

This author's PDF version corresponds to the article as it appeared upon acceptance. Fully formatted PDF versions will be made available soon.

Effect of a walnut-based low carbohydrate diet on fat redistribution and visceral fat accumulation in people with central obesity: A randomized controlled trial

doi: 10.6133/apjcn.202607/PP.0008

Published online: July 2026

Running title: W-LCD on fat redistribution and VFA

Qingqing Dong MSc^{1†}, Xiaohan Tan MSc^{2†}, Ping Feng MSc², Qianqian Tian MSc³, Qing Jiang MSc³, Xiaohua Wang MD, PhD⁴, Lili Wang MSc⁵

¹Department of Gastroenterology, The First Affiliated Hospital of Soochow University, Suzhou, China

²Department of Nursing, The First Affiliated Hospital of Soochow University, Suzhou, China

³Department of Endocrinology, The First Affiliated Hospital of Soochow University, Suzhou, China

⁴Department of Cardiology, The First Affiliated Hospital of Soochow University, Suzhou, China

⁵Department of Urology, The First Affiliated Hospital of Soochow University, Suzhou, China

[†]Both authors contributed equally to this manuscript

Authors' email addresses and contributions:

Qingqing Dong: dqxkyddq@163.com (20215231050@stu.suda.edu.cn)

Contribution: data collecting, analyzing and initial draft writing

Xiaohan Tan: 20235231055@stu.suda.edu.cn

Contribution: methodology and data collecting

Ping Feng: fp1981@foxmail.com (fengping88@suda.edu.cn)

Contribution: data collecting

Qianqian: 20235231052@stu.suda.edu.cn

Contribution: data collecting

Qing Jiang: 99279407@qq.com (jiangqing90@suda.edu.cn)

Contribution: data collecting

Xiaohua Wang: sxwang2001@163.com (wangxiaohua@suda.edu.cn)

Contribution: study designing

Lili Wang: wanglili83476@suda.edu.cn

Contribution: providing funding, study designing, methodology, source and supervision.

Corresponding Author: Ms Lili Wang, Department of Urology, The First Affiliated Hospital of Soochow University, Suzhou 215006, China; No. 899 Pinghai Road, Suzhou City, Jiangsu Province, China. Tel: +86-18896520693. Email: wanglili83476@suda.edu.cn

ABSTRACT

Background and Objectives: The aim of this study was to determine whether a walnut-based low carbohydrate diet (w-LCD) promotes fat redistribution and reduces visceral fat accumulation in people with central obesity (CO). **Methods and Study Design:** This study was a 12-week prospective randomized controlled trial. CO participants were recruited and assigned to the w-LCD group or high-carbohydrate, low-fat diet (HCD) group. According to "2021 Guidelines for Medical Nutrition Therapy of Overweight and Obesity", the HCD program was constructed; based on the HCD program, the w-LCD program was formed by replacing 150 grams/day of staple food with walnuts rich in healthy fat. The visceral fat area (VFA), trunk to leg fat ratio (TLR), and waist-to-hip ratio (WHR) were assessed at baseline and 12 weeks. **Results:** The Group $\times$ Time interaction effects were significant for TLR ($\eta^2 = 0.071$, $p = 0.039$) and VFA ($\eta^2 = 0.093$, $p = 0.014$) with moderate effect, and body mass index (BMI, $\eta^2 = 0.168$, $p = 0.001$) with large effect. Although comparison between groups revealed no significant on TLR, VFA and BMI at 12 weeks, comparison within groups showed all decreased with large effect on TLR ($\eta^2 = 0.174$, $p = 0.001$), VFA ($\eta^2 = 0.393$, $p < 0.001$) and BMI ($\eta^2 = 0.399$, $p < 0.001$) in the w-LCD group over time. **Conclusions:** A w-LCD achieves a significantly greater and faster fat reduction over time compared to the control group, providing a solid evidence-based foundation for dietary management in people with CO.

Key Words: low carbohydrate diet, walnut, fat redistribution, visceral fat accumulation, central obesity

INTRODUCTION

The prevalence of central obesity (CO) is showing a rapidly increasing trend globally.¹ Over the past four decades, the prevalence of CO has increased from 31.3% to 48.3% globally² and from 18.6% to 37.4% in China.³ The important epigenetic feature of CO is the accumulation of excess fat in the abdomen or trunk, which is highly correlated with detrimental visceral fat and decreased subcutaneous fat of legs.⁴⁻⁶ Existing evidence suggests that visceral or trunk fat, but not legs fat in CO is more strongly associated with metabolic disease, including type 2 diabetes (T2D), and cardiovascular diseases (CVD) than overall obesity.⁷⁻¹⁴ In cases of excessive energy, if fat is stored in the legs rather than in trunk or visceral fat depots, the risk of metabolic diseases can be reduced.¹⁵ A national health and screening survey showed that for an increase of one unit in the trunk-to-leg fat ratio (TLR), adolescents had an increase in homeostatic model of assessment of insulin resistance of 0.6 (95%CI: 0.4 - 0.8), in systolic

blood pressure of 4.6 mmHg (95%CI: 1.7 mmHg - 7.4 mmHg), and in diastolic blood pressure of 5.0 mmHg (95%CI: 2.0 mmHg - 7.9 mmHg).¹⁶ Thus, when searching for management strategies for CO populations, reducing visceral fat and changing fat distribution can be targeted accordingly.

Carbohydrate-insulin model (CIM) refers to an increase in the proportion of dietary carbohydrate over time, which stimulates the release of insulin from pancreatic β -cells.¹⁷ Since adipose tissue is very sensitive to insulin, the consumed carbohydrate is more likely to enter adipose tissue, then synthesized fatty acids and triglycerides, stored in adipose tissue, leading to total fat deposition.¹⁷⁻²¹ More importantly, compared with the fat in hips or legs, visceral or trunk adipose tissue has a greater capacity to take up carbohydrates, resulting in excessive accumulation of visceral or trunk fat and abnormal fat distribution.²² Therefore, CIM suggests that lowering the ratio of dietary carbohydrate to fat without changing protein or calorie intake can reduce insulin secretion.²³

Walnuts are the most consumed food by our population and are easy to chew and eat. It is rich in omega-3 polyunsaturated fatty acid (ω -3 PUFA),^{24, 25} which is currently recognized as a healthy nutrient that can increase the quantity of *bifidobacteria* and *lactobacilli* in the gut, which contribute to the production of short-chain fatty acids (SCFAs).²⁶ Then SCFAs bind to G protein-coupled receptor 43 (GPR43) and G protein-coupled receptor 40 (GPR40) of L cells in the ileum and colon, and promote the secretion of glucagon-like peptide-1 (GLP-1) and Peptide YY (PYY) from L cells.^{27, 28} GLP-1 and PYY has a lower ability to promote insulin secretion by pancreatic β cells, thus there is a relative reduction of carbohydrate ingestion by visceral or trunk fat adipose tissue which may be beneficial in improving fat redistribution.²⁹

Therefore, adjusting the composition of dietary nutrition by increasing the proportion of healthy fats such as ω -3 PUFA rich foods while reducing the proportion of carbohydrates, may improve fat redistribution and decrease visceral fat in people with CO. A randomized controlled trial (RCT) showed that after 3 months of intervention, insulin-resistant people in the monounsaturated fatty acids-rich diet group (47% carbohydrate, 38% fat, 15% protein) had a lower TLR than that of the carbohydrate-rich diet group (65% carbohydrate, 20% fat, 15% protein) (2.5 ± 0.2 vs. 2.1 ± 0.2 , $p < 0.05$).³⁰ Another 4w RCT in obese people with T2D found that the visceral fat area (VFA) in low carbohydrate, high fat diet group (LCD, 25% protein, 39% carbohydrate, 35% fat) was reduced by 40cm^2 ($p < 0.05$) from baseline; while there was no difference in the high carbohydrate, low fat diet group (HCD: 26% protein, 62% carbohydrate, 10% fat).³¹ However, the above studies did not explore whether the walnut-

based low carbohydrate (w-LCD) improves the TLR and VFA in the CO population. In addition, there is lack of studies on the effect of w-LCD on TLR and VFA in China.

Therefore, based on the above, the hypothesis of this study is that w-LCD can reduce visceral fat accumulation and promoting fat redistribution while losing weight of CO people in China.

MATERIALS AND METHODS

Study design

This was a 12-week RCT that recruited CO participants by publishing recruitment advertisements at the Physical Examination Centre of the First Affiliated Hospital of Soochow University and the We Chat platform from December 2022 to October 2023. The recruited participants were randomly assigned to either the w-LCD group or the HCD group by a computer-generated random number table, which was hidden by a non-principal researcher. So the method for allocation is blinded. This study was approved by the Ethics Committee of Soochow University (The approval number is SUDA20221228H09 and see Figure 1 in the supplementary materials for details) and registered with the China Clinical Trial Registry (ChiCTR2300068330). All participants signed informed consent, and all methods were performed in accordance with the Declaration of Helsinki.

Study participants

The inclusion criteria for participants in this study were (1) aged 18 to 60 years; (2) meeting the diagnostic criteria for CO of the 2022 edition of the Chinese Expert Consensus on Obesity Prevention and Control for Chinese Residents,³² which were waist circumference (WC) ≥ 90 cm for men and ≥ 85 cm for women, and $24 \text{ kg/m}^2 \leq \text{body mass index (BMI)} \leq 32.5 \text{ kg/m}^2$; (3) good ability of language expression and understanding; (4) voluntary participation. Exclusion criteria for participants were (1) bariatric patients within the past 6 months; (2) nuts allergy; (3) $<45\%$ of dietary CHO to total calories in daily diet as assessed by three-day diet diary;³² (4) eating disorder; (5) gastrointestinal disorders; (6) using diuretics, steroids, beta-blockers, or any medication that may affect glucose metabolism; (7) pregnancy or breastfeeding; (8) microvascular or macrovascular complications; (9) neoplasms and severe organ diseases such as heart failure, liver, kidney and brain insufficiency; and (10) participating in several studies at the same time. Participants were withdrawn from the study if they were (1) unable to implement the w-LCD program ≥ 4 day/w in the w-LCD group and the HCD program ≥ 4 day/w in the HCD group during follow-up;³³ (2) stopped continuing due to adverse

reactions to the w-LCD or HCD program; (3) suffering from a major life event;³⁴ and (4) withdrawal or loss of follow-up due to subjective intention.

Sample size calculation

The formula for estimating the sample content according to the comparison of the means of two independent samples is:³⁵

Evidence from the relevant literature showed that the mean differences and standard deviations for the TLR were 0.4 and 0.2,³⁰ waist-to-hip ratio (WHR) were 0.03 and 0.02,³⁶ and VFA were 39.3 and 10.331 in the LCD group and HCD group, respectively. Therefore, at the $\alpha = 0.05$ and $\beta = 0.20$ levels, the table was $t_{\alpha/2} = 1.96$ and $t_{\beta} = 0.84$, respectively, we calculated that there were 11 or 20 or, 28 cases for each group, respectively. Therefore, the sample size for this study was 28. Considering a 20% dropout rate, at least 35 cases in each group were needed. According to the actual condition, this study is expected to recruit 40 cases in each group.

Intervention

Before the intervention, all participants underwent a 1-week purging period, avoiding all nut supplementation (walnuts, almonds, peanuts, etc.).

HCD group

The 2021 Chinese Guidelines for Medical Nutrition Therapy for Overweight and Obesity recommends a reduction of 500~1000 kcal daily on the target energy intake, i.e., 1200~1400 kcal for men and 1000~1200 kcal for women. In addition, the HCD program for the HCD group is a five-word formula that was developed by our team based on the 2021 Chinese Guidelines for Medical Nutrition Therapy of overweight and obesity³⁷ and expert consultation. The specifics of the five-word formula included 500 g/day of vegetables, 100 g/meal of staple and 200 g/day of fruit, 3 spoons/day of vegetable oil and 30 g/day of oat bran, 4 cups/day of water (250 mL for each cup), 5 types/day of protein-rich foods (50 g of fish or shrimp, 50 g of dried Tofu, 220 mL whole milk, 25 g lean meat, an egg) and 5 g/day of salt).

w-LCD group

Walnuts were used to replace the staple food for 50 grams/meal of the w-LCD program and the rest was the same as the regimen for the HCD group. Walnuts were uniformly purchased (Fenyang Xiangyuan Souvenir Co., Ltd, Fenyang, China). Although walnuts were purchased

and provided free of charge for the first two weeks, they were funded by the participants due to funds were limited for the next 10 weeks, and we packaged them into 50 g/bag for men and 45 g/bag for women and then distributed them in the w-LCD group. The packaged walnuts were distributed every two weeks.

The time of consuming walnuts was with or between meals. Walnuts can be eaten raw or dry-roasted processed, avoiding seasoning processes such as salt-baked, five-spice, or caramelized. All participants received five follow-ups (baseline, 1 week, 4 weeks, 8 weeks, and 12 weeks) which included collecting information on the implementation of the dietary regimen for dietary adherence; asking them if they had, any adverse reactions or major events and excluding participants with poor adherence to the diet program.

Outcomes

Body compositions

TLR, VFA and BMI were measured using the Inbody720 Body Composition Analyser (Biosbody Co., Ltd., Korea) by bioelectrical impedance-based analysis which can provide an accurate analysis of body composition and its correlation coefficient with the gold standard Dual Energy X-Ray Absorptiometry exceeds 0.98.^{38, 39} To ensure the accuracy of the results, it was recommended that participants were fasting in the morning, emptied bowels, refrained from strenuous exercise for 2 hours, and stood still for about five minutes before the test.³⁹ Before testing, participants take off their jackets and only wear a piece of thin clothing without electronic items and metal jewelry; remove their shoes and socks; stand barefoot on the apparatus, and align their heels with the origin to ensure full contact between the feet electrode positions. The researcher input the basic information of participants and recorded their weights shown on the screen of the machine. The participants grabbed the handle with both hands, straightened their arms to the sides, clicked to start detection, and waited for about 2 minutes. In addition, participants were instructed not to keep their bodies still. After testing, the amount of trunk fat (g) and leg fat (g), VFA (cm²), BMI (kg/m²) and body fat percentage (BF%) were recorded and the TLR was the ratio of trunk fat to leg fat.

WHR

The measurement of WC required participants to relax their waist naturally and keep their feet about 30cm apart. Then the researcher wrapped a plastic tape measure around the navel and took the measurement at the end of exhalation. Hip circumference (HC) was measured along

the pubic symphysis and gluteus maximus muscle. The average of the two measurements was taken as the final value and was accurate to 0.1 cm. WHR was the ratio of WC to HC.

Fasting GLP-1, PYY and C-peptide

Participants were informed that they should not change their diet before blood collection and should not consume alcohol for 24 hours.⁴⁰ On the fasting at least 8 h but no more than 16 h in the morning,⁴⁰ 5ml of cubital venous blood was drawn from participants at the Molecular laboratory of the School of Nursing of Soochow University, and immediately placed in separating glue and procoagulant tubes. Within 30 minutes of collection, blood samples are centrifuged for 10 minutes on 4°C and 9000 rpm/min. Subsequently, the centrifuged serum was extracted and stored in a -80°C ultra-low temperature refrigerator. Avoiding serum from being repeatedly freeze-thawed. After all participants had completed the intervention, GLP-1 and C- peptide which represents the level of insulin secretion by pancreatic beta cells were detected by the GLP-1 kit and the C- peptide kit (Bostik Biotechnology Co., Shenyang, China), respectively, based on the Enzyme Linked Immunosorbent Assay (ELISA). The normal ranges for GLP-1, PYY and C-peptide are 0.375 pmol/mL-12 pmol/mL, 5pg/ml-160pg/ml and 0.25ng/mL-8 ng/mL, respectively.

Dietary records

The 3-day dietary record forms were collected from participants at baseline, 1 week, 4 weeks, 8 weeks, and 12 weeks of the intervention, respectively. The researchers provided food weight estimation charts to the participants to help them assess the specific weight of the foods. If some participants still could not accurately estimate the amount of food, an electronic balance scale was provided. Instructed participants to record the time of eating, the names of food intake, the compositions of mixed food such as dumplings, buns and the estimated weight (e.g. 50 g) during 2 workdays and 1 rest day. Then, the Fuhua Nutrition Calculator (V2.7.6.10, Beijing, China) was used to calculate the energy proportion of three macronutrients, which was used to assess the adherence of participants to the corresponding dietary regimen.

Physical activity

The physical activity for the week prior of overweight and obese population was evaluated with the International Physical Activity Questionnaire (IPAQ) at baseline and 12weeks.⁴¹ This questionnaire collected the frequency and total daily duration of walking, moderate exercise,

and vigorous activity. The intensity of physical activity was quantified using metabolic equivalent (MET) values: 3.3 METs for walking, 4.0 METs for moderate activity, and 8.0 METs for vigorous activity. The PA-MET score was computed using the formula: MET level $\times$ minutes of activity $\times$ weekly frequency. Based on these calculations, exercise intensity was categorised into three levels: low (≤ 600 MET-min/week), middle (600–1500 MET-min/week) and high (≥ 1500 MET-min/week).⁴² In addition, weekly exercise time = minutes of daily activity $\times$ weekly frequency.

Statistical analyses

Statistical analyses were performed using IBM SPSS 25.0 (Inc, Chicago, IL, USA). $p < 0.05$ for two-sided was considered statistically significant.

(1) Description of sociodemographic, clinical and nutritional data: continuous variables were expressed as mean $\pm$ standard deviation (SD) if they were normally distributed, otherwise quartiles M (P25, P75); categorical variables were expressed as frequency (%);

(2) Repeated-measures ANOVA was applied to evaluate the continuous outcomes (TLR, WHR, VFA, BMI). The between-subject factor was Group (w-LCD vs. HCD) and the within-subject factor was Time (baseline vs. 12 weeks). Sphericity was checked using Mauchly's test; since the within-subject factor had only two levels, the sphericity assumption was inherently satisfied ($df = 1$). When a significant Group $\times$ Time interaction was detected, standard simple effects analyses with Bonferroni adjustments were performed, Main effects of Time and Group were reported for non-significant interactions. Effect sizes are reported as partial eta squared (η^2) with the following classification to define small ($\eta^2 \leq 0.01$), medium ($0.01 < \eta^2 \leq 0.06$), and large ($\eta^2 \geq 0.14$) effect sizes.⁴³

(3) Continuous variables, including the GLP-1 and C-peptide between the groups were tested by a two-sample independent t-test if they conformed to normal distribution; otherwise the Mann-Whitney test;

(4) The categorical variables such as diarrhoea were tested by the chi-square test;

(5) The intention to treat (ITT) analysis for TLR, WHR and VFA was performed to ensure the reliability of the study results. All randomly allocated 80 participants were retained in their original randomized groups for ITT analysis, irrespective of intervention adherence or study dropout. Missing endpoint outcome data from withdrawn subjects were imputed using the last observation carried forward (LOCF) approach based on their latest available measured values.

RESULTS

Study participants

This study recruited 80 CO populations, who were randomly divided into the w-LCD group ($n = 40$) and the HCD group ($n = 40$). There were 5 participants with a dropout rate of 12.5% (1 lost to follow-up, 4 poor adherence) in the w-LCD group and 11 participants with a dropout rate of 27.5% (1 poor adherence, 1 pregnant, 2 automatic exits, 7 lost to follow-up) in the HCD group who withdrew from the study. Finally, 35 participants in the w-LCD group and 29 participants in the HCD group were analyzed for outcomes in this study (Figure 1).

The mean age of the participants was (37.3 ± 9.80) years; 35 (54.7%) were female. The general characteristics of the participants in the two groups are shown in Table 1. There were no significant differences ($p > 0.05$) between the two groups. In addition, there were no statistically significant differences ($p > 0.05$) in the demographic data, clinical data, nutrition data and body composition between participants who completed the follow-up and those who did not (Supplementary Table 1).

Dietary adherence

Repeated measure ANOVA revealed a significant Group $\times$ Time interaction effect on fat ($\eta^2 = 0.290$, $p < 0.001$) with a large effect size as shown in Table 2. Group effects on fat ($\eta^2 = 0.155$, $p = 0.002$) and carbohydrates ($\eta^2 = 0.184$, $p = 0.001$) were significant with large effect sizes. In addition, the intake of energy, fat, and carbohydrates, as well as protein, decreased over time in both groups. In addition, the ITT analysis was in agreement with the above findings in general except for the Group $\times$ Time interaction of fat ($\eta^2 = 0.029$, $p = 0.081$) showing no statistical difference (Supplementary Table 2).

Furthermore, the carbohydrate and fat energy ratios were about 55% and 25% in both groups at baseline, respectively. During dietary intervention, the carbohydrate and fat energy ratios were to be maintained at about 39% and 42%, respectively, in the w-LCD group, which was in accordance with the w-LCD protocol; the carbohydrate and fat energy ratios were to be maintained at about 57% and 24%, respectively, in the HCD group, which was in accordance with the HCD protocol (Figure 2).

Effect of w-LCD on body composition in CO populations

Repeated measure ANOVA revealed significant Group $\times$ Time interaction effect on TLR ($\eta^2 = 0.071$, $p = 0.039$) and VFA ($\eta^2 = 0.093$, $p = 0.014$) with moderate effect sizes, and on BMI ($\eta^2 = 0.168$, $p = 0.001$) with large effect sizes as shown in Table 3, which confirm that the w-

LCD group achieved a statistically superior trend in lowering TLR, VFA, and BMI over the 12-week period compared to the HCD group. In addition, there was a tendency of significant differences in the Group $\times$ Time interaction effect of WHR ($\eta^2 = 0.052$, $p = 0.072$), indicating that w-LCD has the tendency to potentially reduce WHR.

Although comparisons between groups revealed no significant on TLR, VFA and BMI at 12 weeks, comparisons within groups revealed a reduction in both TLR ($\eta^2 = 0.174$, $p = 0.001$), VFA ($\eta^2 = 0.393$, $p < 0.001$) and BMI ($\eta^2 = 0.399$, $p < 0.001$) in the w-LCD group with large effect sizes compared with those of the HCD group. In addition, WHR ($\eta^2 = 0.147$, $p = 0.002$) and BF% ($\eta^2 = 0.100$, $p = 0.007$) decreased over time with moderate effect sizes in both groups.

Except for the Time $\times$ Group interaction for WHR ($\eta^2 = 0.056$, $p = 0.037$) with moderate effect sizes being significant and the decrease in WHR ($\eta^2 = 0.171$, $p < 0.001$) with moderate effect sizes over time in the HCD group, the other results of the ITT analysis were in agreement with the above findings in general (Supplementary Table 3).

Effect of w-LCD on GLP-1 and C-peptide in CO populations

At 12 weeks, The GLP-1, PYY and C-peptide levels of all participants fluctuated within the normal range, but GLP-1 and PYY was higher [GLP-1: w-LCD 2.10 (1.84, 2.69) pmol/L vs. HCD 1.59 (0.86, 2.14) pmol/L, $Z = -2.343$, $p = 0.019$; PYY: w-LCD (33.1 ± 5.50) pg/ml vs. (26.8 ± 5.93) pg/ml, $t = 3.663$, $p = 0.001$], and C-peptide was lower [w-LCD 1.11 (0.73, 1.29) vs. HCD 1.61 (1.01, 3.64), $Z = -2.242$, $p = 0.025$] in the w-LCD group (Figure 3).

DISCUSSION

The walnut-based low carbohydrate diet (w-LCD) is now an effective nutritional intervention to change fat distribution. In this study, after 12 weeks of intervention, it was found that w-LCD reduced TLR and VFA, and had a decreasing trend on WHR in CO populations. Meanwhile, it could reduce BMI.

W-LCD promoted fat redistribution in CO populations

Fat accumulation is a double-edged sword for the body. On the one hand, trunk fat accumulation has a negative impact on the body, and on the other hand, hip and leg fat accumulation has a protective effect on the body.⁴⁴ A prospective cohort study⁴⁵ including 21,472 people with T2D with 7.7 years of follow-up found that the risk of CVD and death in the highest quartile of trunk fat was higher than those in the lowest quartile of trunk fat,

respectively; while, the risk of CVD and death in the highest quartile of hip and leg fats were lower than in the lowest quartile of hip and leg fats, respectively. Therefore, lowering trunk fat to hip or leg fat may prevent the development of CO-related metabolic diseases.

The TLR, which was measured simply, directly and accurately by bioelectrical impedance-based analysis, is a parameter that is independent of overall obesity and can be used as a good indicator of fat distribution.⁴⁶⁻⁴⁸ However, there are relatively few studies regarding the effect of w-LCD on TLR in CO populations at present. Paniagua et al. performed a 3 months RCT in insulin-resistant populations and showed that the w-LCD group had a lower TLR than that of the HCD group (2.1 ± 0.2 vs. 2.5 ± 0.2 , $p < 0.05$).³⁰ In this study, it was also found that the TLR decreased more in the w-LCD group than in the HCD group. Although the lack of statistical significance at 12-week because both study groups received active dietary interventions, the robust Group $\times$ Time interaction provides the proof that the overall fat reduction over the 12 weeks was significantly higher and faster in the w-LCD group.

In addition, WC can reflect visceral fat content and HC can reflect hip fat content.⁴⁹ Therefore, the WHR, which is widely used in clinical practice, can be used as a simple and inexpensive alternative for indirect estimation of fat distribution.⁵⁰⁻⁵² Farnoosh et al. found no significant difference in WHR between low and medium carbohydrate diets,⁵³ which is inconsistent with this study. It was found that the w-LCD had a tendency to lower WHR in CO populations after 12 weeks of intervention, which may confirm that the dietary program might promote visceral fat redistribution to hip fat. Mei et al. found that the Mediterranean diet combined with LCD significantly reduced WHR ($p < 0.05$) in overweight people.³⁶ Similarly, Magnus et al. found a greater decline of WHR in CO populations in the LCD group compared to conventional diets (-0.05 vs. -0.02).⁵⁴ The reason for the inconsistency may be related to the fact that both groups in the Farnoosh et al. study restricted carbohydrates to different degrees (carbohydrate energy ratio: intervention 15% vs. control 39%), which can both lead to a decrease in WHR.

W-LCD reduced visceral fat accumulation in CO populations

The International Atherosclerosis Society and the Chair of the International Cardiometabolic Risk Task Force on Visceral Obesity jointly stated that visceral adiposity rather than overall obesity is an independent risk factor for the risk of CVD, metabolic disease and death, suggesting that visceral fat may be a more important indicator of the effectiveness of obesity management strategies.⁵⁵ Although the Expert Consensus on Prevention and Control of Obesity in Chinese Residents used WC as a diagnostic criterion for visceral obesity, this

indicator can only estimate the amount of abdominal visceral fat.³² The method fails to exactly reflect the degree of visceral obesity when the organism is suffering from conditions such as ascites.⁵⁶ Instead, the measurement of VFA is more convenient, intuitive and accurate to reflect the level of visceral fat deposition in the human abdomen.⁵⁷

In this study, Although the lack of statistical significance at 12-week, the obvious Group $\times$ Time interaction provides the proof that the overall fat reduction over the 12 weeks was significantly higher and faster in the w-LCD group. It was found that VFA decreased significantly from 113 cm² at baseline to 97 cm² at 12 weeks in the w-LCD group, which is lower than the threshold of visceral fat obesity in Asia ($\leq 100\text{cm}^2$).⁵⁸ In addition, the decrease of VFA in the w-LCD group was higher than that of the HCD group, indicating that w-LCD effectively reduced visceral fat deposition in people with CO. In a 2-month RCT, it was found that very low-energy ketogenic diets (600-800 kcal/d energy, < 50g/d carbohydrate) reduced visceral fat deposition from $170.8 \pm 58.0\text{cm}^2$ to $131.5 \pm 47.7\text{cm}^2$ ($p < 0.05$) in obese people.⁵⁹ The result of Yoh et al. similarly found that VFA decreased by 40 cm² ($p < 0.05$) from baseline in the LCD group (1000 kcal/d energy, 40% carbohydrate, 35% fat) among obese people with T2D after 4 weeks of intervention.³¹ Thus, LCD could reduce visceral fat deposition, but the reduction of VFA in this study was less than that of German and Yoh et al. On the one hand, it may be related to the relatively high carbohydrate energy ratio in this study, which leads to a relative increase of insulin secretion and promotes the entry of carbohydrates into adipose tissue, thus resulting in the accumulation of visceral fat. On the other hand, it may be related to the fact that the overweight population included in this study had lower baseline visceral fat content, which contributed to a lower decline of VFA than that of obese populations. Moreover, VFA decreased significantly in the HCD group from baseline, which may be related to that all participants received dietary fiber (DF) which is rich in oat bran supplementation in this study, which had been reported to reduce insulin secretion and thus relatively decrease visceral fat deposition.⁶⁰

W-LCD induced fat redistribution and visceral fat reduction and its cross sectional relationship with 12-week fasting C peptide and GLP 1 and PYY levels

According to CIM, lowering the ratio of carbohydrates to ω -3 PUFA can inhibit C-peptide and promote GLP-1 production in CO populations, which in turn can lower weight, reduce visceral fat deposition and TLR.¹⁷ Thus, we examined the GLP-1 and C-peptide levels at 12 weeks in both groups.

C-peptide

The physiological pathways through which w-LCD links to altered fat distribution remain to be fully elucidated. CIM suggests that fat cells are the core of the obesity etiology, rather than a site for passive storage of excess energy. Although many factors affect energy storage in fat cells, insulin plays a dominant role in controlling its anabolism.¹⁷ Insulin mainly stimulates glucose intake and inhibits the release of fatty acids in visceral adipose tissue, thereby promoting visceral fat deposition and causing abnormal fat distribution.⁶¹ Torbay et al. injected insulin or saline into rats daily and the fat mass of rats receiving insulin increased after 4 weeks of intervention.⁶² Consistent with this finding, mice with genetically reduced insulin secretion had higher energy expenditure and did not become obese due to diet.⁶³⁻⁶⁵ Furthermore, visceral fat is more sensitive to insulin and more capable of ingesting carbohydrates than hip or leg fat, which may lead to accumulation of visceral fat and abnormal fat distribution.^{22, 66}

Thus, the inhibition of insulin secretion may lower weight, reducing visceral fat deposition and altering fat distribution. The effect of the carbohydrate amount is the most significant among the many factors influencing insulin secretion.¹⁷ A meta-analysis of 17 RCTs involving 1141 obese patients showed that the LCD group had lower fasting insulin levels than the HCD group (MD: -2.24 micro IU mL⁻¹, 95%CI: -2.65, -1.82).⁶⁷ Marti et al. also found that fasting insulin levels decreased from baseline ($-338 \pm 398 \mu\text{U/mL}$, $p = 0.004$) in the LCD group after 6 weeks of intervention.⁶⁸ C-peptide used as an indicator of insulin secretion in this study was also found to be lower in the w-LCD group than that of the HCD group after 12 weeks of intervention. In addition, the fasting C-peptide levels were within the normal range (0.8-4.2 ng/mL) in both groups after dietary intervention. Due to the lack of baseline C-peptide, we could not conclude whether w-LCD maintains or reverses the status of insulin in CO populations. We speculate that the w-LCD demonstrates a cross-sectional statistical association with lower fasting insulin exposure, which concurrently co-exists with reduced VFA and shifts in fat redistribution.

GLP-1 and PYY

GLP-1 and PYY, as gastrointestinal satiety peptide hormone, could weakly promote insulin secretion from pancreatic β -cells, and thus is associated with alter fat distribution and reduce visceral fat deposition.²⁹ Zhao et al. found that GLP-1 analogs led to a reduction in visceral fat and a relative increase in hip and leg fats in high-fat diet-induced obese rats, which may promote fat redistribution.⁶⁹ Rachel et al. knocked out the PYY gene and found that the mice developed visceral fat accumulation.⁷⁰ According to the CIM, increasing the proportion of ω -

3 PUFA can improve intestinal flora and increase the production of SCFAs, which promote GLP-1 and PYY production via GPR43 and GPR40. This study found that fasting GLP-1 and PYY levels were higher in the w-LCD group than those of the HCD group [GLP-1: 2.10 (1.84, 2.69) pmol/L vs. 1.59 (0.86, 2.14)] pmol/L, $Z = -2.343$, $p = 0.019$; PYY: w-LCD (33.1 ± 5.50) pg/mL vs. (26.8 ± 5.93 pg/mL, $t = 3.663$, $p = 0.001$). The promotion of GLP-1 and PYY production may be related to the supplementation of ω -3 PUFA-rich nuts (walnuts) in the LCD group. Similar to this study, a previous study by our team, Ren et al found that the fasting GLP-1 level was higher in the intervention group after 12 weeks of intervention in T2D patients with nuts (almonds) added to LCD.⁷¹ In addition, Liana et al.⁷² found that after a 60g/d walnut intervention, postprandial PYY levels in the walnut group increased significantly compared to baseline. Numao et al.⁷³ and Aaron et al.⁷⁴ also demonstrated that low-carbohydrate, high-fat diet could increase the level of GLP-1 and PYY production. Therefore, we tentatively suggest that w-LCD might be accompanied by higher GLP-1 and PYY, which are statistically correlated with lower visceral adiposity and altered fat distribution.

W-LCD reduced BMI in CO populations

BMI and BF%, as concise and effective indicators to measure obesity, can better reflect the degree of obesity.^{73, 75} These indicators are associated with the blood pressure variability index, which increases with the degree of obesity and thus increases the risk of CVD.⁷⁶ In addition, the risk of diabetes increases with greater obesity-related indicators.⁷⁷ Although we found no significant difference in BMI between the two groups at 12 weeks, which is consistent with the study by Guo et al,⁷⁸ both low-carbohydrate and low-fat diets led to short-term weight loss in obese or overweight people with impaired glucose regulation using generalized estimating equations; the Group $\times$ Time interaction for BMI was significant. This indicates that the BMI in the w-LCD group decreased much more over the course of the study, which may be related to the restriction of carbohydrate intake. A meta-analysis of RCT found that LCD significantly reduced BMI (SMD: -1.66, 95%CI: -2.70, -0.61).⁷⁹ Another reason may be related to walnut supplementation in this study. A dose-response meta-analysis found that walnut intake up to 35 g/d significantly reduced BMI (Coef. = -1.24, $p = 0.041$).⁸⁰ Therefore, increasing walnut intake along with LCD may lead to more reduction in BMI. Although this study did not find that w-LCD could effectively reduce BF%, there was a trend of its decrease. A network meta-analysis by Rubén et al showed that walnuts significantly reduced BF% (SMD: -0.85; 95%CI: -1.20, -0.49).⁸¹

Conclusions

A walnut-based low carbohydrate diet achieves a significantly greater and faster fat reduction over time compared to the control group, providing a solid evidence-based foundation for dietary management in people with CO.

Strengths and limitations

To our knowledge, this is the first study to explore the effect of w-LCD on improving fat redistribution and visceral fat accumulation of CO populations in China. However, there are some limitations. Firstly, due to time limitation, this study was a short-term 12weeks clinical study, and it will be necessary to explore the long-term effects of w-LCD on fat redistribution and visceral fat deposition, as well as to explore participants' adherence to w-LCD programs after the intervention ends in the future. Secondly, due to the limitation of funds, this study only detected fasting GLP-1 and C-peptide at 12 weeks, which could not analyze the causal relationship of w-LCD on the humoral factors. Thus detecting the dynamic postprandial GLP-1 was needed for interpretation of pathogenesis of obesity based on the CIM. In addition, this study did not explore the relationship between w-LCD and metabolic indicators such as insulin resistance and inflammatory markers, which will also be a focus of our future research. Thirdly, due to the fact that this study is a small sample RCT which might limit the statistical power, and stratified analyses such as gender, different baseline obesity level and lipid profile levels cannot be conducted about the effect of w-LCD on the improvement of outcome indicators. Therefore, this study still needs to be validated in larger populations in the future. Fourthly, in this study, the HCD group had a higher dropout rate, which may be related with the less effective weight loss in the HCD group compared to the w-LCD group, weakening their weight loss confidence. Another limitation is the failure to strictly control for isocaloric conditions across the intervention groups. Fifthly, this study employed three-day dietary records as dietary assessment tools, which may introduce self-reporting bias. Therefore, future research should consider utilizing objective biological markers or food diaries to evaluate dietary intake and adherence to dietary regimens more accurately and impartially. Finally, this study did not design a personalized diet based on participants' weight, nature of work, etc., which is another one of the limitations of this study. The intervention program can be improved in the future.

SUPPLEMENTARY MATERIALS

All supplementary tables and figures are available upon request from the editorial office, and are also accessible on the journal's webpage (apjcn.qdu.edu.cn).

ACKNOWLEDGEMENTS

We thank the participants with central obesity who volunteered to participate in this study. We also thank all of the staff in the physical examination centre of the First Affiliated Hospital of Soochow University, who provided us with assistance so as to ensure that the study was conducted.

CONFLICT OF INTEREST AND FUNDING DISCLOSURE

The authors declare no conflict of interest.

This research was funded by the National Natural Science Foundation of China (No.81673146) and CNS Research Fund for DRIs (Grant No. 2021-Water).

REFERENCES

1. Carey DG. Abdominal obesity. *Curr Opin Lipidol*. 1998;9:35-40.doi: 10.1097/00041433-199802000-00008
2. Wong MCS, Huang J, Wang J, et al. Global, regional and time-trend prevalence of central obesity: a systematic review and meta-analysis of 13.2 million subjects. *Eur J Epidemiol*. 2020;35:673-683. doi: 10.1007/s10654-020-00650-3
3. Zhu J, Zhang Y, Wu Y, et al. Obesity and Dyslipidemia in Chinese Adults: A Cross-Sectional Study in Shanghai, China. *Nutrients*. 2022;14:2321.doi: 10.3390/nu14112321
4. Cui L, Chen T, Li Z, et al. Association between dietary related factors and central obesity among married women: China Health and Nutrition Survey. *Appetite*. 2022;168:105785.doi: 10.1016/j.appet.2021.105785
5. Chen W, Wilson JL, Khaksari M, et al. Abdominal fat analyzed by DEXA scan reflects visceral body fat and improves the phenotype description and the assessment of metabolic risk in mice. *Am J Physiol Endocrinol Metab*. 2012;303:E635-643. doi: 10.1152/ajpendo.00078.2012
6. Bays H. Central obesity as a clinical marker of adiposopathy; increased visceral adiposity as asurrogate marker for global fat dysfunction. *Curr Opin Endocrinol Diabetes Obes*. 2014;21:345-351.doi: 10.1097/MED.0000000000000093
7. Ashwell M, Gunn P, Gibson S. Waist-to-height ratio is a better screening tool than waist circumference and BMI for adult cardiometabolic risk factors: systematic review and meta-analysis.*Obes Rev*. 2012;13:275-286. doi: 10.1111/j.1467-789X.2011.00952.x

8. Manolopoulos KN, Karpe F, Frayn KN. Gluteofemoral body fat as a determinant of metabolic health. *Int J Obes (Lond)*. 2010;34:949-959. doi: 10.1038/ijo.2009.286
9. Decoda Study G, Nyamdorj R, Qiao Q, et al. BMI compared with central obesity indicators in relation to diabetes and hypertension in Asians. *Obesity (Silver Spring)*. 2008;16:1622-1635. doi: 10.1038/oby.2008.73
10. Wang Y, Rimm EB, Stampfer MJ, et al. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. *Am J Clin Nutr*. 2005;81:555-563. doi: 10.1093/ajcn/81.3.555
11. Kissebah AH, Vydellingum N, Murray R, et al. Relation of body fat distribution to metabolic complications of obesity. *J Clin Endocrinol Metab*. 1982;54:254-260. doi: 10.1210/jcem-54-2-254
12. Bi X, Seabolt L, Shibao C, et al. DXA-measured visceral adipose tissue predicts impaired glucose tolerance and metabolic syndrome in obese Caucasian and African-American women. *Eur J Clin Nutr*. 2015;69:329-336. doi: 10.1038/ejcn.2014.227
13. Despres JP. Body fat distribution and risk of cardiovascular disease: an update. *Circulation*. 2012;126:1301-1313. doi: 10.1161/CIRCULATIONAHA.111.067264
14. Duman TT, Atak Tel BM, Bilgin S, et al. Evaluation of the effects of intermittent fasting on clinical and laboratory parameters in metabolic syndrome. *Experimental Biomedical Research*. 2025;8:111-119. doi: 10.30714/j-ebr.2025.242
15. Jensen MD. Role of body fat distribution and the metabolic complications of obesity. *J Clin Endocrinol Metab*. 2008;93:S57-63. doi: 10.1210/jc.2008-1585
16. Cioffi CE, Alvarez JA, Welsh JA, et al. Truncal-to-leg fat ratio and cardiometabolic disease risk factors in US adolescents: NHANES 2003-2006. *Pediatr Obes*. 2019;14:e12509. doi: 10.1111/ijpo.12509
17. Ludwig DS, Ebbeling CB. The Carbohydrate-Insulin Model of Obesity: Beyond "Calories In, Calories Out". *JAMA Intern Med*. 2018;178:1098-1103. doi: 10.1001/jamainternmed.2018.2933
18. Ludwig DS, Friedman MI. Increasing adiposity: consequence or cause of overeating? *JAMA*. 2014;311:2167-2168. doi: 10.1001/jama.2014.4133
19. Hall KD. A review of the carbohydrate-insulin model of obesity. *Eur J Clin Nutr*. 2018;72:183. doi: 10.1038/ejcn.2017.156
20. Hall KD, Guyenet SJ, Leibel RL. The Carbohydrate-Insulin Model of Obesity Is Difficult to Reconcile With Current Evidence. *JAMA Intern Med*. 2018;178:1103-1105. doi: 10.1001/jamainternmed.2018.2920
21. Sandforth A, Arreola EV, Hanson RL, et al. Prevention of type 2 diabetes through prediabetes remission without weight loss. *Nat Med*. 2025;31:3330-3340. doi: 10.1038/s41591-025-03944-9
22. Ibrahim MM. Subcutaneous and visceral adipose tissue: structural and functional differences. *Obes Rev*. 2010;11:11-18. doi: 10.1111/j.1467-789X.2009.00623.x

23. Hall KD, Chen KY, Guo J, et al. Energy expenditure and body composition changes after anisocaloric ketogenic diet in overweight and obese men. *Am J Clin Nutr.* 2016;104:324-333.doi: 10.3945/ajcn.116.133561
24. Ramos DE. Walnut production manual. United States: University of California Press; 1997.
25. Liu Y, Li N, Yan N, et al. Protocol for a randomized controlled trial to test the acceptability and adherence to 6-months of walnut supplementation in Chinese adults at high risk of cardiovascular disease. *Nutr J.* 2021;20:3.doi: 10.1186/s12937-020-00660-7
26. Machate DJ, Figueiredo PS, Marcelino G, et al. Fatty Acid Diets: Regulation of Gut Microbiota Composition and Obesity and Its Related Metabolic Dysbiosis. *Int J Mol Sci.* 2020;21:4093.doi: 10.3390/ijms21114093
27. Tsutsumi R, Yamasaki Y, Takeo J, et al. Long-chain monounsaturated fatty acids improve endothelial function with altering microbial flora. *Transl Res.* 2021;237:16-30.doi: 10.1016/j.trsl.2021.03.016
28. Hernandez MAG, Canfora EE, Jocken JWE, et al. The Short-Chain Fatty Acid Acetate in Body Weight Control and Insulin Sensitivity. *Nutrients.* 2019;11: 1943..doi: 10.3390/nu11081943
29. Creutzfeldt W. The incretin concept today. *Diabetologia.* 1979;16:75-85.doi: 10.1007/BF01225454
30. Paniagua JA, Gallego de la Sacristana A, Romero I, et al. Monounsaturated fat-rich diet prevents central body fat distribution and decreases postprandial adiponectin expression induced by a carbohydrate-rich diet in insulin-resistant subjects. *Diabetes Care.* 2007;30:1717-1723.doi: 10.2337/dc06-2220
31. Miyashita Y, Koide N, Ohtsuka M, et al. Beneficial effect of low carbohydrate in low calorie diets on visceral fat reduction in type 2 diabetic patients with obesity. *Diabetes Res Clin Pract.* 2004;65:235-241.doi: 10.1016/j.diabres.2004.01.008
32. Prevention CSO, Section C, NutritionSection CNCS, et al. Expert Consensus on the Prevention and Treatment of Obesity Among Chinese Residents. *Chinese Journal of Preventive Medicine.* 2022;23:321-339.doi: 10.16506/j.1009-6639.2022.05.001
33. Wang LL. The Effect of Different Types of Nuts on Blood Glucose Control in Patients with Type 2 Diabetes [Master's Thesis]. Suzhou: Soochow University; 2017.
34. Wade AT, Davis CR, Dyer KA, et al. A Mediterranean diet supplemented with dairy foods improves mood and processing speed in an Australian sample: results from the MedDairy randomized controlled trial. *Nutr Neurosci.* 2020;23:646-658.doi: 10.1080/1028415X.2018.1543148
35. Zhang MZ, and Lü DB. Practical Medical Statistics and SAS Applications. Suzhou: Suzhou University Press; 2015.
36. Mei S, Ding J, Wang K, et al. Mediterranean Diet Combined With a Low-Carbohydrate Dietary Pattern in the Treatment of Overweight Polycystic Ovary Syndrome Patients. *Front Nutr.* 2022;9:876620.doi: 10.3389/fnut.2022.876620
37. NMMBCE, PAM, CNBCNS, et al. Guidelines for medical nutrition treatment of overweight/obesity in China (2021). *Asia Pac J Clin Nutr.* 2022;31:450-482.doi: 10.6133/apjcn.202209_31(3).0013

38. Yi Y, Baek JY, Lee E, et al. A Comparative Study of High-Frequency Bioelectrical Impedance Analysis and Dual-Energy X-ray Absorptiometry for Estimating Body Composition. *Life (Basel)*. 2022;12: 994. doi: 10.3390/life12070994
39. Li S, Li S, Ding J, et al. Visceral fat area and body fat percentage measured by bioelectrical impedance analysis correlate with glycometabolism. *BMC Endocr Disord*. 2022;22:231. doi: 10.1186/s12902-022-01142-z
40. GBD 2016 Neurology Collaborators. Global, regional, and national burden of neurological disorders, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. 2019;18:459-480. doi: 10.1016/S1474-4422(18)30499-X
41. Ma M, Dong FW, Lan JY. Associations between sleep duration, physical activity, and cognitive impairment in older adults-empirical analysis based on CHARLS data. *Front Public Health*. 2025;13:1589606. doi: 10.3389/fpubh.2025.1589606
42. Li Q, Xiong R, Wang L, et al. Associations of dietary habits, physical activity and cognitive views with gestational diabetes mellitus among Chinese women. *Public Health Nutr*. 2014;17:1850-1857. doi: 10.1017/S1368980013001882
43. Coupland SG, McLeod DR, Wesenberg RL, et al. CAMPS: computer-automated metacarpophalangeal profile system. *Am J Med Genet*. 1985;22:375-381. doi: 10.1002/ajmg.1320220222
44. Pramyothin P, Karastergiou K. What Can We Learn from Interventions That Change Fat Distribution? *Curr Obes Rep*. 2016;5:271-281. doi: 10.1007/s13679-016-0215-x
45. Qiu Z, Lee DH, Lu Q, et al. Associations of Regional Body Fat With Risk of Cardiovascular Disease and Mortality Among Individuals With Type 2 Diabetes. *J Clin Endocrinol Metab*. 2025;110:e372-e381. doi: 10.1210/clinem/dgae192
46. Peppas M, Koliaki C, Hadjidakis DI, et al. Regional fat distribution and cardiometabolic risk in healthy postmenopausal women. *Eur J Intern Med*. 2013;24:824-831. doi: 10.1016/j.ejim.2013.07.001
47. Savgan-Gurol E, Bredella M, Russell M, et al. Waist to hip ratio and trunk to extremity fat (DXA) are better surrogates for IMCL and for visceral fat respectively than for subcutaneous fat in adolescent girls. *Nutr Metab (Lond)*. 2010;7:86. doi: 10.1186/1743-7075-7-86
48. Kouda K, Iki M, Fujita Y, et al. Trunk-to-peripheral fat ratio predicts a subsequent blood pressure in normal-weight pubertal boys: a 3-year follow-up of the Kitakata Kids Health Study. *Environ Health Prev Med*. 2020;25:41. doi: 10.1186/s12199-020-00878-1
49. Li X, Katashima M, Yasumasu T, et al. Visceral fat area, waist circumference and metabolic risk factors in abdominally obese Chinese adults. *Biomed Environ Sci*. 2012;25:141-148. doi: 10.3967/0895-3988.2012.02.003
50. Yan Y, Liu J, Zhao X, et al. Regional Adipose Compartments Confer Different Cardiometabolic Risk in Children and Adolescents: The China Child and Adolescent Cardiovascular Health Study. *Mayo Clin Proc*. 2019;94:1974-1982. doi: 10.1016/j.mayocp.2019.05.026
51. Luo J, Hendryx M, Laddu D, et al. Racial and Ethnic Differences in Anthropometric Measures as Risk Factors for Diabetes. *Diabetes Care*. 2019;42:126-133. doi: 10.2337/dc18-1413

52. Khan I, Chong M, Le A, et al. Surrogate Adiposity Markers and Mortality. *JAMA Netw Open*. 2023;6:e2334836.doi: 10.1001/jamanetworkopen.2023.34836
53. Shemirani F, Djafarian K, Fotouhi A, et al. Effect of Paleolithic-based low-carbohydrate vs. moderate-carbohydrate diets with portion-control and calorie-counting on CTRP6, asprosin and metabolic markers in adults with metabolic syndrome: A randomized clinical trial. *Clin Nutr ESPEN*. 2022;48:87-98.doi: 10.1016/j.clnesp.2021.11.013
54. Holmer M, Lindqvist C, Petersson S, et al. Treatment of NAFLD with intermittent calorie restriction or low-carb high-fat diet - a randomised controlled trial. *JHEP Rep*. 2021;3:100256.doi:10.1016/j.jhepr.2021.100256
55. Neeland IJ, Ross R, Despres JP, et al. Visceral and ectopic fat, atherosclerosis, and cardiometabolic disease: a position statement. *Lancet Diabetes Endocrinol*. 2019;7:715-725.doi: 10.1016/S2213-8587(19)30084-1
56. Zhou B. Analysis of the Correlation Between Visceral Fat Area and Changes in Serum Uric Acid Levels in Patients with Type 2 Diabetes as Determined by Bioimpedance Analysis[Master's Thesis]. Suzhou: Soochow University; 2021.
57. Shuster A, Patlas M, Pinthus JH, et al. The clinical importance of visceral adiposity: a critical review of methods for visceral adipose tissue analysis. *Br J Radiol*. 2012;85:1-10.doi: 10.1259/bjr/38447238
58. Japan Society for the Study of O. New criteria for 'obesity disease' in Japan. *Circ J*. 2002;66:987-992.doi: 10.1253/circj.66.987
59. Cunha GM, Guzman G, Correa De Mello LL, et al. Efficacy of a 2-Month Very Low-Calorie Ketogenic Diet (VLCKD) Compared to a Standard Low-Calorie Diet in Reducing Visceral and Liver Fat Accumulation in Patients With Obesity. *Front Endocrinol (Lausanne)*. 2020;11:607.doi: 10.3389/fendo.2020.00607
60. Zurbau A, Noronha JC, Khan TA, et al. The effect of oat beta-glucan on postprandial blood glucose and insulin responses: a systematic review and meta-analysis. *Eur J Clin Nutr*. 2021;75:1540-1554.doi: 10.1038/s41430-021-00875-9
61. Ludwig DS. Carbohydrate-insulin model: does the conventional view of obesity reverse cause and effect? *Philos Trans R Soc Lond B Biol Sci*. 2023;378:20220211.doi: 10.1098/rstb.2022.0211
62. Torbay N, Bracco EF, Geliebter A, et al. Insulin increases body fat despite control of food intake and physical activity. *Am J Physiol*. 1985;248:R120-124.doi: 10.1152/ajpregu.1985.248.1.R120
63. Botzelli JD, Overby P, Lindo L, et al. Adipose depot-specific upregulation of Ucp1 or mitochondrial oxidative complex proteins are early consequences of genetic insulin reduction in mice. *Am J Physiol Endocrinol Metab*. 2020;319:E529-E539.doi: 10.1152/ajpendo.00128.2020
64. Templeman NM, Skovso S, Page MM, et al. A causal role for hyperinsulinemia in obesity. *J Endocrinol*. 2017;232:R173-R183.doi: 10.1530/JOE-16-0449
65. Page MM, Skovso S, Cen H, et al. Reducing insulin via conditional partial gene ablation in adults reverses diet-induced weight gain. *FASEB J*. 2018;32:1196-1206.doi: 10.1096/fj.201700518R

66. Cara B Ebbeling, Henry A Feldman, Gloria L Klein, et al. Effects of a low carbohydrate diet on energy expenditure during weight loss maintenance: randomized trial. *BMJ*. 2020;371:m4264.doi: 10.1136/bmj.m4264
67. Santos FL, Esteves SS, da Costa Pereira A, et al. Systematic review and meta-analysis of clinical trials of the effects of low carbohydrate diets on cardiovascular risk factors. *Obes Rev*. 2012;13:1048-1066.doi: 10.1111/j.1467-789X.2012.01021.x
68. Hernandez TL, Sutherland JP, Wolfe P, et al. Lack of suppression of circulating free fatty acids and hypercholesterolemia during weight loss on a high-fat, low-carbohydrate diet. *Am J Clin Nutr*. 2010;91:578-585.doi: 10.3945/ajcn.2009.27909
69. Zhao L, Zhu C, Lu M, et al. The key role of a glucagon-like peptide-1 receptor agonist in body fat redistribution. *J Endocrinol*. 2019;240:271-286.doi: 10.1530/JOE-18-0374
70. Batterham RL, Heffron H, Kapoor S, et al. Critical role for peptide YY in protein-mediated satiation and body-weight regulation. *Cell Metab*. 2006;4:223-233.doi: 10.1016/j.cmet.2006.08.001
71. Ren MX. Current Status of Carbohydrate Intake Among Patients with Type 2 Diabetes and the Effect of an Almond-Based Low-Carbohydrate Dietary Intervention on Their Levels of Depression [Master's Thesis]. Suzhou: Soochow University; 2021.
72. Guarneiri LL, Paton CM, Cooper JA. Appetite responses to pecan-enriched diets. *Appetite*. 2022;173:106003.doi: 10.1016/j.appet.2022.106003
73. Numao S, Kawano H, Endo N, et al. Short-term low carbohydrate/high-fat diet intake increases postprandial plasma glucose and glucagon-like peptide-1 levels during an oral glucose tolerance test in healthy men. *Eur J Clin Nutr*. 2012;66:926-931.doi: 10.1038/ejcn.2012.58
74. Aaron Hengist, Christina M Sciarrillo, Juen Guo, et al. Gut-derived appetite hormones do not explain energy intake differences in humans following low-carbohydrate vs low-fat diets. *Obesity (Silver Spring)*. 2024; 32:1689-1698. doi:10.1002/oby.24104
75. Litwic A, Edwards MH, Dennison EM, et al. Epidemiology and burden of osteoarthritis. *Br Med Bull*. 2013;105:185-199.doi: 10.1093/bmb/lds038
76. Liu JP, Dang W, Zhang J. Correlation between Different Obesity Indices and Blood Pressure Variability in Patients with Primary Hypertension. *Chinese Journal of Hypertension*. 2021;29:1083-1091.doi: 10.16439/j.issn.1673-7245.2021.11.012
77. Wang YX, Ren XN, Xue J, et al. Multifactorial Analysis and Intervention Strategies for Diabetes Caused by Overweight and Obesity in Adults in the Shenyang Region. *Public Health and Preventive Medicine*. 2021;32:100-104.doi: 10.3969/j.issn.
78. Guo H, Wang L, Huang X, et al. Effects of low-carbohydrate vs low-fat diets on weight loss and metabolic risk factors in obese/overweight individuals with impaired glucose regulation: A randomized controlled trial. *Asia Pac J Clin Nutr*. 2022;31:512-519.doi: 10.6133/apjcn.202209_31(3).0018
79. Silverii GA, Cosentino C, Santagiuliana F, et al. Effectiveness of low-carbohydrate diets for long-term weight loss in obese individuals: A meta-analysis of randomized controlled trials. *Diabetes Obes Metab*. 2022;24:1458-1468.doi: 10.1111/dom.14709

80. Fang Z, Dang M, Zhang W, et al. Effects of walnut intake on anthropometric characteristics: A systematic review and dose-response meta-analysis of randomized controlled trials. *Complement Ther Med*. 2020;50:102395.doi: 10.1016/j.ctim.2020.102395
81. Fernandez-Rodriguez R, Mesas AE, Garrido-Miguel M, et al. The Relationship of Tree Nuts and Peanuts with Adiposity Parameters: A Systematic Review and Network Meta-Analysis. *Nutrients*. 2021;13: 2251.doi: 10.3390/nu13072251

Not Proof Read

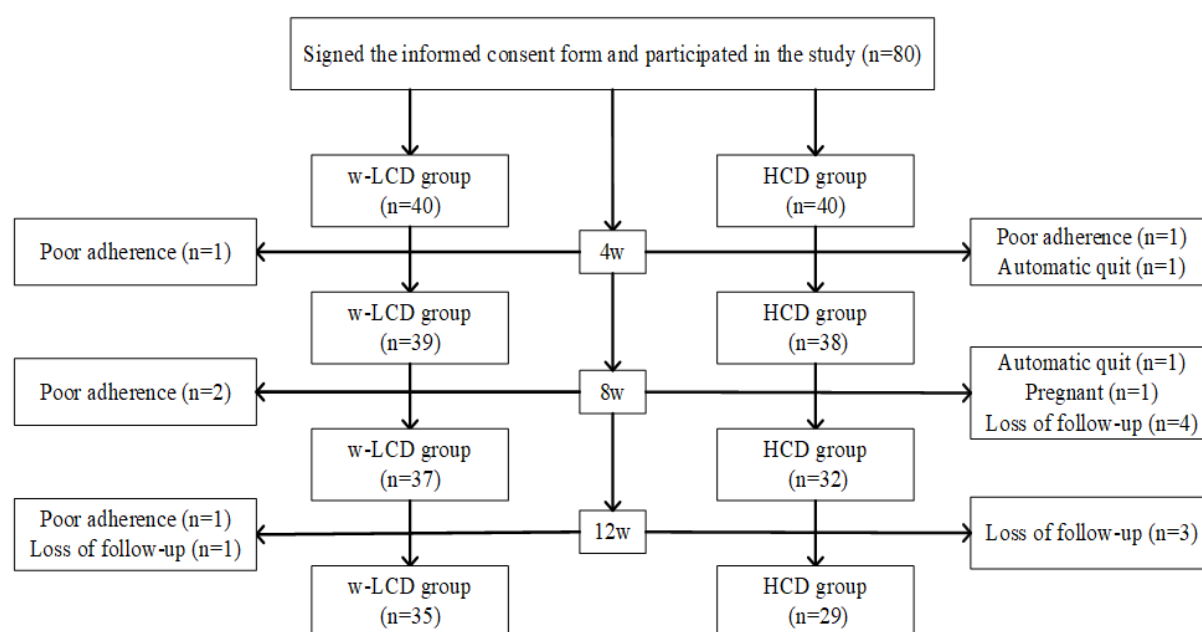


Figure 1. The flowchart of inclusion

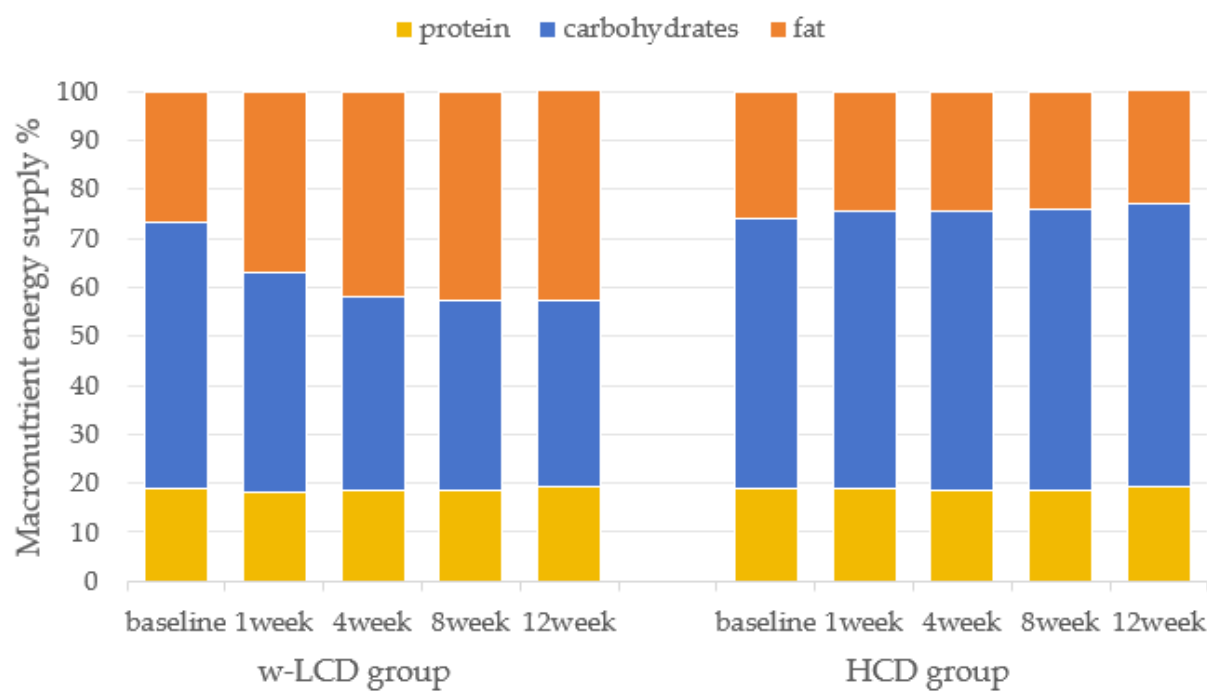


Figure 2. The trend of the macronutrient energy supply % in the w-LCD group and HCD group

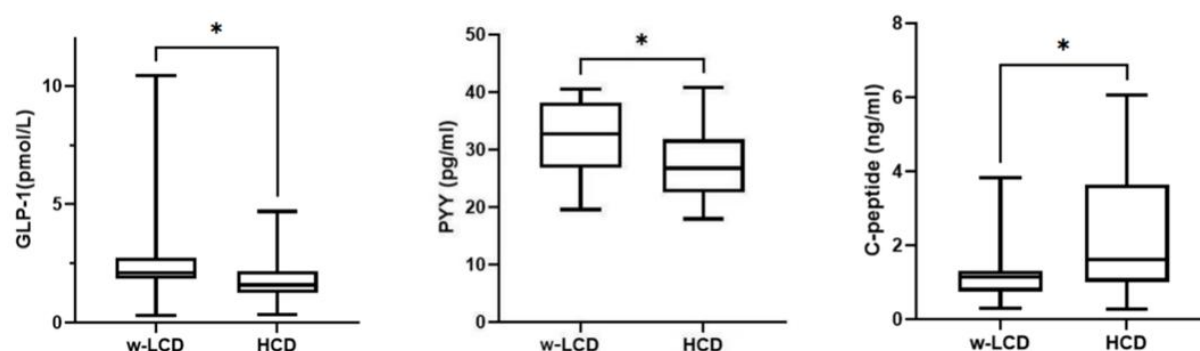


Figure 3. Comparison of GLP-1(pmol/L), PYY (pg/mL) and C-peptide (ng/mL) between the two groups at 12weeks. w-LCD, walnut-based low carbohydrate diet; HCD, high-carbohydrate low-fat diet; GLP-1, Glucagon-like peptide-1. PYY, Peptide YY. * $p < 0.05$

Table 1. Comparison of demographic and clinical data between the two groups

Variables	w-LCD group (n=35) mean±SD/n (%)	HCD group (n=29) mean±SD/n (%)	t/χ ²	p
Age (years)	36.9 ± 10.2	37.9 ± 9.5	-0.392 [†]	0.696
Sex				
Male	17.0 (48.6)	12.0 (41.4)	0.331 [†]	0.565
Education				
≤Middle school	1.00 (2.90)	1.00 (3.40)	-	>0.999
Marital status				
Married	25.0 (71.4)	25.0 (86.2)	2.027 [†]	0.155
Occupation				
On the job	27.0 (77.1)	27.0 (93.1)	1.973 [§]	0.160
Medical insurance				
Yes	34.0 (97.1)	29.0 (100)	-	>0.999
Monthly personal income (yuan)				
≤5000	8.00 (22.9)	2.00 (6.90)	1.973 [§]	0.160
Smoking				
Yes	3.00 (8.60)	4.00 (13.8)	0.070 [§]	0.792
Drinking				
Yes	8.00 (22.9)	9.00 (31.0)	0.544 [†]	0.461
Quality of sleep			3.498 [¶]	0.171
Poor	0.00 (0.00)	3.00 (10.3)		
Moderate	20.0 (57.1)	16.0 (55.2)		
Good	15.0 (42.9)	10.0 (34.5)		
Sleeping time (h/d)	7.00 ± 0.60	6.90 ± 0.90	0.305 [†]	0.761
Other diseases				
Yes	2.00 (5.70)	5.00 (17.2)	1.142 [§]	0.285
SBP (mmHg)	122 ± 12.2	121 ± 10.8	0.119 [†]	0.906
DBP (mmHg)	77.4 ± 10.9	74.7 ± 9.10	1.032 [†]	0.306
Heart rate (bpm)	77.2 ± 8.80	77.9 ± 8.70	-0.281 [†]	0.779
Exercise at baseline (min/weeks)	84.0 ± 79.8	72.8 ± 107	0.480 [†]	0.633
Exercise at 12weeks (min/weeks)	69.4 ± 78.4	66.9 ± 104	0.111 [†]	0.912
Exercise intensity at baseline			1.313 [¶]	0.587
Low	24.0 (68.6)	22.0 (75.9)		
Moderate	7.00 (20.0)	6.00 (20.7)		
High	4.00 (11.4)	1.00 (3.40)		
Exercise intensity at 12weeks			1.649 [¶]	0.484
Low	24.0 (68.6)	24.0 (82.8)		
Moderate	5.00 (14.3)	2.00 (6.90)		
High	6.00 (17.1)	3.00 (10.3)		
Energy at baseline (kcal/d)	1746 ± 412	1892 ± 512	-1.217 [†]	0.228

w-LCD, walnut-based low carbohydrate diet; HCD, high-carbohydrate low-fat diet; SBP, systolic blood pressure; DBP, diastolic blood pressure.

[†]Two-sample independence t-test; [§]chi-square test; [§]Calibrated chi-square test; [¶]Fisher exact test.

Table 2. Comparison of total energy, macronutrients and DF intake between the two groups over time

Variables	w-LCD group (n=35) mean±SD/n(%)	HCD group (n=29) mean±SD/n(%)	Repeated Measures ANOVA [$\eta^2(p)$]			Post-hoc testing	
			Time effect	Group effect	Time × group interaction effect	Simple effects analyses	
						η^2	p
Energy (kcal/d)			0.653 (<0.001 ^{***})	0.023 (0.245)	0.013 (0.381)		
Baseline	1746 ± 412	1892 ± 512					
12 weeks	1162 ± 224	1211 ± 326					
Carbohydrate (g/d)			0.631 (<0.001 ^{***})	0.184 (0.001 ^{**})	0.046 (0.099)		
Baseline	238 ± 70.5	262 ± 81.6					
12 weeks	112 ± 32.9	175 ± 51.5					
Fat (g/d)			0.193 (<0.001 ^{***})	0.155 (0.002 ^{**})	0.290 (<0.001 ^{***})		
Baseline	52.1 ± 18.5	54.7 ± 21.3					
12 weeks	55.2 ± 11.2	31.3 ± 11.3				0.004	0.612
η^2	0.013	0.353				0.519	<0.001 ^{***}
p	0.379	<0.001 ^{***}					
Protein (g/d)			0.489 (<0.001 ^{***})	0.005 (0.256)	0.022 (0.591)		
Baseline	81.7 ± 25.9	89.0 ± 32.1					
12 weeks	55.0 ± 9.90	58.7 ± 18.2					
DF (g/d)			0.031 (0.175)	<0.001 (0.913)	<0.001 (0.875)		
Baseline	18.6 ± 11.9	18.7 ± 14.5					
12 weeks	21.3 ± 4.80	21.04 ± 5.10					

w-LCD, walnut-based low carbohydrate diet; HCD, high-carbohydrate low-fat diet; DF, dietary fiber.

^{**} p <0.01; ^{***} p <0.001.

Table 3. Comparison of body composition between the two groups over time

Variables	w-LCD group (n=35) mean±SD/n(%)	HCD group (n=29) mean±SD/n(%)	Repeated Measures ANOVA [$\eta^2(p)$]		Time × group interaction effect	Post-hoc testing	
			Time effect	Group effect		Simple effects analyses	
						η^2	<i>p</i>
TLR							
Baseline	1.80 ± 0.19	1.82 ± 0.16	0.101 (0.012*)	0.012 (0.347)	0.071 (0.039*)	<0.001	0.863
12 weeks	1.74 ± 0.20	1.81 ± 0.15				0.033	0.154
η^2	0.174	0.002					
<i>p</i>	0.001**	0.756					
WHR							
Baseline	0.93 ± 0.05	0.92 ± 0.04	0.147 (0.002**)	0.004 (0.643)	0.052 (0.072)		
12 weeks	0.90 ± 0.03	0.91 ± 0.09					
η^2							
VFA (cm ²)							
Baseline	113 ± 33.4	116 ± 109	0.367 (<0.001***)	0.013 (0.364)	0.093 (0.014*)	0.001	0.766
12 weeks	97.4 ± 31.7	109 ± 31.6				0.036	0.132
η^2	0.393	0.082					
<i>p</i>	<0.001***	0.022*					
BMI (kg/m ²)							
Baseline	27.6 ± 2.49	27.3 ± 2.26	0.296 (<0.001***)	0.004 (0.632)	0.168 (0.001**)	0.005	0.597
12 weeks	26.1 ± 2.63	27.0 ± 2.69				0.029	0.180
η^2	0.399	0.018					
<i>p</i>	<0.001***	0.292					
BF (kg)							
Baseline	28.9 ± 16.6	26.2 ± 5.34	0.113 (0.006)	0.001 (0.837)	0.047 (0.086)		
12 weeks	22.9 ± 6.42	24.8 ± 5.70					
η^2							
BF%							
Baseline	35.9 ± 14.6	34.5 ± 6.34	0.100 (0.007**)	0.001 (0.936)	0.038 (0.209)		
12 weeks	30.8 ± 7.43	32.6 ± 7.44					
η^2							

w-LCD, walnut-based low carbohydrate diet; HCD, high-carbohydrate low-fat diet; DF, dietary fiber.

p* < 0.01; *p* < 0.001.