



Association of low-carbohydrate diet score and carbohydrate quality with visceral adiposity and lipid accumulation product

Fatemeh Gholami¹, Fahime Martami¹, Parivash Ghorbaninezhad¹, Amin Mirrafiei¹, Mojdeh Ebaditabar¹, Samira Davarzani¹, Nadia Babaei¹, Kurosh Djafarian² and Sakineh Shab-Bidar^{1*}

¹Department of Community Nutrition, School of Nutritional Sciences and Dietetics, Tehran University of Medical Sciences (TUMS), Tehran, Iran

²Department of Clinical Nutrition, School of Nutritional Sciences and Dietetics, Tehran University of Medical Sciences (TUMS), Tehran, Iran

(Submitted 24 November 2021 – Final revision received 3 April 2022 – Accepted 27 April 2022 – First published online 16 May 2022)

Abstract

The present study examined the association between low-carbohydrate diet (LCD) score, glycemic index (GI), and glycemic load (GL) with visceral fat level (VFL) and lipid accumulation product (LAP). This cross-sectional study was conducted on 270 adults (118 men and 152 women) aged between 18–45 living in Tehran, Iran, between February 2017 and December 2018. Dietary intake was assessed using a validated semi-quantitative food frequency questionnaire (FFQ). Body composition were also assessed. We used analyses of covariance and binary logistic regression to explore associations after controlling for age, energy intake (model 1), education, smoking status, physical activity, occupation, marriage and metabolic diseases. There were no significant differences between tertiles of GI, GL and LCD for means of anthropometric measures, LAP and VFL index in men, while women in the highest tertile of GI and GL had significantly higher mean LAP in the crude model ($P = 0.02$) and model 1 ($P = 0.04$), which disappeared after controlling for other confounders ($P = 0.12$). Moreover, the OR and CIs for having high LAP and VFL was not associated with dietary GI, GL and LCD in crude and adjusted models. However, chance of high VFL reduced by 65% and 57% among women with high adherence to LCD score (OR = 0.35, 95% CI = 0.16–0.78, $P = 0.01$) and model 1 (OR = 0.43, 95% CI = 0.18–1, $P = 0.05$), respectively. However, this significant association disappeared after controlling for other confounders ($P = 0.07$). Overall, we found carbohydrate quality and LCD score are not associated with LAP and VFL index. However, gender-specific relationship should not be neglect and warrants further investigation.

Key words: Score: Glycaemic index: Glycaemic load: Visceral fat level: Lipid accumulation product

Obesity is a growing public health problem in the world that is closely related to increased cardiovascular disorders⁽¹⁾. Based on the previous evidence, central rather than general obesity may specifically increase the risk of diabetes, metabolic syndrome, CHD, certain cancers and mortality rate^(2–9).

Visceral adipose tissue (VAT) is a hormonally effective part of body fat mass that is collected within the abdominal cavity, near the digestive processes^(10,11). VAT is considered an independent risk factor for metabolic syndrome (MetS) due to its role in glucose⁽¹²⁾, lipid metabolism⁽¹³⁾ and controlling blood pressure (BP)⁽¹⁴⁾. It has been shown that patients with visceral obesity may promote atherosclerosis and are particularly prone to CVD⁽¹⁵⁾. More sensitive and specific biomarkers than classical indicators (BMI, waist circumferences and waist:hip ratio) are needed to enable health professionals to evaluate visceral adipose tissue. Lipid accumulation product (LAP) index is a biomarker of central fat

accumulation that has newly been developed and has been advocated as a correct indicator of the risk of insulin resistance, MetS, type 2 diabetes and CVD^(16,17). LAP is also correlated with abnormal glucose homeostasis and insulin resistance, as well as increased alanine aminotransferase, in healthy people⁽¹⁸⁾. BMI does not distinguish between fat and lean tissues. The components of LAP include waist size as a measure of truncal fat and serum triglyceride that are related to insulin levels as a risk factor of CVD, and it has been reported that LAP is a stronger indicator of cardiovascular risk in USA people than BMI^(19,20).

The lifestyle component plays an important role in the aetiology of obesity⁽²¹⁾. Visceral obesity and the aforementioned cardiometabolic disease may be linked through potentially modifiable lifestyle factors that provoke fat accumulation, such as special dietary habits⁽²²⁾. Since carbohydrate is the main source of energy among the Iranian population⁽²³⁾, it is of great

* Corresponding author: Sakineh Shab-Bidar, email s.shabbidar@gmail.com

interest to reveal how carbohydrate intakes link to the risk of fat deposition. Besides dietary pattern and macronutrient intake, carbohydrate quality is another important factor that has previously been shown to have a link with MetS components^(24,25). It has been proposed that VAT is changed by the qualitative aspects of nonenergetic diets, although evidence of a macronutrient combination of diet and VAT is still limited⁽²⁶⁾.

Recent research shows that energy expenditure, mainly in the form of carbohydrates or fat, for 3 months does not affect visceral fat and MetS in a low-processed, low-glycaemic diet⁽²⁶⁾. There have been numerous studies to investigate the relationship between glycaemic index (GI) and glycaemic load (GL) with obesity in adults with inconclusive associations. Taking a low-fat diet as a strategy to lose weight and reduce visceral fat was developed in many previous trials^(27,28). However, in a low-fat diet, the percentage of dietary carbohydrates increases and contributed to an increment in blood sugar and consequently higher fat storage by the increased level of insulin⁽²⁹⁾. Because of inconsistent findings, many questions remain still open regarding the relationship between low-carbohydrate diet (LCD) score and carbohydrate quality and visceral adiposity. Then, understanding the real association may help us to design interventions to reduce abdominal obesity and its associated complications. Therefore, we aimed to investigate the relationship between LCD score and dietary GI and GL with visceral fat level (VFL) and LAP in adults in Tehran.

Participants and methods

Study design

This cross-sectional study was conducted on 270 adults, aged between 18 and 45 years, who lived in Tehran between February 2017 and December 2018. The sample was chosen using a convenient sampling method within different health centres in Tehran. Health centres were selected randomly from different areas of Tehran. We applied advertisements, distribution of flyers in public locations and information sessions at health care centres. People were invited to participate in the study after the clarification of the objectives and benefits of the research. We calculated the sample size of 254 using the formula $N = ((Z_{1-\alpha/2})^2 \times (S)^2) / d^2$ considering ' α ' = 0.05, ' d ' = 4% and the effect size = 1.5⁽³⁰⁾. Totally, 270 participants were selected for inclusion to compensate for the potential exclusion of participants due to under- and over-reporting of total energy intake.

The current study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all methods including human subjects were approved by the ethical standards of the Tehran University of Medical Sciences, which established the protocol and informed consent form (Ethic Number: IR.TUMS.VCR.REC.1396-4306). All members signed written informed consent before the start of the study.

Eligibility criteria

Participants with specific diets, such as weight loss and weight gain diets, adults with chronic diseases afflicting the sleeping metabolic rate, including diabetes, hormonal and CVD, pregnant

and lactating women, receiving any special medication or supplement (slimming medicine, hormone, sedative, supplements including thermogenic substances, such as caffeine and green tea and linoleic acid conjugate) were excluded from the study.

Demographics

Additional covariates include age, gender, smoking status (non-smoker, former smoker or current smoker), marital status (married or single), education status (high as above the diploma or low as under diploma), medical history (underlying disease such as diabetes, hypertension, stroke, cancer or dyslipidaemia) and physical activity level (low, moderate or vigorous) were collected using approved questionnaires.

Physical activity

Physical activity was evaluated using the International Physical Activity Questionnaire (IPAQ), which is an interview-administered instrument, by an experienced interviewer. IPAQ was previously validated and is consistent in the Iranian population⁽³¹⁾. Data were collected regarding walking, moderate and vigorous activity, in the preceding week. Also, the time and regularity of activity days were recorded. During the present study, we used the short form of the IPAQ (the 'last 7-day recall' version of the IPAQ-SF), which records three intensity levels of activity based on the metabolic equivalents (MET). Eventually, MET were categorised as low (< 600 MET-min/week), moderate (600–3000 MET-min/week) and vigorous (> 3000 MET-min/week).

Blood pressure

BP was measured twice using a standard mercury sphygmomanometer. Participants were questioned about the consumption of tea and coffee, physical activity and a full bladder. They were in a seated position comfortably for 15 min before the BP reading. The mean of the two measured systolic BP (SBP) and diastolic BP (DBP) were reported.

Anthropometry measurements and body composition

Weight was measured using digital scales (Seca model 808; Seca, measurement accuracy ± 0.1 kg), whilst height was measured using a stadiometer (Seca, measurement accuracy ± 0.1 cm) in health care centres. BMI was calculated using measured weight (kg)/height (metres) squared. waist circumference (WC) was measured between the lower rib and iliac crest, at the widest portion, with light clothing, using a tape metre (Seca 201) without any pressure on the body⁽³²⁾. Hip circumference was measured at the maximal level over light clothing, using a non-stretch tape measure, without putting pressure on the body surface. Waist: hip ratio (WHR) was calculated as waist circumference (cm) divided by hip circumference (cm). VFL was measured using bioimpedance analysis (InBody S10, JMW140; InBody). Also, to improve measurement accuracy, people were advised not to engage in moderate to vigorous physical activity 1-to-2 h before using the bioimpedance analysis devices and to empty their bladder before measurement. To minimise the possible measurement error, a single trained dietitian did all the procedures.



Definitions

LAP is based on a combination of WC and the fasting concentration of TG, two markers that are associated with insulin resistance and cardiovascular risk. It was developed based on the data of the third National Health and Nutrition Examination Survey (NHANES III), on a sample of mostly non-Hispanic blacks and Mexican Americans, conducted between 1988 and 1994. The sample contained 4447 men and non-pregnant 4733 women, over the age of 18 years. It has been shown to have a strong and reliable diagnostic accuracy in Iranian adults previously⁽³³⁾. LAP was calculated as $(\text{waist circumference (WC)} - 65) \times \text{TG}$ in men and $(\text{WC} - 58) \times \text{TG}$ in women⁽³⁴⁾.

Dietary assessment

The dietary intake of participants was assessed by a valid and reliable semi-quantitative FFQ, which contained 168 food items including low-fat and high-fat dairy, a variety of vegetables, fruits, grains, different meats, sweets and sugary foods, nuts, fats and oils and legumes⁽³⁴⁾. Esfahani *et al.* assessed the repeatability of food groups included in the FFQ designed for the Tehran Lipid and Glucose Study (TLGS) and found it to be reliable and valid for assessing the intake of a variety of foods. The FFQ was directed by trained dietitians to limit any possible miscalculation, via face-to-face interviews, asking participants to describe their frequency of consumption of each food item, during the past year on a daily, weekly or monthly basis. These data were then converted to daily intake using Nutritionist IV software, which was based on a modified USA Department of Agriculture dietary composition for Iranian foods⁽³⁵⁾. A trained dietitian manually entered the data after excluding under and overreporting and entrants with missing data.

Assessment of dietary glycaemic index and glycaemic load

We estimated the total dietary GI by using the following formula: $\Sigma (\text{GI}_a \times \text{available carbohydrate}_a) / \text{total available carbohydrate}$, where available carbohydrate was calculated as total carbohydrate_a minus fibre_a⁽³⁶⁾. The total carbohydrate and fibre contents of the foods were derived from the USA Department of Agriculture food composition table. Of the 101 carbohydrate-containing foods in the FFQ, GI values for only six foods could be derived from the Iranian GI table⁽³⁷⁾ because the table does not cover the GI of all available foods. The GI values for the remaining items are obtained from the international table of GI⁽³⁸⁾, and the GI online database maintained by the University of Sydney⁽³⁹⁾. For items that were not on the table, they were estimated based on similar physical and chemical foods. All derived GI values were relative to glucose as the reference food. The GI of composite mixed meals were estimated based on the GI of individual food components⁽³⁶⁾. The dietary GL was calculated as $(\text{total GI} \times \text{total available carbohydrate}) / 100$ and expressed as g/d⁽³⁶⁾. We used the energy-adjusted quantity of total carbohydrate intake measured through the residual process, as suggested by Willett *et al.*⁽⁴⁰⁾

LCD

We calculated an LCD score for each participant based on the method introduced by Hamilton *et al.* with some adjustment in maximum score⁽⁴¹⁾. Participants were divided into quintiles of carbohydrate, protein and fat intake as a percentage of energy intakes. Women in the lowest quintile of carbohydrate intake were given a score of 4 and those in the highest quintile were given a score of 0. Other quintiles (second, third and fourth) were given the corresponding scores (3, 2 and 1, respectively). For protein and fat, we used the same scoring, but the order was reversed. Women in the lowest quintile were given a score of 0 for that macronutrient and those in the highest quintile of protein and fat intake were given a score of 4. Other quintiles (second, third and fourth) were given the corresponding scores (1, 2 and 3, respectively). To create the LCD score, macronutrient scores were summed and ranged from 0 to 12. The lowest score (0) indicated the highest carbohydrate intake and the lowest fat and protein intake, and the highest score (12) indicated the lowest carbohydrate intake and the highest protein and fat intake. Therefore, a high LCD score would represent a participant with high adherence to a low-carbohydrate diet and vice versa.

Laboratory investigation

Participants asked to be fast for at least 8–12 h. Then, 10 ml fasting-blood samples were collected between the hours of 07.00–10.00 in acid-washed test tubes without anticoagulants. After storing at room temperature for 30 min and clot formation, blood samples were centrifuged at 1500 g for 20 min. Serums were stored at -80°C until future testing. Glucose was included by the enzymatic (glucose oxidase) colorimetric method, using a commercial kit (Pars Azmun). Serum total cholesterol (TC) and HDL-cholesterol was measured using a cholesterol oxidase phenol 4-amino antipyrine method, and TG was estimated using a glycerol-3 phosphate oxidase phenol 4-amino antipyrine enzymatic method.

Statistical analysis

Energy-adjusted dietary GI, GL and LCD score were used to classify participants into tertiles. According to the type of variables, the comparison of quantitative mean variables between the tertiles of subject characteristics and anthropometric measurement was performed using ANOVA (one-way variance analysis) and comparison of qualitative variables distribution between the tertiles with χ^2 square test. The multivariate-adjusted means of anthropometric measures across tertiles of GI, GL and LCD score were compared using ANCOVA. To obtain the OR with 95% CI for a higher LAP index and VFL (based on the median values), we applied binary logistic regression in crude and multivariate-adjusted models. We used age and energy intake for the first model. Model 2 was adjusted for variables in model 1 plus education, smoking status, physical activity, occupation, marriage and underlying diseases. All statistical analyses were performed using Statistical Package for Social Science (SPSS version 26.0; IBM Corp.). The significance level of <0.05 was considered for statistical analysis.



Results

Of the 270 participants, 118 and 152 were men and women, respectively. The average age was 38 in men and 35.7 in women. Mean of GI, GL and CQI was 65.4 ± 2.9 , 265 ± 18.5 and 9.5 ± 4.5 , respectively.

The general characteristics of participants are presented in [Table 1](#). Participants with the highest GI tended to have a higher body weight ($P=0.01$), height ($P=0.01$), WC ($P=0.04$), WHR ($P=0.04$) and DBP ($P=0.03$). The mean of body weight ($P<0.001$), height ($P=0.01$), BMI ($P=0.02$), WC ($P<0.001$), WHR ($P=0.03$) and DBP ($P=0.05$) were also significantly different across tertiles of the GL. Participants with the highest GI and GL tended to be more smoker and male. Within the LCD tertiles, participants in the third tertile were less uneducated, male current smoker and physically active and to have chronic disease and more likely to be married, educated and employed compared with those in the first tertile.

The association between dietary intake of participants according to tertiles of LCD, GI and GL was shown in [Table 2](#). Comparing dietary intakes across GL and GI tertiles, we found that participants in the top tertile had a higher intake of energy, carbohydrate and fiber compared with those in the bottom tertile. Participants in the highest tertile of LCD had higher intake of carbohydrates, protein and fat intake compared with those in the lowest tertile.

Crude and multivariable-adjusted means of anthropometric indicators across tertiles of dietary GI, GL and LCD are shown in [Table 3](#). Neither in crude nor in adjusted models found a significant difference in anthropometric measures and indexes, LAP and VFL index comparing tertiles of dietary GL, GI and LCD. After adjustment for age and energy intake, women in the top tertile of GI and GL tended to have higher LAP compared with those in the bottom tertile. However, the significant association was disappeared after controlling for other potential confounders.

Crude and multivariable-adjusted OR (95% CI) for LAP and VFL within tertiles of LCD, GI and GL are depicted in [Table 4](#). There was no association between dietary GI and GL and LCD with LAP and VFL index in crude and adjusted models.

Gender-stratified crude and multivariable-adjusted OR and 95% CI for LAP and VFL across tertiles of dietary GI, GL and LCD are provided in [Table 5](#). There was no significant association between dietary GI and GL and LCD with LAP and VFL index in men. We found a significant association between LCD score and VFL in women in the crude model (OR = 0.35, 95% CI = 0.16–0.78, $P=0.01$). Women in the highest tertile of LCD score compared with those in the lowest tertile had lower chance of a high VFL (≥ 112.95) by 57% (OR = 0.43, 95% CI = 0.18–1.00, $P=0.01$) when controlled for age and energy intake in model 1. However, this significant association disappeared after controlling for other potential confounders (OR = 0.45, 95% CI = 0.19, 1.08, $P=0.07$).

Discussion

In the present study, no significant relationship was observed between GI, GL and LCD with the VFL index after controlling

for the potential confounders. Although it should be noted that when we stratified analyses by gender, women in the highest tertile of GL and GI had a significantly higher LAP after controlling for age and energy intake. Furthermore, in women who consumed lower amounts of carbohydrate and higher contents of fat and protein odds of having high VFL reduced by 65% in crude model. However, after adjusting the potential confounders, this association was no longer significant. In our study, participants in the top tertile of LCD score consumed a smaller percentage of their diet as carbohydrates and a greater amount of protein and fat. The total energy and fibre intake did not differ among the tertiles. Interestingly, in this study, the mean average of carbohydrate intake in the highest tertile (49%) was still above the definition of LCD ($< 45\%$ of total energy)⁽⁴²⁾.

Our study found no significant relationship between LCD score, GI and GL and OR of high LAP and VFL. Previous research concerning these associations in other populations has shown mixed reports. In a cross-sectional study among 209 Iranian women aged 20–50 years, the odds of obesity and cardiovascular risk factors were not associated with different tertiles of LCD score⁽⁴³⁾.

In contrast to our results, a large prospective study, which included 48 631 men and women from five countries participating in the European Prospective Investigation into Cancer and Nutrition study, concluded that a diet with low GI may prevent visceral adiposity⁽⁴⁴⁾. However, in the insulin resistance atherosclerosis study on 979 American adults, GI and GL were not related to measures of insulin sensitivity, BMI, WC and adiposity⁽⁴⁵⁾. In another study by Mazidi *et al.*, on American men and women, participated in the cohort of National Health and Nutrition Examination Surveys, it was found that participants who followed a diet high in carbohydrates, sugar, total fat and saturated fat, had a high LAP and VAI levels. In contrast, those who followed a diet high in vitamins, minerals and fibre and had lower LAP and VAI levels⁽⁴⁶⁾. A Danish cohort study in 185 men and 191 women found that high-GI diets may lead to weight gain, body fat mass and WC were associated with women, but not in men⁽⁴⁷⁾.

Discrepancies in findings of studies can be explained as follows: our study population consisted of adults with a normal BMI and WC, while in many of trials concerning the effects of GL and GI on anthropometric measurement, the study population had type 2 diabetes or MetS with higher mean weight, BMI, and probably abnormal body composition^(48–50). Our findings are in line with the fact that Iranians adults receive more than 60% of their energy from carbohydrates, especially refined grains with a high GI and GL⁽⁵¹⁾. However, the variance of intake of carbohydrate in this population was not high, which may explain the non-significant results we found in the study. Demographic, cultural and economic differences between different regions and populations can also affect different findings across studies. Inconclusive results among studies may also be due to the age and gender differences. The association of carbohydrate quantity and quality with chronic diseases has been widely published without enough publication stratified by age and gender. It has been reported that GI of sucrose is theoretically 50% of glucose for all the populations. However, Ishii *et al.* showed that GI was



Table 1. General characteristics of study participants across tertiles of low-carbohydrate diet score, glycaemic index and glycaemic load (Mean values and standard deviations)

	GI							GL							LCD						
	T1	T2	T3	<i>P</i> ²				T1	T2	T3	<i>P</i>				T1	T2	T3	<i>P</i>			
Age (years)	12.9	36.1	12.4	35.2	13.8	38.4	0.23	36.3	13.2	35.1	12.2	38.3	13.7	0.27	41.4	13	34.1	13.9	33.7	11.1	< 0.001
Weight (kg)	14.8	68.1	15.8	72.8	16.1	77.2	0.01	67.9	14.7	72.7	16.2	77.5	16	< 0.001	73.6	13.5	71.1	17.5	73.1	17.3	0.58
Height (cm)	9.6	164	9.3	169	10.2	170	0.01	164	9.5	169	9.4	170	10.2	0.01	167	9.8	167	10	169	9.9	0.09
BMI (kg/m ²)	24.9	4.4	25.3	4.8	26.5	4.6	0.06	24.9	4.4	25.5	4.9	26.6	4.5	0.02	26.3	4.2	25.2	4.7	25.1	4.9	0.16
WC (cm)	86.6	11.8	89.3	12.2	92.8	13.1	0.04	86.5	11.6	89.1	12.5	93.1	12.8	0.00	91	10.7	88.5	13.9	89	13.2	0.35
WHR	0.88	0.06	0.90	0.05	0.91	0.07	0.04	0.89	0.06	0.90	0.06	0.92	0.07	0.03	0.91	0.05	0.89	0.07	0.89	0.06	0.17
SBP (mm Hg)	107	19.3	112	17	113	19.1	0.08	108	19.5	112	16.8	113	20.6	0.10	112	22.1	111	18.4	110	16.5	0.73
DBP (mm Hg)	69.3	8.6	69.5	12.8	73	9.8	0.03	69.3	8.8	69.7	12.7	72.8	9.9	0.05	72	13.1	70.1	9	69.6	9.06	0.26
Sex	% < 0.001							% < 0.001							% < 0.001						
Female	44	33.3	22.7					44.7	32.7	22.7					36	28.7	35.3				
Male	19.5	33.9	46.6					18.6	34.7	46.6					35.6	27.1	37.3				
Education	0.67							0.73							< 0.001						
Illiterate	0	0	100					0	0	100					100	0	0				
Under diploma	35	40	25					35	35	30					70	20	10				
Diploma	32	28	40					32	28	40					44	24	32				
Educated	33.5	34.5	32					33.5	35	31.5					29.9	29.9	40.1				
Occupation	0.16							0.73							< 0.001						
Employee	28	32.9	39.2					28	31.5	40.6					35	28.7	36.4				
Housekeeper	38.6	36.4	25					36.4	38.6	25					50	13.6	36.4				
Retired	33.3	23.8	42.9					38.1	23.8	38.1					52.4	38.1	9.5				
Unemployed	41.7	36.7	21.7					41.7	38.3	20					21.7	33.3	45				
Marriage	0.16							0.12							< 0.001						
Single	35.7	36.5	27.8					35.7	37.4	27					22.6	34.8	42.6				
Married	31.7	31.7	36.6					31	31.7	37.3					46.5	21.1	32.4				
Divorced	28.6	0	71.4					28.6	0	71.4					28.6	57.1	14.3				
Dead spouse	33.3	66.7	0					66.7	33.3	0					66.7	33.3	0				
Life style	0.370							0.370							0.75						
Alone	37.5	20.8	41.7					37.5	20.8	41.7					29.2	29.2	41.7				
With someone	32.8	34.8	32.4					32.8	34.8	32.4					36.5	27.9	35.7				
Smoking	< 0.001							< 0.001							0.36						
Not smoking	35.8	35.8	28.4					35.3	36.2	28.4					37.1	27.2	35.8				
Quit smoking	14.3	28.6	57.1					21.4	21.4	57.1					35.7	42.9	21.4				
Smoker	18.2	13.6	68.2					18.2	13.6	68.2					22.7	27.3	50				
Activity score	0.338							0.238							< 0.001						
Low	31.4	40.2	28.4					31.4	40.2	28.4					47.1	23.5	29.4				
Moderate	36.9	28.8	34.2					37.8	28.8	33.3					33.3	31.5	35.1				
High	29.1	30.9	40					27.3	30.9	41.8					20	29.1	50.9				
Metabolic diseases	0.349							0.319							0.04						
Yes	28.9	28.9	42.2					31.1	26.7	42.2					44.4	35.6	20				
No	34.2	34.7	31.1					33.8	35.1	31.1					33.8	26.6	39.6				

GI, glycaemic index; GL, glycaemic load; LCD, low-carbohydrate diet score; T, tertile; WC, waist circumference; WHR, waist:hip ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; Y, year; Cm, centimetre; kg/m², kilogram/metre²; Mm Hg, millimetres of mercury. All values are means ± standard deviation. P values result from ANOVA for quantitative variables and χ^2 test for qualitative variables.

Carbohydrate intake and lipid accumulation product

Table 2. Dietary intake of participants according to tertiles of low-carbohydrate diet score, glycaemic index and glycaemic load (Mean values and standard deviations)

	GI									GL									LCD																
	T1			T2			T3			P	T1			T2			T3			P	T1			T2			T3			P					
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean		SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD		Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean		SD				
Energy (Kcal/d)	1703	754	2418	485	3051	1048	< 0.001	1724	772	2413	502	3034	1051	< 0.001	2456	1097	2238	766	2444	959	0.27	220	220	2444	959	0.27	220	220	2444	959	0.27				
Carbohydrate (% of total energy)	55.8	7.94	56.8	7.90	58.8	7.36	0.03	55.7	7.92	56.9	7.87	58.8	7.37	0.02	65	4.01	57.4	2.71	49.1	4.60	< 0.001	48	4.01	57.4	2.71	49.1	4.60	< 0.001	48	4.01	57.4	2.71	49.1	4.60	< 0.001
Fat (% of total energy)	31.3	7.85	30.3	7.36	27.9	6.58	0.01	31.5	7.97	30.1	7.14	27.8	6.61	0.00	23.5	4.55	30	4.78	35.9	6.12	< 0.001	23.5	4.55	30	4.78	35.9	6.12	< 0.001	23.5	4.55	30	4.78	35.9	6.12	< 0.001
Protein (% of total energy)	15.2	3.41	14.9	3.14	15.4	3.19	0.62	15.1	3.44	14.9	3.12	15.3	3.19	0.70	13.6	1.67	14.7	2.55	17	3.91	< 0.001	13.6	1.67	14.7	2.55	17	3.91	< 0.001	13.6	1.67	14.7	2.55	17	3.91	< 0.001
Total fibre (g/d)	11.6	4.03	16.1	5.97	20.5	9.13	< 0.001	11.9	4.68	16	5.83	20.3	9.16	< 0.001	16.9	7.21	15.5	7.07	15.7	8.37	0.41	16.9	7.21	15.5	7.07	15.7	8.37	0.41	16.9	7.21	15.5	7.07	15.7	8.37	0.41

GI, glycaemic index; GL, glycaemic load; LCD, low-carbohydrate diet score; T, tertile. P values result from ANOVA for quantitative variables. All values are means ± standard deviation.

different in young *v.* older men in which younger men responded (82 %) better than older men (73.6%)⁽⁵²⁾. In a study by Fan *et al.*⁽⁵³⁾, the results showed a gender modification on the effects of glycaemic index and glycaemic load on cardiovascular risk, with higher risk of CVD in women but not in men. Also, the risk of mortality from strokes increased in women with higher adherence to GI⁽⁵⁴⁾. In a study by Ishii *et al.*, they also showed that GI of sucrose in young men was not significantly different from the GI of glucose in men, but GI of sucrose in young women was 67.8 % of GI of glucose, and there was a different response according to the age and gender when used substances of distinct structures such as glucose or sucrose⁽⁵²⁾. There are gender differences in fat mass and fat free mass throughout life that leads to a difference in energy metabolism and utility of macronutrients^(55,56), which may be related to sex steroids, differences in insulin resistance or metabolic effects of other hormones such as leptin. In a study by Tamopolsky *et al.*, it was found that there was a gender differences in absolute and relative carbohydrate intake that can be related to the inability of women to carbohydrate load⁽⁵⁷⁾.

To measure adiposity indicators, the gold standard methods like computed tomography scan or MRI and dual-energy X-ray absorptiometry can be used. However, using such examinations in general practice are limited due to being expensive and less availability and need for expertise in medical imaging. In this study we used LAP as a measure of the accumulation of fat mass in the body. Many previous studies have shown that the LAP as a simple, reliable and inexpensive tool was closely related to chronic disease like diabetes, CVD, poly cystic ovarian syndrome, obesity and Mets⁽⁵⁸⁻⁶⁰⁾. It also can be used as a reliable indicator of visceral adiposity for early detection of cardiovascular risk in different age groups^(61,62) and populations^(63,64). In addition, some studies reported that bioimpedance analysis has enough reliability to show adipose tissue distribution and to monitor visceral fat⁽⁶⁵⁻⁶⁷⁾. However, it was not confirmed in other studies.

This study is one of the few studies in developing countries investigating the relationship between GI, GL and LCD with LAP and VFL index. We also applied body composition analysis and measured two newly developed indicators of visceral fat accumulation rather only classical anthropometric indexes. Another strength of this study is controlling for a wide range of potential confounders to achieve an independent association. Valid questionnaires have been also used to collect data that can support the accuracy of the findings. However, despite the strengths of our study, some limitations need to be considered. First, due to the cross-sectional nature of the present study, causal relationships between GI, GL and LCD cannot be inferred from LAP and VFL. Like all epidemiological studies, participants' incorrect classification due to FFQ use is inevitable. However, we tried to improve some of the wrong classifications by using trained nutritionists to collect the relevant data. Despite the modification of several confounders, the potential effects of residual confounders cannot be ruled out. In addition, our findings from representative sample of Tehran are only generalisable to Iranian population and may be not be generalised to the Iranian population. We also did not use the gold standard methods to measure VFL and did not compare the result of LAP index with actual physical measurements of visceral adiposity by dual-

Table 3. Gender-stratified crude and multivariable-adjusted means for anthropometric measures across tertiles of low-carbohydrate diet score, glycaemic index and glycaemic load (Mean values and standard deviations)

	GI							GL						LCD								
	T1		T2		T3		P ²	T1		T2		T3		P	T1		T2		T3		P	
	Mean	SD	Mean	SD	Mean	SD		Mean	SD	Mean	SD	Mean	SD		Mean	SD	Mean	SD	Mean	SD		
Men Weight (kg)																						
<i>n</i> (117)																						
Crude	82.1	15.5	79.9	14.1	83.6	14.6	0.48	81.9	15.8	80.1	14	83.6	14.6	0.51	80.7	11.9	82.5	18	83.1	14.5	0.73	
Model 1	82.1	15.5	79.9	14.1	83.6	14.6	0.69	81.9	15.8	80.1	14	83.6	14.6	0.75	80.7	11.9	82.5	18	83.1	14.5	0.56	
Model 2	82.1	15.5	79.9	14.1	83.6	14.6	0.77	81.9	15.8	80.1	14	83.6	14.6	0.80	80.7	11.9	82.5	18	83.1	14.5	0.43	
BMI (kg/m²) <i>n</i>(117)																						
Crude	26.1	4.19	25.6	4.2	26.9	4.14	0.33	26	4.27	25.7	3.96	26.9	4.11	0.34	26.4	3.25	26.6	5.06	26	4.11	0.84	
Model 1	26.1	4.19	25.6	4	26.9	4.14	0.68	26	4.27	25.7	3.96	26.9	4.14	0.74	26.4	3.25	26.6	5.06	26	4.16	0.61	
Model 2	26.1	4.19	25.6	4	26.9	4.14	0.80	26	4.27	25.7	3.96	26.9	4.14	0.85	26.4	3.25	26.6	5.06	26	4.16	0.49	
WC (cm) <i>n</i>(117)																						
Crude	92.9	13.5	91.8	11.2	95.9	12.7	0.26	92.5	13.7	92	11.1	95.9	12.7	0.28	93.7	9.58	94.5	15.5	93.7	12.6	0.95	
Model 1	92.9	13.5	91.8	11.2	95.9	12.7	0.62	92.5	13.7	92	11.1	95.9	12.7	0.68	93.7	9.58	94.5	15.5	93.7	12.6	0.70	
Model 2	92.9	13.5	91.8	11.2	95.9	12.7	0.67	92.5	13.7	92	11.1	95.9	12.7	0.89	93.7	9.58	94.5	15.5	93.7	12.6	0.45	
WHR <i>n</i>(117)																						
Crude	0.9	0.08	0.9	0.06	0.93	0.07	0.21	0.9	0.08	0.91	0.06	0.93	0.07	0.20	0.92	0.05	0.92	0.08	0.91	0.07	0.82	
Model 1	0.9	0.08	0.9	0.06	0.93	0.07	0.58	0.9	0.08	0.91	0.06	0.93	0.07	0.59	0.92	0.05	0.92	0.08	0.91	0.07	0.88	
Model 2	0.9	0.08	0.91	0.06	0.93	0.07	0.82	0.9	0.08	0.91	0.06	0.93	0.07	0.84	0.92	0.05	0.92	0.08	0.91	0.07	0.69	
LAP <i>n</i>(110)																						
Crude	3190	2415	3647	2365	3737	2424	0.66	3175	2474	3643	2333	3737	2424	0.66	3370	1832	3604	2696	3804	2648	0.72	
Model 1	3190	2415	3647	2365	3737	2424	0.88	3175	2474	3643	2333	3737	2424	0.87	3370	1832	3604	2696	3804	2648	0.43	
Model 2	3190	2415	3647	2365	3737	2424	0.85	3175	2474	3643	2333	3737	2424	0.86	3370	1832	3604	2696	3804	2648	0.46	
VFL(cm²) <i>n</i>(116)																						
Crude	80.8	49.9	86.1	39.8	102	45.3	0.07	83.6	49.2	84.4	40.6	102	45.3	0.08	91.2	38.3	98.8	50.1	90.4	47.9	0.70	
Model 1	80.8	49.9	86.1	39.8	102	45.3	0.31	83.6	49.2	84.4	40.6	102	45.3	0.31	91.2	38.3	98.8	50.1	90.4	47.9	0.38	
Model 2	80.8	49.9	86.1	39.8	102	44.3	0.50	83.6	49.2	84.4	40.6	102	45.3	0.50	91.2	38.3	98.8	50.1	90.4	47.9	0.14	
Women Weight (kg)																						
<i>n</i> (149)																						
Crude	63.3	11.1	67.4	14.9	66.7	13.8	0.20	63.3	11	66.8	15.5	67.6	13.1	0.21	68.3	12.3	63.1	11.8	64.4	14.7	0.11	
Model 1	63.3	11.1	67.4	14.9	66.7	13.8	0.40	63.3	11	66.8	15.5	67.6	13.1	0.76	68.3	12.3	63.1	11.8	63.1	14.5	0.65	
Model 2	63.2	11.2	67.4	14.9	66.4	13.9	0.42	63.2	11	66.8	15.5	67.3	13.2	0.69	68.2	12.4	62.9	11.9	64.4	14.7	0.62	
BMI (kg/m²) <i>n</i>(149)																						
Crude	24.5	4.44	25	5.47	25.8	5.42	0.45	24.5	4.41	24.8	5.63	26.2	5.16	0.27	26.2	4.93	24.2	4.36	24.4	4.36	0.07	
Model 1	24.5	4.44	25	5.47	25.8	5.42	0.84	24.5	4.11	24.8	5.63	26.2	5.16	0.95	26.2	4.93	24.2	4.36	24.4	5.47	0.81	
Model 2	24.5	4.46	25	5.47	25.8	5.49	0.91	24.4	4.43	24.8	5.63	26.1	5.23	0.95	26.2	4.97	24.1	4.38	24.4	5.47	0.75	
WC (cm) <i>n</i>(149)																						
Crude	84.4	10.3	87.3	12.7	88	12.4	0.25	84.5	10.2	86.8	13.1	88.6	12	0.22	89.1	11.2	84.2	10.9	84.9	12.4	0.07	
Model 1	84.4	10.3	87.3	12.7	88	12.4	0.61	84.5	10.2	86.8	13.1	88.6	12	0.94	89.1	11.2	84.2	10.9	84.9	12.4	0.65	
Model 2	83.9	10.1	87.4	12.9	87.5	12.6	0.36	84.5	10.3	86.8	13.1	88.5	12.2	0.92	89	11.3	84.1	11	84.9	12.4	0.62	
WHR <i>n</i>(149)																						
Crude	0.88	0.05	0.89	0.05	0.89	0.06	0.17	0.88	0.05	0.89	0.05	0.9	0.06	0.21	0.9	0.05	0.88	0.05	0.88	0.05	0.04	
Model 1	0.88	0.05	0.89	0.05	0.89	0.06	0.58	0.88	0.05	0.89	0.05	0.9	0.06	0.86	0.9	0.05	0.88	0.05	0.88	0.05	0.44	
Model 2	0.88	0.05	0.89	0.05	0.89	0.06	0.71	0.88	0.05	0.89	0.05	0.9	0.06	0.88	0.9	0.05	0.88	0.05	0.88	0.05	0.45	
LAP <i>n</i>(143)																						
Crude	2886	1902	2845	1872	4036	2922	0.02	2815	1869	2927	1946	4059	2883	0.02	3354	2516	2909	1611	3119	2336	0.63	
Model 1	2886	1902	2845	1872	4036	2922	0.04	2815	1869	2927	1946	4059	2883	0.02	3354	2516	2909	1611	3119	2336	0.93	
Model 2	2906	1911	2845	1872	4115	2933	0.12	2833	1879	2927	1946	4138	2892	0.06	3339	2529	2939	1618	3119	2336	0.81	
VFL (cm²) <i>n</i>(149)																						
Crude	109	46.4	112	49	122	52.7	0.47	110	45.7	110	50.7	123	51.4	0.37	124	48.3	106	45.5	107	50.5	0.10	
Model 1	109	46.4	112	49	122	52.7	0.70	110	45.7	110	50.7	123	51.4	0.76	124	48.3	106	45.5	107	50.5	0.75	
Model 2	110	46.8	112	49	122	53.6	0.64	110	46	110	50.7	123	52.2	0.59	124	48.8	106	46	107	50.5	0.78	

Carbohydrate intake and lipid accumulation product

Table 4. Crude and multivariable-adjusted OR (95 % CI) for LAP and VFL tertiles of low-carbohydrate diet score, glycaemic index and glycaemic load* (Odd ratio and 95 % confidence intervals)

	T1	P value†	T2		P value	T3		P value
			OR	95 % CI		OR	95 % CI	
GI VFL(cm²) ≥ 99.3								
Crude	1 reference	0.64	0.93	0.52, 1.68	0.82	1.22	0.68, 2.21	0.49
Model 1‡	1 reference	0.92	0.95	0.50, 1.83	0.90	1.09	0.51, 2.30	0.82
Model 2§	1 reference	0.71	0.82	0.41, 1.61	0.56	1.06	0.48, 2.35	0.87
LAP ≥ 2736.3								
Crude	1 reference	0.21	1.29	0.70, 2.37	0.40	1.73	0.94, 3.18	0.07
Model 1	1 reference	0.86	1.17	0.60, 2.28	0.64	1.21	0.56, 2.60	0.62
Model 2	1 reference	0.89	1.18	0.59, 2.36	0.63	1.10	0.49, 2.43	0.81
GL VFL (cm²) ≥ 99.3								
Crude	1 reference	0.57	0.85	0.47, 1.53	0.60	1.17	0.65, 2.11	0.59
Model 1	1 reference	0.84	0.87	0.45, 1.67	0.69	1.04	0.49, 2.18	0.90
Model 2	1 reference	0.57	0.95	0.43, 2.09	0.91	0.72	0.36, 1.44	0.35
LAP ≥ 2736.3								
Crude	1 reference	0.15	1.42	0.77, 2.61	0.25	1.81	0.98, 3.33	0.05
Model 1	1 reference	0.64	1.34	0.69, 2.61	0.38	1.34	0.63, 2.86	0.43
Model 2	1 reference	0.59	1.43	0.71, 2.85	0.31	1.21	0.55, 2.64	0.62
LCD VFL (cm²) ≥ 99.3								
Crude	1 reference	0.25	0.81	0.44, 1.49	0.50	0.62	0.35, 1.09	0.09
Model 1	1 reference	0.67	1.12	0.58, 2.16	0.72	0.85	0.46, 1.55	0.60
Model 2	1 reference	0.63	1.39	0.70, 2.75	0.34	1.12	0.58, 2.13	0.72
LAP ≥ 2736.3								
Crude	1 reference	0.75	0.80	0.42, 1.50	0.48	0.83	0.46, 1.49	0.54
Model 1	1 reference	0.92	1.12	0.52, 2.19	0.73	1.11	0.60, 2.08	0.72
Model 2	1 reference	0.89	1.13	0.55, 2.30	0.72	1.16	0.60, 2.24	0.65

GI, glycaemic index; GL, glycaemic load; LCD, low-carbohydrate diet score; LAP, lipid accumulation product; VFL, visceral fat level.

* Data are OR (95 % CI).

† Obtained from logistic regression.

‡ Model I: adjusted for age and energy intake.

§ Model II: additionally adjusted for age and energy intake, education, smoking status, physical activity, occupation, marriage and metabolic diseases.

Table 5. Gender-stratified crude and multivariable-adjusted OR (95 % CI) for LAP and VFL tertiles of low-carbohydrate diet score, glycaemic index and glycaemic load* (Odd ratio and 95 % confidence intervals)

	T1	P value†	T2		P value	T3		P value
			OR	95 % CI		OR	95 % CI	
GI								
Men VFL(cm²) ≥ 87.4 n (118)								
Crude	1 reference	0.17	1.20	0.42, 3.43	0.73	2.26	0.83, 6.13	0.10
Model 1‡	1 reference	0.40	1.11	0.37, 3.31	0.84	1.89	0.62, 5.76	0.28
Model 2§	1 reference	0.65	1.09	0.34, 3.48	0.87	1.59	0.49, 5.14	0.43
LAP ≥ 3145.5 n (118)								
Crude	1 reference	0.27	1.65	0.56, 4.89	0.36	2.30	0.82, 6.46	0.11
Model 1	1 reference	0.72	1.45	0.47, 4.44	0.51	1.58	0.50, 5.01	0.43
Model 2	1 reference	0.82	1.47	0.43, 5.05	0.53	1.30	0.36, 4.70	0.68
Women VFL ≥ 112.95 n (152)								
Crude	1 reference	0.27	0.92	0.44, 1.93	0.84	1.82	0.78, 4.24	0.16
Model 1	1 reference	0.50	0.58	0.22, 1.55	0.28	0.83	0.25, 2.77	0.77
Model 2	1 reference	0.32	0.50	0.18, 1.40	0.19	0.85	0.24, 3.02	0.80
LAP ≥ 2575 n (152)								
Crude	1 reference	0.21	0.88	0.41, 1.89	0.76	1.92	0.81, 4.57	0.13
Model 1	1 reference	0.53	0.74	0.28, 1.97	0.55	1.27	0.38, 4.18	0.69
Model 2	1 reference	0.52	0.62	0.22, 1.79	0.38	1.04	0.29, 3.75	0.94
GL								
Men VFL ≥ 87.4 n (118)								
Crude	1 reference	0.17	1.06	0.37, 3.07	0.90	2.10	0.76, 5.76	0.14
Model 1	1 reference	0.41	0.98	0.32, 2.94	0.97	1.74	0.56, 5.34	0.33
Model 2	1 reference	0.66	0.95	0.29, 3.06	0.93	1.45	0.44, 4.77	0.53
LAP ≥ 3145.5 n (118)								
Crude	1 reference	0.20	2	0.66, 6.05	0.22	2.63	0.91, 7.63	0.07

Table 5. (Continued)

	T1	P value†	T2		P value	T3		P value
			OR	95 % CI		OR	95 % CI	
Model 1	1 reference	0.54	1.78	0.56, 5.58	0.32	1.84	0.56, 5.96	0.30
Model 2	1 reference	0.68	1.79	0.51, 6.29	0.36	1.51	0.41, 5.58	0.53
Women VFL ≥ 112.95 n (152)								
Crude	1 reference	0.28	0.98	0.47, 2.06	0.97	1.87	0.80, 4.35	0.14
Model 1	1 reference	0.65	0.72	0.27, 1.85	0.49	1.06	0.34, 3.31	0.91
Model 2	1 reference	0.43	0.62	0.23, 1.64	0.34	1.11	0.34, 3.60	0.85
LAP ≥ 2575 n (152)								
Crude	1 reference	0.22	0.95	0.44, 2.03	0.89	1.98	0.83, 4.69	0.12
Model 1	1 reference	0.55	0.88	0.33, 2.29	0.79	1.51	0.48, 4.70	0.47
Model 2	1 reference	0.77	0.88	0.31, 2.46	0.81	1.30	0.37, 4.52	0.67
LCD								
Men VFL ≥ 87.4 n (118)								
Crude	1 reference	0.91	1.20	0.46, 3.08	0.70	1	0.43, 2.34	0.99
Model 1	1 reference	0.56	1.67	0.60, 4.64	0.32	1.48	0.59, 3.74	0.39
Model 2	1 reference	0.39	1.85	0.62, 5.49	0.26	1.92	0.68, 5.39	0.21
LAP ≥ 3145.5 n (118)								
Crude	1 reference	0.76	0.69	0.26, 1.82	0.46	0.85	0.35, 2.05	0.72
Model 1	1 reference	0.92	0.90	0.32, 2.51	0.85	0.84	1.10, 0.43	0.84
Model 2	1 reference	0.94	1.03	0.34, 3.04	0.95	1.18	0.41, 3.35	0.75
Women VFL ≥ 112.95 n (152)								
Crude	1 reference	0.02	0.47	0.21, 1.06	0.07	0.35	0.16, 0.78	0.01
Model 1	1 reference	0.14	0.66	0.27, 1.59	0.36	0.43	0.18, 1	0.05
Model 2	1 reference	0.19	0.76	0.30, 1.92	0.57	0.45	0.19, 1.08	0.07
LAP ≥ 2575 n (152)								
Crude	1 reference	0.99	0.96	0.42, 2.18	0.92	0.96	0.44, 2.09	0.91
Model 1	1 reference	0.68	1.46	5.91, 3.62	0.41	1.32	0.57, 3.08	0.51
Model 2	1 reference	0.68	1.47	0.54, 4.04	0.44	1.43	0.57, 3.61	0.43

GI, glycaemic index; GL, glycaemic load; LCD, low-carbohydrate diet score; LAP, lipid accumulation product; VFL, visceral fat level.

* Data are OR (95 % CI).

† Obtained from logistic regression.

‡ Model I: adjusted for age and energy intake.

§ Model II: additionally adjusted for age and energy intake, education, smoking status, physical activity, occupation, marriage and metabolic diseases.

energy X-ray absorptiometry, MRI or computed tomography scan. Moreover, people in Middle East countries like Iran consume higher intake of their energy from carbohydrate, and then a modified version of LCD score with a new range is needed to investigate associations.

Conclusion

Our finding suggests that carbohydrate quality indexes and LCD score are not associated with LAP and VFL index. However, women with a high adherence to LCD tended to have lower VFL. Further well-designed studies are required to confirm these findings.

Acknowledgements

This paper is a part of the master thesis of Fatemeh Gholami. Special thanks go to all those who participated in this study.

This study has been granted by Tehran University of Medical Sciences (Grant No: 33 887).

S. S. B. contributed to conception/design of the research; N. B. and S. D. contributed to acquisition of data. F. G., F. M. and A.M. participated in the analysis and interpretation of the data; F. G., M. E. and P. G. drafted the manuscript; K. D., S. S. B. and F. M. critically revised the manuscript and S. S. B. agrees

to be fully accountable for ensuring the integrity and accuracy of the work. All authors read and approved the final manuscript.

All authors hereby declare that they have no conflict of interest.

References

1. Arroyo-Johnson C & Mincey KD (2016) Obesity epidemiology worldwide. *Gastroenterol Clin* **45**, 571–579.
2. Shelgikar K, Yajnik C & Hockaday T (1991) Central rather than generalized obesity is related to hyperglycaemia in Asian Indian subjects. *Diabetic Med* **8**, 712–717.
3. McKeigue P, Shah B & Marmot M (1991) Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* **337**, 382–386.
4. Djausal AN (2015) Effect of central obesity as risk factor of metabolic syndrome. *J Majority* **4**, 9–22.
5. Arsenault B, Rana J, Lemieux I, *et al.* (2010) Physical inactivity, abdominal obesity and risk of coronary heart disease in apparently healthy men and women. *Int J Obes* **34**, 340–347.
6. Kanaya AM, Vittinghoff E, Shlipak MG, *et al.* (2003) Association of total and central obesity with mortality in postmenopausal women with coronary heart disease. *Am J Epidemiol* **158**, 1161–1170.
7. Nagrani R, Mhatre S, Rajaraman P, *et al.* (2016) Central obesity increases risk of breast cancer irrespective of menopausal and hormonal receptor status in women of South Asian Ethnicity. *Eur J Cancer* **66**, 153–161.

8. Harvie M, Hooper L & Howell A (2003) Central obesity and breast cancer risk: a systematic review. *Obes Rev* **4**, 157–173.
9. Hamer M, O'Donovan G, Stensel D, *et al.* (2017) Normal-weight central obesity and risk for mortality. *Ann Intern Med* **166**, 917–918.
10. Brown JC, Harhay MO & Harhay MN (2017) Anthropometrically-predicted visceral adipose tissue and mortality among men and women in the third national health and nutrition examination survey (NHANES III). *Am J Hum Biol* **29**, e22898.
11. Després JP (2012) Body fat distribution and risk of cardiovascular disease: an update. *Circulation* **126**, 1301–1313.
12. Hayashi T, Boyko EJ, Leonetti DL, *et al.* (2003) Visceral adiposity and the risk of impaired glucose tolerance: a prospective study among Japanese Americans. *Diabetes Care* **26**, 650–655.
13. Kim SK, Kim HJ, Hur KY, *et al.* (2004) Visceral fat thickness measured by ultrasonography can estimate not only visceral obesity but also risks of cardiovascular and metabolic diseases. *Am J Clin Nutr* **79**, 593–599.
14. Hayashi T, Boyko EJ, Leonetti DL, *et al.* (2004) Visceral adiposity is an independent predictor of incident hypertension in Japanese Americans. *Ann Intern Med* **140**, 992–1000.
15. Fujimoto WY, Bergstrom RW, Boyko EJ, *et al.* (1999) Visceral adiposity and incident coronary heart disease in Japanese-American men. The 10-year follow-up results of the Seattle Japanese-American Community Diabetes Study. *Diabetes Care* **22**, 1808–1812.
16. Mirmiran P, Bahadoran Z & Azizi F (2014) Lipid accumulation product is associated with insulin resistance, lipid peroxidation, and systemic inflammation in type 2 diabetic patients. *Endocrinol Metab* **29**, 443–449.
17. Wakabayashi I & Daimon T (2014) A strong association between lipid accumulation product and diabetes mellitus in Japanese women and men. *J Atheroscler Thromb* **21**, 282–288.
18. Oh JY, Sung YA & Lee HJ (2013) The lipid accumulation product as a useful index for identifying abnormal glucose regulation in young Korean women. *Diabetic Med: J Br Diabetic Assoc* **30**, 436–442.
19. Kahn HS (2005) The 'lipid accumulation product' performs better than the body mass index for recognizing cardiovascular risk: a population-based comparison. *BMC Cardiovasc Disord* **5**, 26.
20. Ray L, Ravichandran K & Nanda SK (2018) Comparison of lipid accumulation product index with body mass index and waist circumference as a predictor of metabolic syndrome in Indian population. *Metab Syndrome Relat Disord* **16**, 240–245.
21. Jebb SA & Moore MS (1999) Contribution of a sedentary lifestyle and inactivity to the etiology of overweight and obesity: current evidence and research issues. *Med Sci Sport Exerc* **31**, S534–41.
22. Bulló M, Casas-Agustench P, Amigó-Correig P, *et al.* (2007) Obesity and comorbidities: the role of diet. *Public Health Nutr* **10**, 1164–1172.
23. Bahreinian M & Esmailzadeh A (2012) Opinion: quantity and quality of carbohydrate intake in Iran: a target for nutritional intervention. *Arch Iran Med* **15**, 648–649.
24. Slyper AH (2013) The influence of carbohydrate quality on cardiovascular disease, the metabolic syndrome, type 2 diabetes, and obesity—an overview. *J Pediatr Endocrinol Metab* **26**, 617–629.
25. de Mello Fontanelli M, Sales CH, Carioca AAF, *et al.* (2018) The relationship between carbohydrate quality and the prevalence of metabolic syndrome: challenges of glycemic index and glycemic load. *Eur J Nutr* **57**, 1197–1205.
26. Veum VL, Laupsa-Borge J, Eng Ø, *et al.* (2017) Visceral adiposity and metabolic syndrome after very high-fat and low-fat isocaloric diets: a randomized controlled trial. *Am J Clin Nutr* **105**, 85–99.
27. Kendall A, Levitsky DA, Strupp BJ, *et al.* (1991) Weight loss on a low-fat diet: consequence of the imprecision of the control of food intake in humans. *Am J Clin Nutr* **53**, 1124–1129.
28. Shai I, Schwarzfuchs D, Henkin Y, *et al.* (2008) Weight loss with a low-carbohydrate, Mediterranean, or low-fat diet. *N Engl J Med* **359**, 229–241.
29. Mazloom Z, Kazemy F, Tabatabai S, *et al.* (2009) Comparison of the effect of low-glycemic index *v.* low-fat diet on body fat and waist-hip ratio in obese women. *J Gorgan Univ Med Sci* **11**, 33–38.
30. Amini MR, Shahinfar H, Babaei N, *et al.* (2020) Association of dietary patterns with visceral adiposity, lipid accumulation product, and triglyceride-glucose index in Iranian adults. *Clin Nutr Res* **9**, 145.
31. Moghaddam MHB, Aghdam F, Asghari Jafarabadi M, *et al.* (2012) The Iranian Version of International Physical Activity Questionnaire (IPAQ) in Iran: content and Construct validity, factor structure, internal consistency and stability. *World Appl Sci J* **18**, 1073–1080.
32. Kouchi M (2014) *Anthropometric Methods for Apparel Design: Body Measurement Devices and Techniques*. *Anthropometry, Apparel Sizing and Design*. pp. 67–94. Duxford: Elsevier.
33. Motamed N, Razmjou S, Hemmasi G, *et al.* (2016) Lipid accumulation product and metabolic syndrome: a population-based study in northern Iran, Amol. *J Endocrinol Investig* **39**, 375–382.
34. Esfahani FH, Asghari G, Mirmiran P, *et al.* (2010) Reproducibility and relative validity of food group intake in a food frequency questionnaire developed for the Tehran Lipid and Glucose Study. *J Epidemiol* **20**, 150–158.
35. Haytowitz D, Lemar L, Pehrsson P, *et al.* (2011) *USDA National Nutrient Database for Standard Reference, Release 24*. Washington, DC: US Department of Agriculture.
36. Wolever TM, Yang M, Zeng XY, *et al.* (2006) Food glycemic index, as given in glycemic index tables, is a significant determinant of glycemic responses elicited by composite breakfast meals. *Am J Clin Nutr* **83**, 1306–1312.
37. Taleban F & Esmaeili M (1999) *Glycemic Index of Iranian Foods*. National Nutrition and Food Technology Research Institute Publication.
38. Foster-Powell K, Holt SH & Brand-Miller JC (2002) International table of glycemic index and glycemic load values: 2002. *Am J Clin Nutr* **76**, 5–56.
39. TUoSgiaGd (2005) Glycemic Index Research and GI News. <https://glycemicindex.com> (accessed April 2022).
40. Willett W & Stampfer MJ (1986) Total energy intake: implications for epidemiologic analyses. *Am J Epidemiol* **124**, 17–27.
41. Halton TL, Willett WC, Liu S, *et al.* (2006) Low-carbohydrate-diet score and the risk of coronary heart disease in women. *N Engl J Med* **355**, 1991–2002.
42. Last AR & Wilson SF (2006) Low-carbohydrate diets. *Am Fam Phys* **73**, 1942–1948.
43. Jafari-Maram S, Daneshzad E, Brett NR, *et al.* (2019) Association of low-carbohydrate diet score with overweight, obesity and cardiovascular disease risk factors: a cross-sectional study in Iranian women. *J Cardiovasc Thorac Res* **11**, 216–223.
44. Romaguera D, Ångquist L, Du H, *et al.* (2010) Dietary determinants of changes in waist circumference adjusted for body mass index—a proxy measure of visceral adiposity. *PLoS One* **5**, e11588.
45. Liese AD, Schulz M, Fang F, *et al.* (2005) Dietary glycemic index and glycemic load, carbohydrate and fiber intake, and measures of insulin sensitivity, secretion, and adiposity in the Insulin Resistance Atherosclerosis Study. *Diabetes Care* **28**, 2832–2838.





46. Mazidi M, Gao HK & Kengne AP (2018) Lipid accumulation product and visceral adiposity index are associated with dietary patterns in adult Americans. *Medicine* **97**, e0322.
47. Hare-Bruun H, Flint A & Heitmann BL (2006) Glycemic index and glycemic load in relation to changes in body weight, body fat distribution, and body composition in adult Danes. *Am J Clin Nutr* **84**, 871–879.
48. Melanson KJ, Summers A, Nguyen V, *et al.* (2012) Body composition, dietary composition, and components of metabolic syndrome in overweight and obese adults after a 12-week trial on dietary treatments focused on portion control, energy density, or glycemic index. *Nutr J* **11**, 57.
49. Rajabi S, Mazloom Z, Zamani A, *et al.* (2015) Effect of low glycemic index diet versus metformin on metabolic syndrome. *Int J Endocrinol Metab* **13**, e23091.
50. Brand-Miller J, Hayne S, Petocz P, *et al.* (2003) Low-glycemic index diets in the management of diabetes: a meta-analysis of randomized controlled trials. *Diabetes Care* **26**, 2261–2267.
51. Kearney J (2010) Food consumption trends and drivers. *Philos Trans R Soc London, Ser B* **365**, 2793–2807.
52. Ishii Y, Shimizu F, Ogawa M, *et al.* (2016) Gender differences in foods uptakes, glycemic index, BMI, and various plasma parameters between young men and women in Japan. *Integr Food Nutr Metab* **3**, 427–430.
53. Fan J, Song Y, Wang Y, *et al.* (2012) Dietary glycemic index, glycemic load, and risk of coronary heart disease, stroke, and stroke mortality: a systematic review with meta-analysis. *PLoS One* **7**, e52182.
54. Takao T, Ogawa M, Ishii Y, *et al.* (2016) Different glycemic responses to sucrose and glucose in old and young male adults. *J Nutr Food Sci* **6**, 2.
55. Tarnopolsky MA (2000) Gender differences in metabolism; nutrition and supplements. *J Sci Med Sport* **3**, 287–298.
56. Wu BN & O'Sullivan AJ (2011) Sex differences in energy metabolism need to be considered with lifestyle modifications in humans. *J Nutr Metab* **2011**, 391809.
57. Tarnopolsky MA, Zawada C, Richmond LB, *et al.* (2001) Gender differences in carbohydrate loading are related to energy intake. *J Appl Physiol* **91**, 225–230.
58. Glinborg D, Petersen MH, Ravn P, *et al.* (2016) Comparison of regional fat mass measurement by whole body DXA scans and anthropometric measures to predict insulin resistance in women with polycystic ovary syndrome and controls. *Acta Obstet Gynecol Scand* **95**, 1235–1243.
59. Xia C, Li R, Zhang S, *et al.* (2012) Lipid accumulation product is a powerful index for recognizing insulin resistance in non-diabetic individuals. *Eur J Clin Nutr* **66**, 1035–1038.
60. Er L-K, Wu S, Chou H-H, *et al.* (2016) Triglyceride glucose-body mass index is a simple and clinically useful surrogate marker for insulin resistance in nondiabetic individuals. *PLoS One* **11**, e0149731.
61. Mazidi M, Gao H-K & Kengne AP (2018) Lipid accumulation product and visceral adiposity index are associated with dietary patterns in adult Americans. *Medicine* **97**, e0322.
62. Pires LAV, Tofano RJ, Barbalho SM, *et al.* (2021) Lipid accumulation product: reliable marker for cardiovascular risk detection? *Open J Epidemiol* **11**, 267–277.
63. Wang H, Chen Y, Sun G, *et al.* (2018) Validity of cardiometabolic index, lipid accumulation product, and body adiposity index in predicting the risk of hypertension in Chinese population. *Postgraduate Med* **130**, 325–333.
64. Hosseinpahan F, Barzin M, Mirbolouk M, *et al.* (2016) Lipid accumulation product and incident cardiovascular events in a normal weight population: Tehran Lipid and Glucose Study. *Eur J Prev Cardiol* **23**, 187–193.
65. Gao B, Liu Y, Ding C, *et al.* (2020) Comparison of visceral fat area measured by CT and bioelectrical impedance analysis in Chinese patients with gastric cancer: a cross-sectional study. *BMJ Open* **10**, e036335.
66. Qin Q, Yang Y, Chen J, *et al.* (2021) Bioelectrical impedance analysis versus quantitative computer tomography and anthropometry for the assessment of body composition parameters in China. *Sci Rep* **11**, 1–10.
67. Andreoli A, Melchiorri G, De Lorenzo A, *et al.* (2002) Bioelectrical impedance measures in different position and *v.* dual-energy X-ray absorptiometry (DXA). *J Sports Med Phys Fitness* **42**, 186.