{ kg/m}^2$); NB, normal BMI ($18.5 \leq \text{BMI} < 24.0 \text{ kg/m}^2$).

Figure 2

Comparison of fatty acids among 4 ethnic minorities

A, comparison of dietary fatty acids (C16:0, C18:0, C20:3) among 4 minorities; B, comparison of plasma fatty acids (C16:0, C18:0, C20:3 n-6) among 4 minorities; *, compared with Hani people, $P < 0.05$.

Figure 3

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

A, B, C, canonical correlation analysis of dietary fatty acids and plasma fatty acids in the Bulang people; D, E, F, canonical correlation analysis of dietary fatty acids and plasma fatty acids in the Naxi people; G,

H, I, canonical correlation analysis of dietary fatty acids and plasma fatty acids in all people. r, canonical correlation coefficient.

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