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Improved fatty acid profile reduces body fat and arterial stiffness in obese adolescents upon combinatorial intervention with exercise and dietary restriction

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ABSTRACT

Objective: In order to examine the effect of 4-week combinatorial intervention with exercise training and dietary restriction on serum fatty acids, and to explore the correlation of intervention-induced improvement of serum fatty acid profile with the reduction of body fat and arterial stiffness.
Methods: Thirty-three obese adolescents were randomized into the intervention (n = 19) and control (n = 14) groups. The participants from the intervention group were subjected to 4-week combinatorial intervention with exercise training and dietary restriction while the participants from the control group maintained regular activities and diet habits. Anthropometry, serum fatty acids and arterial stiffness were measured before and after 4-week intervention.
Results: The participants upon combinatorial intervention revealed the improved body compositions and serum fatty acid profile, and reduced arterial stiffness when compared with their basal levels and the control participants ($p < 0.05$). Moreover, the decrease in myristic acid, stearic acid, arachidic acid, behenic acid, palmitoleic acid, and dihomo-γ-linolenic acid, was associated with the reduction in body fat. A positive correlation between arachidonic acid and left brachial ankle pulse velocity was observed, and the increase in docosahexaenoic acid was associated with the reduction of left brachial ankle pulse wave velocity and the enhancement of right ankle brachial index.
Conclusion: The 4-week combinatorial intervention is a useful strategy to improve serum fatty acid profile along with the reduction of body fat and arterial stiffness in obese adolescents.

1. Introduction

Overweight and obesity are known as the 21st century epidemic diseases and seriously affect the population's life quality. Globally, 15–20% of adolescents are at risk of overweight, and additional 15% are at risk of obesity.[1] Obesity induced by excessive fat accumulation is prone to cause arterial stiffness, cardiovascular diseases (CVD), inflammation and type 2 diabetes.[2,3]

Arterial stiffness as a key characteristic of atherosclerosis is positively associated with obesity,[4] and can increase the risk of CVD in obese people.[5] Brachial-ankle pulse wave velocity (baPWV) and ankle-brachial index (ABI) are the reliable indicators of systemic and peripheral arterial stiffness, respectively.[6,7] Abnormally high baPWV and low ABI are associated with high body fat and body mass index (BMI) in obese subjects,[8,9] but exercise or/and diet restriction could reduce fat mass and baPWV, and increase ABI in obese adolescents.[10–13] Thus, the reduction of arterial stiffness and body fat through exercise training or/and diet intervention is essential to the prevention and control of CVD in obese subjects.

Serum polyunsaturated fatty acids (PUFAs) are significantly correlated with the intake of polyunsaturated fat, while saturated fatty acids (SFAs) and monounsaturated fatty acids (MUFAs) may

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reflect a diet rich in saturated fat; therefore, serum fatty acid profile in human body is a biomarker to reflect the intake of fatty acids within weeks.[14,15] Moreover, SFAs can expand white adipose tissues, increase oxidative stress and inflammation, and then lead to insulin resistance by impaired insulin signal in multiple tissues[16–18]; MUFAs can reduce adipocyte size and suppress lipogenesis by promoting the oxidation of fatty acids, thereby alleviating obesity, diabetes and CVD[19]; n-3 PUFAs, beneficial to anti-inflammation and anti-oxidation,[20,21] can mitigate blood cholesterol accumulation and enhance insulin sensitivity by modulating signal pathways of lipoprotein metabolism.[22] Compared with persons with normal body weights, obese people have higher levels of serum SFAs and MUFAs, and lower levels of n-3 PUFAs,[23,24] thereby leading to the development of chronic diseases, such as insulin resistance, obesity and metabolic syndrome.[18,25] Increasing evidence has demonstrated that serum fatty acids in obese subjects may be changed by an open-ended exercise or/and diet intervention after at least two months.[26–28] However, it is unclear whether the serum fatty acid profile could be improved by a 4-week combinatorial intervention with exercise training and dietary restriction (a closed residential camp), and whether the intervention-induced improvement of serum fatty acid profile is associated with the reduction of body fat and arterial stiffness in obese adolescents. Therefore, we hypothesized that the 4-week closed residential camp is beneficial for the improvement of serum fatty acid profile and the reduction of body fat and arterial stiffness in obese adolescents.

2. Methods

2.1. Participants and study design

Fifty obese adolescents with the age of 14–18 years old were recruited for eligibility assessment. Those who do not match the obesity criteria defined by body mass index reference norm for screening overweight and obesity in Chinese children and adolescents were excluded. The persons with smoking habit and diagnosed cardiovascular, lung, kidney, liver or gastrointestinal, bone and infectious diseases, or behavioral or psychological disorders were also excluded. Then, the qualified forty participants were randomly and averagely assigned to the intervention group and the control group. Due to the interruption by quitting camp, traveling and upper respiratory tract infection, 33 obese adolescents (age: 15.89 ± 1.87 years old; BMI: 32.07 ± 3.33 kg/m^2) who completed the whole camp program were included for the final analysis (CONSORT flow diagram as shown in Fig. 1). All interventions were conducted according to the ethical guidelines of the Declaration of Helsinki, and approved by the Medical Ethics Committee of Wuhan Sports University (No. 2019004). The trial was registered on the Chinese Clinical Trail Registry (Registration Number: ChiCTR1900025684). All participants signed a written informed consent about the risks, discomforts and benefits associated with this study.

The participants from the intervention group were undertaken in a closed residential camp with uniformed management for their accommodation, diet and exercise training during the 4-week intervention period. The participants from the control group were not involved in any special exercise training and diet protocols, and maintained their regular lifestyle (diet and physical activity) for the whole intervention duration. All participants were provided with a series of physical examinations before and after intervention in Hubei Key Laboratory of Exercise Training and Monitoring at Wuhan Sports University. Body compositions, serum FA profile, and indicators for arterial stiffness of all participants in this study were determined at the identical condition.

2.2. Exercise training and dietary restriction

The participants from the intervention group were subjected to exercise training and dietary restriction on the second day after joining in the camp. According to the principle of calorie control and balanced nutrition, the dietician formulated participants' diets. Total daily energy intake was calculated by the following formula: energy intake (kcal) = standard body weight (kg) × 20–25 kcal/kg, and standard body weight (kg) = height (cm)−105. Considering physical fitness and the demand to maximize fat catabolism, the balanced diet was composed of 50% carbohydrates, 30% protein and 20% fat, and calories were distributed at three meals with 40%, 40% and 20%, respectively, which has been validated in our previous publication.[29] The carbohydrates consumed by participants were mainly derived from starch, fruits and vegetables; protein was mainly acquired from eggs, meat and fish; fat mainly came from vegetable oil and fish oil. Moreover, the recipes were weekly adjusted according to body weights of the participants, and any other extra calories were prohibited.

Participants from the intervention group were engaged in exercise training with 6 days per week and 3 times each day for 4 consecutive weeks. Daily training periods were 9:00–11:00, 15:00–17:00 and 19:00–20:30. Each exercise training period included 10–15 min warm-up and cool-down. Exercise training was composed of aerobic exercise, resistance exercise and core training. Aerobic exercise included jogging, swimming, table tennis, tennis, badminton, basketball, spinning, rope skipping and climbing at the intensity of 60–70% maximum heart beat (HR_{max}). Resistance exercise included upper and lower limb, chest and back strength training in gymnasium. Core training was composed of strength and stability training, including fitball, supine leg-lifting, back bridge, prostrate elbow brace, push-ups and sit-ups on the mat. More intervention details related to exercise training and dietary intake were referred in our previously published study.[29]

2.3. Measurement of body compositions and arterial stiffness

Waist circumference (WC) was measured at the midpoint between the lower rib and the iliac crest using a tape measure. Hip circumference (HC) was taken at the largest point at the level of the greater trochanters. Body composition variables, such as body weight, BMI, body fat (BF) and fat mass index (FMI), were determined by body composition analyzer (X-Scan Plus II, Jawon, Korea) according to bioelectrical impedance analysis. ABI and baPWV were measured by a noninvasive automatic device (BP-203RPE III, OMRON, Japan) after a 5-min rest in the supine position.

2.4. Blood sample collection and serum FA profile determination

At 8:00 a.m., 5 mL of fasting venous blood samples were collected with a vacuum serum tube without anticoagulant at the baseline and after 4-week intervention. After standing at room temperature for 1–2 h, blood samples were centrifuged at 3000 rpm for 15 min at 4 °C. The aliquots of the serum were stored at −80 °C for future analysis.

Approximately 200 μL of serum sample were mixed with 400 μL of methanol and 1 mL of *n*-hexane. The mixture was vortexed thoroughly for 1 min and sonicated for 5 min at 4 °C and then centrifuged at 8000 rpm for 5 min. Then, 750 μL of supernatant were harvested for drying under nitrogen flow. After adding 2 mL of concentrated sulfuric acid/methanol solution with a volume ratio of 5% and 25 μL of butylated hydroxytoluene (BHT)/methanol solution with a mass ratio of 0.2%, the mixture was vortexed for 1 min and heated at 90 °C water bath for 60 min. After cooling to room temperature, the mixture with adding 1 mL of *n*-hexane and 2 mL

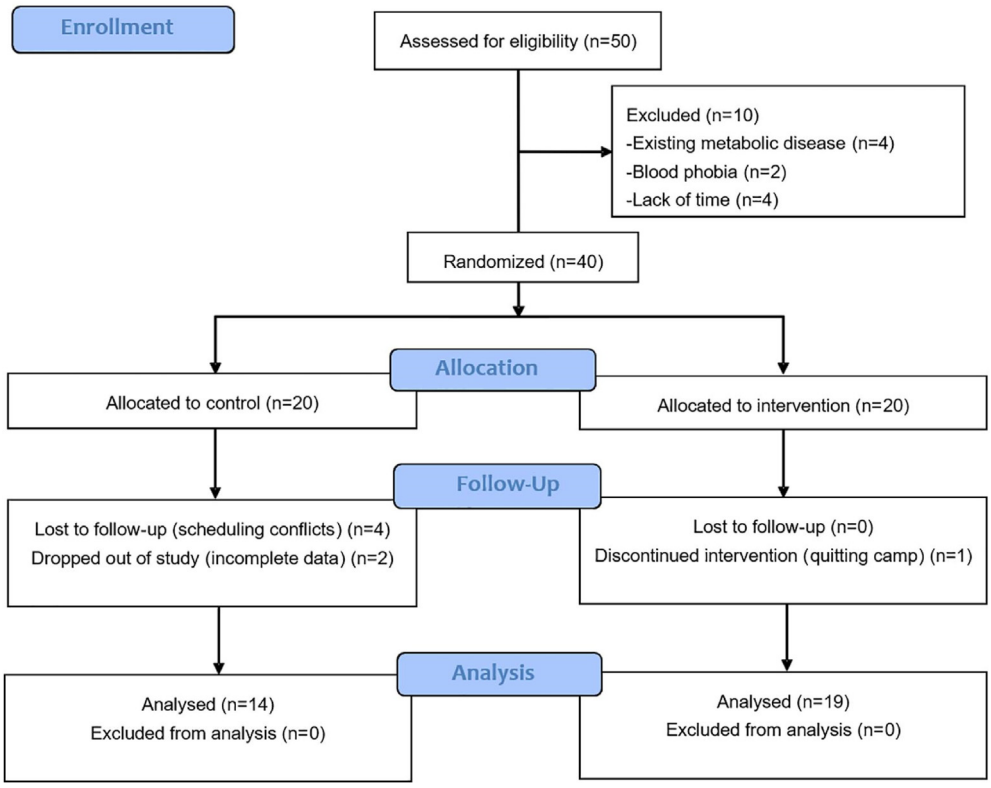

Fig. 1. Flow diagram of participants through the study program.

of saturated sodium chloride was vortexed thoroughly for 1 min and centrifuged at 3500 rpm for 5 min at 4 °C. Then, 500 μL of supernatant were dried under nitrogen stream after transferred into another tube, and dissolved in 100 μL of *n*-hexane. Finally, 70 μL of supernatant were used for the analysis of FA profile by gas chromatography coupled with mass selective detector (GC-MSD) (7890A GC-5975C MSD, Agilent, USA).

2.5. Statistical analysis

All data were presented as mean ± standard deviation (M ± SD). SPSS 20.0 (SPSS Inc, Chicago, IL, USA) was used for statistical analysis. The Shapiro-Wilk test was performed to examine the normality of the data. A two-way ANOVA with repeated measures [group (control and intervention) × time (before and after 4 weeks)] was used to examine the interaction and major effect on each outcome. If a significant interaction effect was noted, the simple effect test was used for within and between group pairwise comparisons. Based on significant variables, Pearson's correlation coefficients were applied to assess the correlations among fatty acid levels, body compositions, and indicators of arterial stiffness after 4-week intervention. The significant difference was considered at $p < 0.05$.

3. Results

3.1. The characteristics of the participants

The characteristics of the participants from both groups before and after 4-week intervention are shown in Tables 1 and 2. There was no significant difference in basal parameters between two groups ($p > 0.05$). Similarly, no changes in all parameters in the control group before and after 4 weeks were observed ($p > 0.05$).

3.2. Combinatorial intervention modulated body compositions and reduced arterial stiffness

The 4-week combinatorial intervention with exercise training and dietary restriction could lead to the significant reduction in body weight, BMI, BF, FMI, WC and WHR of obese adolescent participants, and improvement in right ABI relative to their basal levels and to those of the participants from the control group ($p < 0.05$). Similarly, HC and left baPWV were also significantly reduced after 4-week intervention ($p < 0.01$), as shown in Table 1.

3.3. Combinatorial intervention improved serum FA profile

After 4-week intervention, the participants from the intervention group revealed the significant reduction in myristic acid (C14:0), pentadecanoic acid (C15:0), stearic acid (C18:0), arachidic acid (C20:0), behenic acid (C22:0), total SFAs, palmitoleic acid (C16:1 n-7), oleic acid (C18:1 n-9), gadoleic acid (C20:1 n-9), total MUFAs, dihomo-γ-linolenic acid (C20:3 n-6), arachidonic acid (C20:4 n-6), n-6 PUFAs, α-linolenic acid (C18:3 n-3), and total PUFAs, and increase in docosahexaenoic acid (C22:6 n-3) when compared with their basal levels and those of the participants from the control group ($p < 0.05$), as shown in Table 2.

3.4. Improved serum FA profile was correlated with body compositions and arterial stiffness

In order to explore the relationships among these variables, we further analyzed the correlations between the amplitude of significantly changed parameters. As shown in Table 3, the decrease in C14:0 were associated with the reduction in BF ($r = 0.477$, $p = 0.039$); the reduction in C18:0 were associated with the decrease in body weight ($r = 0.606$, $p = 0.006$), BF ($r = 0.567$,

Table 1
Characteristics of the participants before and after a 4-week combinatorial intervention.

Variable	Control group (n = 14)		Intervention group (n = 19)	
	Pre	Post	Pre	Post
Body compositions				
Body weight (kg)	88.07 ± 13.46	88.92 ± 13.84	86.86 ± 14.36	77.83 ± 13.36***#
BMI (kg/m^2)	32.19 ± 2.56	32.49 ± 2.62	31.98 ± 3.87	28.20 ± 3.49***##
Body fat (kg)	29.69 ± 4.78	30.44 ± 5.24	30.53 ± 8.69	23.94 ± 8.06***#
FMI (kg/m^2)	10.92 ± 1.65	11.17 ± 1.70	11.23 ± 2.92	8.65 ± 2.66***##
WC (cm)	100.57 ± 8.01	100.98 ± 7.93	102.05 ± 11.50	92.49 ± 9.80***#
HC (cm)	109.93 ± 5.34	109.75 ± 5.82	111.30 ± 10.19	105.43 ± 10.80***
WHR	0.91 ± 0.04	0.92 ± 0.04	0.92 ± 0.05	0.88 ± 0.05***#
Arterial stiffness				
Right baPWV (cm/s)	978.57 ± 72.70	977.29 ± 55.20	974.47 ± 137.36	953.68 ± 151.58
Left baPWV (cm/s)	976.29 ± 52.06	975.57 ± 82.24	971.53 ± 137.08	925.53 ± 137.12**
Right ABI	0.98 ± 0.11	0.98 ± 0.13	0.97 ± 0.10	1.14 ± 0.12***##
Left ABI	1.05 ± 0.14	1.04 ± 0.11	1.02 ± 0.13	1.04 ± 0.07

Note: All data were presented as mean ± standard deviation (M ± SD).
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$ relative to pre.
#$p < 0.05$, ##$p < 0.01$ relative to the control.
BMI, body mass index; FMI, fat mass index; WC, waist circumference; HC, hip circumference; WHR, Waist-hip ratio; baPWV, brachial ankle pulse wave velocity; ABI, ankle brachial index.

Table 2
Serum fatty acid profile before and after 4-week combinatorial intervention.

Variable (μg/mL)	Control group (n = 14)		Intervention group (n = 19)	
	Pre	Post	Pre	Post
SFAs	677.58 ± 93.42	694.98 ± 42.69	687.93 ± 172.81	565.22 ± 127.81**##
C12:0	1.66 ± 0.36	1.67 ± 0.40	1.56 ± 0.29	1.63 ± 0.28
C14:0	16.01 ± 4.02	16.32 ± 3.02	15.66 ± 6.32	9.03 ± 1.90***###
C15:0	4.28 ± 0.63	4.45 ± 0.74	4.36 ± 0.86	3.57 ± 0.47**###
C16:0	434.53 ± 55.63	447.21 ± 19.85	445.92 ± 112.89	395.07 ± 92.06
C17:0	9.55 ± 2.29	9.39 ± 2.33	9.47 ± 2.77	9.06 ± 2.15
C18:0	168.80 ± 32.24	172.09 ± 28.02	167.93 ± 46.97	112.10 ± 34.69***###
C20:0	5.57 ± 2.63	5.75 ± 1.18	5.64 ± 1.62	4.16 ± 1.30**##
C22:0	10.11 ± 4.04	10.68 ± 3.90	10.05 ± 2.62	7.97 ± 2.85**#
C23:0	18.24 ± 7.12	18.41 ± 5.91	18.44 ± 5.37	15.45 ± 3.92
C24:0	8.83 ± 3.20	9.01 ± 3.28	8.89 ± 2.58	7.20 ± 1.69
MUFAs	272.25 ± 64.68	275.05 ± 49.66	267.57 ± 83.66	175.30 ± 50.67***###
C16:1 n-7	38.81 ± 12.40	37.92 ± 8.52	35.61 ± 12.47	25.32 ± 7.73**###
C18:1 n-9	206.06 ± 46.06	208.49 ± 31.75	205.95 ± 64.67	144.12 ± 44.73***###
C20:1 n-9	27.38 ± 16.00	28.64 ± 14.40	26.01 ± 14.49	5.86 ± 1.75***###
PUFAs	1067.82 ± 170.13	1083.67 ± 152.94	1086.84 ± 236.71	827.53 ± 190.37***###
n-6	1002.72 ± 161.39	1019.57 ± 145.70	1021.32 ± 225.63	761.20 ± 177.05***###
C18:2 n-6	597.67 ± 98.90	599.20 ± 100.60	592.07 ± 168.69	485.52 ± 149.70
C20:2 n-6	6.80 ± 1.71	6.67 ± 2.18	6.95 ± 2.34	5.84 ± 1.88
C20:3 n-6	31.74 ± 13.62	32.69 ± 12.94	30.50 ± 11.68	15.41 ± 3.83***###
C20:4 n-6	366.51 ± 95.42	381.01 ± 88.07	391.81 ± 128.23	254.43 ± 73.28***###
n-3	65.10 ± 21.68	64.10 ± 21.12	65.52 ± 21.25	66.33 ± 16.06
C18:3 n-3	21.85 ± 6.72	22.84 ± 7.76	20.79 ± 8.72	13.61 ± 5.87***###
C20:5 n-3	14.20 ± 6.78	13.30 ± 5.50	12.83 ± 5.12	11.65 ± 4.20
C22:6 n-3	29.04 ± 14.17	27.96 ± 13.67	31.90 ± 13.29	41.07 ± 11.29**##

Note: All data were presented as mean ± standard deviation (M ± SD).
*$p < 0.05$, **$p < 0.01$, and ***$p < 0.001$ relative to pre.
#$p < 0.05$, ##$p < 0.01$, and ###$p < 0.001$ relative to the control.
SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid. C12:0, lauric acid; C14:0, myristic acid; C15:0, pentadecanoic acid; C16:0, palmitic acid; C17:0, margaric acid; C18:0, stearic acid; C20:0, arachidic acid; C22:0, behenic acid; C23:0, tricosanoic acid; C24:0, lignoceric acid; C16:1 n-7, palmitoleic acid; C18:1 n-9, oleic acid; C20:1 n-9, gadoleic acid; C18:2 n-6, linoleic acid; C20:2 n-6, eicosadienoic acid; C20:3 n-6, dihomo-γ-linolenic acid; C20:4 n-6, arachidonic acid; C18:3 n-3, α-linolenic acid; C20:5, n-3, eicosapentaenoic acid; C22:6 n-3, docosahexaenoic acid.

$p = 0.011$); the reduction in C20:0 were associated with the decrease in body weight ($r = 0.651$, $p = 0.003$), BF ($r = 0.557$, $p = 0.013$); the reduction in C22:0 were associated with the decrease in body weight ($r = 0.587$, $p = 0.008$), BMI ($r = 0.509$, $p = 0.026$), BF ($r = 0.656$, $p = 0.002$) and FMI ($r = 0.472$, $p = 0.041$); and the decrease in total SFAs was related to the reduction in body weight ($r = 0.620$, $p = 0.005$), BMI ($r = 0.485$, $p = 0.036$), BF ($r = 0.615$, $p = 0.005$). Meanwhile, the decrease in C16:1 n-7 was related to a reduction in body weight ($r = 0.529$, $p = 0.02$), BMI ($r = 0.502$, $p = 0.029$), BF ($r = 0.591$, $p = 0.008$), and the decrease in total MUFAs was related to the reduction in body weight ($r = 0.477$, $p = 0.039$). Regarding serum PUFAs, the decrease in C20:3 n-6 was positively correlated with reduction of body weight ($r = 0.626$, $p = 0.004$), BMI ($r = 0.497$, $p = 0.031$), BF ($r = 0.542$, $p = 0.016$), and the decrease in C20:4 n-6 was positively correlated with reduction of left baPWV ($r = 0.501$, $p = 0.029$). In addition, the increase in C22:6 n-3 were associated with the increase of right ABI ($r = 0.495$, $p = 0.031$) and the reduction of left baPWV ($r = -0.489$, $p = 0.034$), respectively.

Table 3
Correlation between fatty acids and body compositions, anthropometric and arterial stiffness.

FA Para *r*	C14:0	C18:0	C20:0	C22:0	SFA	C16:1 n-7	MUFA	C20:3 n-6	C20:4 n-6	C22:6 n-3
Body weight	0.373	0.606**	0.651**	0.587**	0.620**	0.529*	0.477*	0.626**	−0.397	0.451
BMI	0.226	0.430	0.405	0.509*	0.485*	0.502*	0.253	0.497*	−0.381	0.431
Body Fat	0.477*	0.567*	0.557*	0.656**	0.615**	0.591**	0.294	0.542*	−0.341	0.447
FMI	0.324	0.325	0.262	0.472*	0.388	0.442	0.021	0.374	−0.230	0.311
baPWV(L)	−0.146	−0.308	−0.404	−0.439	−0.443	−0.153	−0.217	−0.178	0.501*	−0.489*
ABI (R)	−0.334	0.205	0.235	0.121	0.227	0.062	0.319	0.121	−0.420	0.495*

Note: *$p < 0.05$ and **$p < 0.01$ significant correlation between serum FAs and parameters under intervention condition; n = 19.
FA, fatty acid; *r*, correlation coefficient; Para, parameter.

4. Discussion

Obesity is extremely prevalent in developed countries and has even reached up to the alarming level in developing countries,[30] and regulating the balance of serum FA profile by lifestyle interventions can prevent or relieve the development of obesity and relevant disorders in obese population.[31,32] Higher energy intake and less energy expenditure can establish a positive energy balance and increase body mass (energy storage), of which 60–80% is usually stored as body fat. The residual glucose, amino acids and short-chain fatty acids/medium-chain fatty acids metabolized by the liver will form triglycerides, which are transported by apolipoprotein to peripheral blood and stored in peripheral tissues and organs together with triglycerides formed by long-chain fatty acids in chylomicrons.[33,34] According to previous reports, serum SFAs, especially C16:0 and C18:0, are positively associated with the mass of trunk fat and visceral adipose tissues,[35] so higher intake of SFAs could increase body fat and inflammatory biomarkers for inducing obesity and obesity-related diseases.[36,37] Furthermore, the widely advocated Mediterranean diets play a crucial role in maintaining body weight and preventing obesity,[30] because MUFAs in these diets can promote lipid oxidation and suppress lipogenesis.[22,38] However, the positive effect of MUFAs may be weakened or displaced by SFAs with deleterious properties,[39] and the higher serum MUFA levels in obese subjects are related to the development of abdominal adiposity, fatty liver and insulin resistance.[40] Therefore, it is essential to reduce serum SFAs and MUFAs by therapeutic approaches in obese populations. In the present study, both exercise training and dietary restriction have been applied for promoting energy expenditure to exceed energy intake, which will establish a state of negative energy balance and reduce body fat in obese subjects. Triglycerides stored in peripheral tissues and organs are hydrolyzed to glycerol and fatty acids by adipose triglyceride lipase. Fatty acids are then transferred to mitochondria for degradation by the β-oxidation pathway.[33,34] The 4-week combinatorial intervention results in prominent changes in body compositions and the decrease in SFAs and MUFAs, especially C14:0, C15:0, C18:0, C20:0, C22:0, total SFAs, C16:1 n-7, C18:1 n-9, C20:1 n-9 and total MUFAs, which is consistent with previous reports for more than 2-month body weight management.[26–28,41] The 4-week closed residential camp (combinatorial intervention with exercise training and dietary restriction) can rescue abnormal serum SFA and MUFA levels accompanied with the reduction of body compositions. Furthermore, the reduction of C14:0, C18:0, C20:0, C22:0, total SFAs and C16:1 n-7 was observed to be associated with the decrease in BF. These results suggest that 4-week combinatorial intervention-induced BF reduction may contribute to the reduction of serum SFA and MUFA in obese adolescents. Taking into the limited consecutive free time of adolescent students with studying assignments, a closed residential camp with large volume of exercise training and dietary restriction during summer vacation was further explored based on its significant benefits of previous intervention program on weight management and redox homeostasis in obese adolescents.[29] Although exercise training during summer vacation is large volume, total exercise intensity is still at the moderate level, which is highly suitable for the people with strong desire to lose body weight within a relatively short-term interventional period. Therefore, 4-week intervention in the present study was chosen to explore its effect on serum fatty acids, body fat and arterial stiffness in obese adolescents, whose positive efficiency for body weight loss can be beneficial for the popularization among other obese populations with limited time, not only obese adolescent students.

The contentious harm of n-6 PUFA lies in competition with n-3 PUFA for desaturase and elongase involved in the biosynthesis of PUFAs.[42] Obese population has higher serum n-6 PUFA and lower n-3 PUFA levels than those of the population with normal body weight, thereby leading to the accumulation of adipose tissues, the occurrence of pro-inflammation, and the development of cardiovascular diseases.[43,44] Moreover, baPWV and ABI can provide a comprehensive assessment for systemic and peripheral arterial stiffness, and abnormal high baPWV and low ABI indicate high risks of cardiovascular accidents.[45] So, it is essential to normalize serum n-6 PUFA, n-3 PUFA, baPWV and ABI by therapeutic approaches in obese populations. C20:3 n-6 as a key inflammation marker can predict the risk of visceral fat accumulation and has a strong correlation with inflammatory markers in obesity[46,47]; C20:4 n-6 is a substrate for some pro-inflammatory metabolites that are the common denominators for CVD and MS[2,48]; C22:6 n-3 has the positive effect on antioxidant, anti-inflammatory and antithrombotic parameters.[49] In the current study, 4-week intervention-induced reduction of C20:3 n-6 and C20:4 n-6 was associated with the decrease of body fat and left baPWV, respectively; in contrast, the increase of C22:6 n-3 was associated with the decrease of left baPWV and the increase of right ABI, suggesting the contribution of reduced serum C20:3 n-6 by decreased BF, and reduced arterial stiffness in obese adolescents upon 4-week intervention by reduced C20:4 n-6 and increased C22:6 n-3. Based on above analysis, the reduction in body fat mass is beneficial for the decreased levels of C14:0, C18:0, C20:0, C22:0, total SFAs, C16:1 n-7 and C20:3 n-6, and the decreased level of C20:4 n-6 and increased level of C22:6 n-3 is beneficial for the reduction of arterial stiffness, suggesting that the reduction of body fat induced by a 4-week combinatorial exercise training and dietary restriction program could explain the partial contribution to the improvement of serum FA profile. Additionally, the improvement of serum FA profile could partially contribute to the reduction of arterial stiffness in obese adolescents.

5. Conclusion

The 4-week closed residential camp (combinatorial intervention

with exercise training and dietary restriction) is a promising and effective strategy for improving serum FA profile along with the reduction of body fat and arterial stiffness in obese adolescents.

Author statement

Lei Xu: Acquisition of data, Analysis and/or interpretation of data, Drafting the manuscript, Revising the manuscript critically for important intellectual content; **Xiaoyu Zou**: Acquisition of data; **Zhiqiang Gao**: Acquisition of data; **Caifeng Mao**: Acquisition of data; **Hang Su**: Acquisition of data; **Chunyan Li**: Conception and design of study, Analysis and/or interpretation of data, Drafting the manuscript, Revising the manuscript critically for important intellectual content; **Ning Chen**: Conception and design of study, Analysis and/or interpretation of data, Drafting the manuscript, Revising the manuscript critically for important intellectual content; Approval of the version of the manuscript to be published (the names of all authors must be listed).

Declaration of competing interest

All authors have no conflicts of interest relevant to this article.

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