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The Association Between Low Carbohydrate Diet and Resting Metabolic Rate in Overweight and Obese Women: A Cross-Sectional Study

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ABSTRACT

Resting metabolic rate (RMR) accounts for most daily energy expenditure. The low carbohydrate diet (LCD) attenuates decreases in RMR. This study aims to investigate the relationship between an LCD and RMR status among overweight and obese women. We enrolled 291 overweight and obese women in this cross-sectional study. Body mass index (BMI), fat mass, fat-free mass, visceral fat, and insulin level were assessed. RMR was measured using indirect calorimetry. LCD score (LCDS) was measured using a validated semi-quantitative food frequency questionnaire. Analysis of variance, independent sample t-test, and Multinomial logistic regression tests were used. Results showed no relationship between LCDS and deviation of normal RMR (DNR) even after adjust for confounders (increased [Inc.] RMR: odds ratio [OR], 0.97; 95% confidence interval [CI], 0.92–1.01; p = 0.20; decreased [Dec.] RMR: OR, 0.97; 95% CI, 0.94–1.00; p = 0.14). Some components of LCDS had no significant association with DNR, such as carbohydrate and Dec. RMR in adjusted model (OR, 1.62; 95% CI, 0.98–1.37; p = 0.08) and monounsaturated fatty acids and Dec. RMR in adjusted model (OR, 0.48; 95% CI, 0.21–1.10, p = 0.08). However, refined grains had a significant association with Inc. RMR in crude model (OR, 0.87; 95% CI, 0.77–0.99, p = 0.04). There is no association between LCDS and RMR status.

Keywords: Resting metabolic rate; Obesity; Overweight; Low carbohydrate diet

INTRODUCTION

Obesity, which is a serious, current health problem, affects 400 million adults worldwide [1]. Obesity is also a major public health problem in Iran, where 21.7% percent of the adult population are obese [2]. In the Iranian population, women are more obese, so obesity is reported in 57% of women and 42.8% of men [3]. Obesity is characterized as a chronic multifactorial disorder with a genetic basis caused by surplus fat tissue accumulation. It leads to many severe comorbidities, such as insulin resistance, hypertension, and diabetes mellitus [1,4-6]. The traditional treatment for obesity includes a combination of low-calorie diet therapy with enhanced physical activity and nutritional education [1]. Apart from low-fat diets, low-carbohydrate diets (LCDs) are also popular [7].

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Conflict of Interest
The authors declare that they have no competing interests.

Author Contributions
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LCD decreases the consumption of carbohydrates to 20 to 60 g/day (typically less than 45% of the daily caloric intake) while enhancing protein and fat intake [8,9]. Low-carbohydrate, high-fat, and high-protein diets (referred to as LCD) effectively improve weight loss and provide notable improvements in lipid profiles and glycemic control [10,11]. Some studies show that LCD results in rapid weight loss because of increased energy expenditure via ketogenesis or simply by appetite repression because of the high protein content. Protein is more satiating than carbohydrates and fats, and it seems to influence thermogenesis, thereby influencing satiety [1].

Several studies have recommended that LCD (< 45% energy from carbohydrates) attenuate decreases in resting metabolic rate (RMR), with proposed mechanisms including changed substrate availability and endocrine-mediated influences on anabolic and catabolic pathways. Furthermore, some studies have recommended that LCD may support the preservation of fat-free mass (FFM) and preferential loss of fat mass (FM), which would also attenuate decreases in RMR [12,13].

This is the first study to investigate the relationship between RMR and LCD score (LCDS) in an adult population to the best of the researchers' knowledge. Accordingly, this study was carried out to . examine LCDS with deviation of normal RMR (DNR) deviation among a group of adult Iranian women.

MATERIALS AND METHODS

Participants

This cross-sectional research included 291 adult women aged between 18 and 56 referred to health centers in Tehran in 2018. Blood samples and anthropometric measurements were taken in the Nutrition and Biochemistry Laboratory of the School of Nutritional and Dietetics at Tehran University of Medical Sciences. Participants were in good general health, with a body mass index (BMI) in the range of 25–49 kg/m². The exclusion criteria for the study were as follows: regular use of medicine, history of hypertension, cardiovascular disease, diabetes mellitus, and impaired renal and liver function, alcohol consumption, smoking, pregnancy, lactation period, and menopause. Furthermore, participants were excluded from a chronic disease affecting their diet, those who had been following an arbitrary special dietary regimen, and those with any significant body weight fluctuations over the past year.

This study was conducted according to the guidelines laid down in the Declaration of Helsinki. All procedures involving human subjects were approved by the Ethics Commission of Tehran University of Medical Sciences (IR.TUMS.VCR.REC.1396.2615), and all participants signed written informed consent.

Energy expenditure measurements

RMR was measured by indirect calorimetry (spirometer METALYZERR 3B-R3; Cortex Biophysik GmbH, Leipzig, Germany). According to the manufacturer's instructions, gas ventilation and exchange is calibrated before each test. The RMR is evaluated by measuring the amount of O_2 consumed and CO_2 produced. The RMR was assessed in the morning, after a comfortable night's sleep, and following a 10–12 hours fasting. Participants were asked to avoid caffeine or alcohol consumption and severe exercise for a day before RMR measurements. After reclining in a steady-state and a supine position in a quiet room, the

RMR was measured for 30 minutes. The respiratory exchange ratio and oxygen uptake (VO2) were analyzed within the middle 20 minutes of the resting period. Predictive RMR was determined using the Harris-Benedict equation, which considers the weight, height, and age of participants [14].

Body composition measurement

According to the manufacturer's instructions, body composition, including weight, BMI, FM, and FFM were acquired using a multi-frequency bioelectrical impedance analyzer Inbody 770 scanner (Inbody Co., Seoul, Korea). This electrical impedance analyzer calculates the 5 resistance of body tissues to the flow of an electrical signal sent through both hands and feet. The amount and proportion of bodily FFM and FM can be measured as the current flows more efficiently through certain parts of the body.

Biochemical parameters and hormonal assay

Venous blood samples were collected in the morning (8–10 A.M.) after 10-12 hours of fasting by a qualified phlebotomist. Within 30 to 45 minutes after each sample was collected, the blood was centrifuged for 15 minutes. Following separation, the serum was removed and frozen at −80℃ for later analysis. Fasting blood sugar (FBS) levels were evaluated by a colorimetric method based on the GOD-PAP method, triglyceride (TG) was assessed by GPO-PAP, and low-density lipoprotein cholesterol (LDL-C) was evaluated by the direct method [15].

An immunoinhibition assay was used to measure high-density lipoprotein cholesterol (HDL-C) and total cholesterol levels. Pars Azmoon kit was used for all assessments (Pars Azmoon Inc., Tehran, Iran) except insulin. Serum insulin concentrations were analyzed using the enzyme-linked immunosorbent assay (ELISA) method (Human insulin ELISA kit, Monobind Inc., Lake Forest, CA, USA).

DNR and calculation method

After examining the values of the body composition analysis, RMR components, and biochemical characters and comparing them with RMR status, the participants were categorized into 3 groups: increased RMR (Inc. RMR), normal RMR, and decreased RMR (Dec. RMR), based on the score of the deviation from normal RMR. Deviation of normal RMR was measured by indirect calorimetry (METALYZERR 3B-R3). The cutoff points for the groups were as follows: Inc. RMR (> 5% standard deviation [SD] of normal RMR), normal RMR (−5% SD < −5% SD).

Homeostasis model assessment (HOMA) and insulin sensitivity quantitative insulin sensitivity check index (ISQUICKI) calculations

Insulin resistance was estimated by HOMA. The HOMA was calculated according to the following equation [16]:

$$HOMA = [\text{Fasting Plasma Glucose (mmol/L)} \times \text{Fasting Plasma Insulin (mIU/L)}]/22.5.$$

Insulin sensitivity quantitative insulin sensitivity check index (ISQUICKI) was assessed by [17]:

$$ISQUICKI = 1/[\log (\text{Fasting Insulin}) + \log (\text{Fasting Glucose})].$$

Calculation of LCDS

A validated and reliable 168-item food frequency questionnaire (FFQ) was used to assess the dietary intake of participants. This semi-quantitative questionnaire consisted of standard portion sizes for each food item and was designed according to the Willett method. A trained nutritionist asked participants to determine the frequency of consumption of each food item during the previous year, based on serving sizes [18]. Food intakes reported in household measures were then converted to grams of food per day using the nutritionist IV software (version 7.0; N-Squared Computing, Salem, OR, USA) [19]. The participants included in the current study were divided into 11 strata based on their scores in the following 7 categories: carbohydrates refined grains, vegetable protein intake, monounsaturated fatty acids (MUFA), n3/n6 polyunsaturated fatty acids (PUFA) (expressed as a percentage of energy intake), as well as fiber (g/1,000 kcal), and glycemic load (GL). Dietary GL (g/d) was estimated as: (Total Glycemic Index × Total Available Carbohydrate)/100 [20]. Women in the lowest stratum of refined grains, carbohydrates, and GL were given a score of 10, and those in the highest stratum were given a score of 0. For n3/n6 PUFA, MUFA, fiber, and vegetable protein 7 intakes, the order of the strata was reversed. The points for the seven items were added together to create the overall score, named the "LCDS", which ranged from 0 to 70. Therefore, higher LCDS demonstrated closer adherence to LCD [21].

Assessment of other variables

The short, interviewer-administered International Physical Activity Questionnaire-Short Form was used in this study. The level of physical activity of participants was calculated as Met.h/d [22]. For height measurements, subjects were in a standing position without shoes, in contact with the wall with their head, shoulders, heels, and hips, and their height was recorded to the nearest 0.1 cm using a tape measure.

Statistical analysis

All statistical analysis was performed using the IBM SPSS software version 22.0 (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test checked the normal distribution of data. An independent sample t-test was used for assessed differences between groups with low and high adherence to LCD. One analysis of variance (ANOVA) and re-analyses assessed the differences between RMR status groups by the general linear model were performed to adjust for confounders' effects. Collinear variables did not enter into the model. Post-hoc multiple comparison analysis resulting from the LCD procedure demonstrated the significant differences between groups. Multinomial logistic regression was used to assess the association of DNR and LCDS and its components. Results were presented as odds ratios (ORs) and 95% confidence intervals (CIs) compared with the DNR groups.

RESULTS

Study population characteristics

Three hundred and four healthy obese women enrolled in this cross-sectional study. The mean (± SD) age, height, weight, and BMI of the study participants were 36.49 ± 8.38 years, 161.38 ± 5.90 cm, 80.89 ± 12.45 kg, and 31.04 ± 4.31 kg/m², respectively (**Table 1**). The mean body composition, RMR components, biochemical and anthropometric characteristics of subjects are shown in **Table 1**.

Table 1. Study population characteristics (n = 291)

Parameters	Minimum	Maximum	Mean ± SD
Age (yr)	17	56	36.49 ± 8.38
Height (cm)	142	179	161.38 ± 5.90
Weight (kg)	59.50	136.60	80.89 ± 12.45
BMI (kg/m²)	24.20	49.60	31.04 ± 4.31
RMR parameters			
RMR measure (kcal/day)	952.00	2,480.00	1,575.00 ± 259.71
RMR normal (kcal/day)	1,425.00	2,548.00	1,720.40 ± 152.36
Deviation normal (%)	−44.00	40.00	−8.47 ± 12.59
RMR/kg body weight (kcal/day/kg)	9.30	32.50	19.59 ± 3.09
Body composition analysis			
Body fat mass (kg)	19.40	74.20	34.04 ± 8.69
Fat free mass (kg)	35.30	67.70	46.80 ± 5.64
Skeletal muscle mass (kg)	18.90	37.90	25.69 ± 3.33
Soft lean mass (kg)	26.10	63.80	44.02 ± 5.37
Blood parameters			
FBS (mg/dL)	67.00	137.00	87.49 ± 9.64
Insulin (mIU/mL)	6.67	65.89	15.68 ± 6.06
Total cholesterol (mg/dL)	104.00	344.00	185.30 ± 35.77
TG (mg/dL)	37.00	512.00	122.11 ± 69.29
HDL-C (mg/dL)	18.00	87.00	46.58 ± 10.86
LDL-C (mg/dL)	34.00	156.00	95.30 ± 24.12
HOMA	1.29	16.59	3.43 ± 1.53
ISQUICKI	0.39	0.68	0.54 ± 0.04

SD, standard deviation; BMI, body mass index; RMR, resting metabolic rate; FBS, fasting blood sugar; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; HOMA, homeostasis model assessment; ISQUICKI, insulin sensitivity quantitative insulin sensitivity check index.

Study participant characteristics between high and low adherence of LCD

All participants were categorized based on the LCDS and divided into 2 groups (**Table 2**). As shown in **Table 2**, participants with high adherence to an LCD had significantly higher LDL-C (p = 0.03). However, subjects with high adherence to an LCD had higher total cholesterol (p = 0.22) and HDL-C (p = 0.10) than the low adherence LCD group, but these findings were not statistically significant. There were no significant differences in terms of height, weight, RMR, RMR/kg body weight, normal deviation, Respiratory Quotient, FFM, FBG, and TG between the two groups (p > 0.05) (**Table 2**).

Association of studied variations and RMR status

The differences between the normal, Dec. RMR, and Inc. RMR groups were analyzed through one-way ANOVA tests (**Table 3**). Subjects in the Inc. RMR group had significantly higher height (p < 0.006), RMR measurement (p < 0.0001), RMR/kg body weight (p < 0.0001), skeletal muscle mass (p < 0.0001), and soft lean mass (p < 0.0001), compared to the Dec. RMR group. After adjusting for age, energy intake, physical activity, and FFM, all significant results remained robust. Furthermore, bodily FM (p = 0.01) and ISQUICKI (p = 0.02) became significant (**Table 3**).

The association of LCDS and RMR across DNR

Multivariate-adjusted models with 95% CIs for the association between LCDS and RMR across DNR are presented in **Table 4**. In the crude model, no significant association was found between LCDS with Inc. RMR and Dec. RMR (Inc. RMR: OR, 0.97; 95% CI, 0.93–1.01; p = 0.20 and Dec. RMR: OR, 0.98; 95% CI, 0.96–1.01; p = 0.31). Furthermore, after adjustment for confounders, the associations remained unchanged. Therefore, no significant association was found between LCDS with Inc. RMR and Dec. RMR (Inc. RMR: OR, 0.97; 95% CI, 0.92–1.01; p = 0.20 and Dec. RMR: OR, 0.97; 95% CI, 0.94–1.00; p = 0.14).

Table 2. Study participant characteristics between high and low adherence to LCD (n = 291)

Parameters	Low-adherence (n = 132)	High-adherence (n = 159)	p[†]	p[‡]
LCD[*]				
Age (yr)	36.52 ± 8.49	36.47 ± 8.56	0.96	0.62
Height (cm)	161.06 ± 5.99	161.46 ± 5.89	0.56	0.75
Weight (kg)	80.88 ± 12.68	80.41 ± 11.73	0.74	0.49
BMI (kg/m²)	31.21 ± 4.43	30.86 ± 4.21	0.48	0.50
RMR parameters				
RMR measure (kcal/day)	1,580.80 ± 270.32	1,570.93 ± 248.76	0.74	0.92
RMR normal (kcal/day)	1,720.93 ± 270.32	1,570.93 ± 248.76	0.73	0.76
Deviation normal (%)	−8.01 ± 13.46	−8.54 ± 11.96	0.72	0.84
RMR/kg body weight (kcal/day/kg)	19.64 ± 3.21	19.67 ± 3.04	0.92	0.49
Body composition analysis				
Body fat mass (kg)	34.07 ± 9.01	33.84 ± 8.29	0.82	0.81
Fat free mass (kg)	46.97 ± 5.68	46.56 ± 5.49	0.53	0.72
Skeletal muscle mass (kg)	25.78 ± 3.33	25.57 ± 3.28	0.86	0.78
Soft lean mass (kg)	44.29 ± 5.36	43.69 ± 5.27	0.34	0.46
Blood parameters				
FBS (mg/dL)	87.01 ± 9.76	87.92 ± 9.52	0.46	0.34
Insulin (mIU/mL)	16.08 ± 6.97	15.31 ± 5.35	0.31	0.22
Total cholesterol (mg/dL)	181.96 ± 35.00	188.23 ± 37.19	0.15	0.22
TG (mg/dL)	199.87 ± 67.75	124.77 ± 72.90	0.58	0.66
HDL-C (mg/dL)	45.76 ± 10.40	47.73 ± 11.19	0.15	0.10
LDL-C (mg/dL)	91.82 ± 21.13	97.89 ± 26.38	**0.04**	**0.03**
HOMA	3.51 ± 1.80	3.34 ± 1.29	0.39	0.25
ISQUICKI	0.541 ± 0.049	0.545 ± 0.048	0.55	0.41

Data are indicated as mean ± SD otherwise indicated. The bold-styled values mean they are statistically significant.
LCD, low-carbohydrate diet; BMI, body mass index; RMR, resting metabolic rate; FBS, fasting blood sugar; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; HOMA, homeostasis model assessment; ISQUICKI, insulin sensitivity quantitative insulin sensitivity check index; SD, standard deviation.
[*]This diet is defined based on LCDS. The adherence to a low-carb diet is based on the median of the population (cut point = 36).
[†]The p values are from ANOVA.
[‡]The p values are from the general linear model. After adjustment for age, FFM, energy intake, and physical activity (METs/d). Collinear variables did not enter into the model.

The association of LCDS components and RMR across DNR

The participants' dietary components of LCDS, based on DNR groups, are shown in **Table 5**. Differences in some LCDS components between DNR such as GL, vegetable protein intake, n3/n6 PUFA, and fiber (g/1,000 kcal) were non-significant, even after adjustment for the potential confounders. However, the adjusted model of carbohydrate (% energy) and MUFA were marginal for Dec. RMR, but it was not statistically significant (OR, 0.15; 95% CI, 0.98–1.37; p = 0.08) and RMR (OR, 0.48; 95% CI, 0.21–1.10; p = 0.08), respectively. For refined grains, participants with a higher intake were at a 13% lower risk for Inc. RMR (OR, 0.87; 95% CI, 0.77–0.99; p = 0.04). However, after adjusting for confounders, the significance disappeared (OR, 0.93; 95% CI, 0.81–1.08; p = 0.39).

DISCUSSION

An LCD might be able to reduce the development of obesity. Previous studies had indicated links between LCD and obesity [23,24]. Therefore, this paper sought to test the effect of LCDs on the possible link between obesity and deviation of normal RMR in overweight and obese women.

The findings of the current study indicate that high adherence to an LCD is associated with higher LDL-C. This result may be attributed to the replacement of carbohydrates with fats in

Table 3. Association of studied variation and RMR status (n = 291)

Parameters	Dec. RMR (n = 167)	Normal RMR (n = 87)	Inc. RMR (n = 37)	p*	p†
RMR status					
Age (yr)	36.30 ± 7.92	30.36 ± 9.01	37.05 ± 9.13	0.88	0.94
Height (cm)	160.50 ± 5.54	162.44 ± 6.24	163.25 ± 6.17	**0.006**	**0.002**
Weight (kg)	80.35 ± 12.10	80.83 ± 12.09	83.80 ± 14.26	0.30	0.39
BMI (kg/m²)	31.10 ± 4.32	30.70 ± 3.84	31.42 ± 5.14	0.64	0.79
RMR parameters					
RMR measure (kcal/day)	1,425.00 ± 179.98	1,713.90 ± 167.01	1,945.10 ± 163.24	**< 0.0001**	**< 0.0001**
RMR normal (kcal/day)	1,714.40 ± 151.64	1,726.20 ± 149.64	1,734.60 ± 164.42	0.70	0.66
RMR/kg body weight (kcal/day/kg)	17.83 ± 2.17	21.19 ± 1.65	24.03 ± 2.90	**< 0.0001**	**< 0.0001**
Body composition analysis					
Body fat mass (kg)	34.33 ± 8.54	33.66 ± 8.10	33.85 ± 10.49	0.81	**0.01**
Fat free mass (kg)	45.75 ± 5.20	47.48 ± 6.06	49.68 ± 5.03	**< 0.0001**	**< 0.0001**
Skeletal muscle mass (kg)	25.09 ± 3.10	26.08 ± 3.58	27.35 ± 2.89	**< 0.0001**	**< 0.0001**
Soft lean mass (kg)	42.97 ± 4.99	44.75 ± 5.69	46.83 ± 4.76	**< 0.0001**	**< 0.0001**
Blood parameters					
FBS (mg/dL)	86.70 ± 8.89	89.45 ± 11.07	88.06 ± 10.51	0.15	0.21
Insulin (mIU/mL)	15.18 ± 5.37	16.17 ± 7.78	16.75 ± 5.38	0.28	0.15
Total cholesterol (mg/dL)	183.22 ± 33.29	187.80 ± 38.45	188.10 ± 41.80	0.60	0.34
TG (mg/dL)	120.15 ± 66.36	128.56 ± 81.90	116.93 ± 52.72	0.65	0.65
HDL-C (mg/dL)	46.44 ± 11.01	46.48 ± 11.50	47.16 ± 9.70	0.94	0.84
LDL-C (mg/dL)	95.76 ± 23.81	96.00 ± 24.19	89.43 ± 24.24	0.39	0.37
HOMA	3.29 ± 1.34	3.67 ± 2.08	3.62 ± 1.13	0.19	0.54
ISQUICKI	0.548 ± 0.049	0.537 ± 0.047	0.532 ± 0.043	0.10	**0.02**

Data are indicated as mean ± SD otherwise indicated. Post-hoc multiple comparison analysis from LCD procedure used to demonstrate significant differences between groups. The bold-styled values mean they are statistically significant.

RMR, resting metabolic rate; Dec. RMR, decreased RMR; Inc. RMR, increased RMR; FBS, fasting blood sugar; TG, triglyceride; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; HOMA, homeostasis model assessment; ISQUICKI, insulin sensitivity quantitative insulin sensitivity check index; SD, standard deviation.

*The p values are from ANOVA.

†The p values are from the general linear model. After adjustment for age, FFM, energy intake, and physical activity (METs/d). Collinear variables did not enter into the model.

Table 4. The association of LCDS and RMR across DNR (n = 291)

LCDS	DNR	β	OR (95% CI)	p*
Crude model	Dec. RMR	−0.01	0.98 (0.96–1.01)	0.31
	Inc. RMR	−0.02	0.97 (0.93–1.01)	0.20
Model 1	Dec. RMR	−0.01	0.98 (0.96–1.01)	0.30
	Inc. RMR	−0.02	0.97 (0.93–1.01)	0.19
Model 2	Dec. RMR	−0.01	0.98 (0.95–1.01)	0.24
	Inc. RMR	−0.02	0.97 (0.94–1.01)	0.24
Model 3	Dec. RMR	−0.02	0.97 (0.94–1.00)	0.13
	Inc. RMR	−0.02	0.97 (0.93–1.01)	0.23
Model 4	Dec. RMR	−0.02	0.97 (0.94–1.00)	0.14
	Inc. RMR	−0.02	0.97 (0.92–1.01)	0.20

Normal RMR status considered as the reference category. Model 1: Adjusted for age. Model 2: Further adjusted for FFM. Model 3: Further adjusted for physical activity (METs/d). Model 4: Further adjusted for energy intake.

LCDS, low carbohydrate diet score; RMR, resting metabolic rate; DNR, deviation of normal RMR; OR, odds ration; CI, confidence interval; Dec. RMR, decreased RMR; Inc. RMR, increased RMR.

*The p values are from multinomial logistic regression.

an LCD [25]. This finding was consistent with previous observations that fat intake results in an increase in LDL-C [26]. Also, this result showed the association between an LCD and increases in total cholesterol and HDL-C. Based on previous studies, a lower dietary intake of carbohydrates has been associated with higher concentrations of HDL-C [25,27].

The other finding of this research is that increased RMR associated with lower FM and increased RMR is strongly associated with higher height, RMR/kg body weight, skeletal muscle mass, soft lean mass, and ISQUICKI. Results of Hirsch et al. [28] study on 49

Table 5. The association of LCDS components and RMR across DNR (n = 304)

Category	LCDS	DNR	β	OR (95% CI)	p*
GL	Crude model	Dec. RMR	0.0001	1.00 (0.99–1.00)	0.85
		Inc. RMR	0.0001	1.00 (1.00–1.00)	0.57
	Adjusted model†	Dec. RMR	0.0001	1.00 (0.99–1.00)	0.65
		Inc. RMR	0.0001	1.00 (1.00–1.00)	0.20
Carbohydrates (% energy)	Crude model	Dec. RMR	0.11	1.11 (0.95–1.30)	0.15
		Inc. RMR	0.18	1.20 (0.95–1.51)	0.11
	Adjusted model	Dec. RMR	0.15	1.62 (0.98–1.37)	**0.08**
		Inc. RMR	0.19	1.22 (0.94–1.58)	0.13
MUFA (% energy)	Crude model	Dec. RMR	−0.53	0.58 (0.27–1.24)	0.16
		Inc. RMR	−0.42	0.65 (0.21–0.03)	0.46
	Adjusted model	Dec. RMR	−0.71	0.48 (0.21–1.10)	**0.08**
		Inc. RMR	−0.60	0.54 (0.15–1.88)	0.33
Vegetable protein intake (% energy)	Crude model	Dec. RMR	−0.11	0.89 (0.72–1.10)	0.28
		Inc. RMR	−0.17	0.83 (0.55–1.25)	0.39
	Adjusted model	Dec. RMR	−0.21	0.88 (0.71–1.09)	0.26
		Inc. RMR	−0.23	0.78 (0.48–1.29)	0.34
Refined grains (% energy)	Crude model	Dec. RMR	0.001	1.00 (0.92–1.08)	0.99
		Inc. RMR	−0.13	0.87 (0.77–0.99)	**0.04**
	Adjusted model	Dec. RMR	−0.008	0.99 (0.90–1.09)	0.87
		Inc. RMR	−0.06	0.93 (0.81–1.08)	0.39
n3/n6 PUFA ratio	Crude model	Dec. RMR	0.33	1.40 (0.37–5.24)	0.61
		Inc. RMR	−0.34	0.70 (0.10–4.78)	0.72
	Adjusted model	Dec. RMR	0.61	1.84 (0.42–7.95)	0.41
		Inc. RMR	−0.09	0.90 (0.11–7.41)	0.92
Fiber (g/1,000 kcal)	Crude model	Dec. RMR	0.006	1.00 (0.95–1.05)	0.81
		Inc. RMR	0.01	1.01 (0.94–1.08)	0.75
	Adjusted model	Dec. RMR	0.0001	1.00 (0.94–1.05)	0.99
		Inc. RMR	0.06	1.06 (0.97–1.16)	0.14

The normal RMR status considers as the reference category. The bold-styled values mean they are statistically significant.
LCDS, low carbohydrate diet score; RMR, resting metabolic rate; DNR, deviation of normal RMR; OR, odds ration; CI, confidence interval; Dec. RMR, decreased RMR; Inc. RMR, increased RMR; GL, glycemic load; MUFA, monounsaturated fatty acids; PUFA, polyunsaturated fatty acids.
*The p values are from multinomial logistic regression.
†Adjusted model: adjusted for age, FFM, physical activity (METs/d), energy intake.

overweight and obese adults shows that greater amounts of lean mass were associated with a higher RMR. FM has the least metabolic activity in a normal weight individual, accounting for less than 5% of total RMR [29]. However, in obese and overweight individuals, FM has a greater metabolic impact [30]. A previous finding showed that FM was associated with increased metabolic rate in women with up to 40% body fat.

In contrast, FM was associated with a significantly decreased metabolic rate in women with greater than 40% body fat [31]. However, the FM in obese and overweight individuals has a greater metabolic impact [32], both directly by altering substrate oxidation and metabolic rate. Indirectly, by chronic changes in hormonal concentrations [33], Muscle mass specifically is the main location for substrate oxidation and is correlated with enhanced health status, including improved insulin and glucose adjustment. However, the correlation between body composition (specifically metabolic function) and lean mass is still unclear [34].

This study found no significant association between LCDS and RMR status. This result is in line with previous studies, which showed that LCD failed to increase energy expenditure compared to low-fat diets in the obese population after 6 to 12 months of intervention [24,34]. Previous studies have suggested that dietary carbohydrates are among the factors thought to influence metabolic adaptation [35] and may decrease reductions in RMR through mechanisms associated with substrate availability and autonomic and hormonal activity [36].

Gillingham et al. [36] reported that there was no significant correlation between the consumption of MUFA and modulate resting or postprandial energy expenditure among 21 overweight and obese young adults in Boston. However, findings from other studies have reported that dietary increases in MUFA [37,38], and the ratio of MUFA to saturated fatty acid or polyunsaturated fatty acid, increased the thermic effect of food and/or fat oxidation [39,40]. Astrup and colleagues [41] have reported a moderate correlation between insulin and RMR in females [42]. Moreover, previous studies revealed significant differences in RMR in individuals with insulin resistance [43,44]. Higher RMR in individuals with type 2 diabetes, contrasted with non-diabetics, which has been suggested, is due to insulin resistance [41]. Refined grains had diminished insulin sensitivity, and one of the first responses to alternations in insulin sensitivity is the change in hepatic insulin clearance rates [45]. Several mechanisms have been suggested to explain the increased RMR in individuals with insulin resistance, including futile substrate cycling, plasma glucagon, increases in protein turnover, and sympathetic nervous system activity [46]. The other proposed mechanism was an increase in gluconeogenesis. It has been put forward that enhanced free fatty acid concentrations in individuals with insulin resistance contribute to 12 increased hepatic glucose output and excessive rates of gluconeogenesis, depending upon the oxidation of the fatty acids and consequently the increased energy expenditure rate in these samples [46,47] In support of this pathway, following improvements in glycemic control, a decrease in resting energy expenditure was reported.

CONCLUSION

In conclusion, this study's nuanced findings highlight that an LCD has no significant correlation with DNR.

This study was the first to assess the possible relationship between LCD and DNR in obese women to the researchers' knowledge. At the cross-sectional research, we could not determine causality more randomized clinical trials and prospective observational studies, as well as further cohort research designs, are expected to affirm the possible link between this LCD and DNR in obese people. The major limitation of our study was the relatively small number of participants and the same-sex sample. Because the study population was confined to women, the generalizability of men was limited; therefore, large samples for both sexes were needed to solve this problem.

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REFERENCES

1. Triffoni-Melo AT, Dick-de-Paula I, Portari GV, Jordao AA, Garcia Chiarello P, Diez-Garcia RW. Short-term carbohydrate-restricted diet for weight loss in severely obese women. Obes Surg 2011;21:1194-202.
   PUBMED | **CROSSREF**

2. Rahmani A, Sayehmiri K, Asadollahi K, Sarokhani D, Islami F, Sarokhani M. Investigation of the prevalence of obesity in iran: a systematic review and meta-analysis study. Acta Med Iran 2015;53:596-607.
   PUBMED

3. Barak F, Falahi E, Keshteli AH, Yazdannik A, Esmaillzadeh A. Adherence to the Dietary Approaches to Stop Hypertension (DASH) diet in relation to obesity among Iranian female nurses. Public Health Nutr 2015;18:705-12.
   PUBMED | CROSSREF

4. Bastien M, Poirier P, Lemieux I, Després JP. Overview of epidemiology and contribution of obesity to cardiovascular disease. Prog Cardiovasc Dis 2014;56:369-81.
   PUBMED | CROSSREF

5. Sajjadi SF, Mirzaei K, Khorrami-Nezhad L, Maghbooli Z, Keshavarz SA. Vitamin D status and resting metabolic rate may modify through expression of vitamin D receptor and peroxisome proliferator-activated receptor gamma coactivator-1 alpha gene in overweight and obese adults. Ann Nutr Metab 2018;72:43-9.
   PUBMED | CROSSREF

6. Rahimlou M, Mirzaei K, Keshavarz SA, Hossein-Nezhad A. Association of circulating adipokines with metabolic dyslipidemia in obese versus non-obese individuals. Diabetes Metab Syndr 2016;10:S60-5.
   PUBMED | CROSSREF

7. Essah PA, Levy JR, Sistrun SN, Kelly SM, Nestler JE. Effect of weight loss by a low-fat diet and a low-carbohydrate diet on peptide YY levels. Int J Obes 2010;34:1239-42.
   PUBMED | CROSSREF

8. Last AR, Wilson SA. Low-carbohydrate diets. Am Fam Physician 2006;73:1942-8.
   PUBMED

9. Hu T, Mills KT, Yao L, Demanelis K, Eloustaz M, Yancy WS JrKelly TN, He J, Bazzano LA. Effects of low-carbohydrate diets versus low-fat diets on metabolic risk factors: a meta-analysis of randomized controlled clinical trials. Am J Epidemiol 2012;176 Suppl 7:S44-54.
   PUBMED | CROSSREF

10. Tonstad S, Malik N, Haddad E. A high-fibre bean-rich diet versus a low-carbohydrate diet for obesity. J Hum Nutr Diet 2014;27 Suppl 2:109-16.
    PUBMED | CROSSREF

11. Grieb P, Kłapcińska B, Smol E, Pilis T, Pilis W, Sadowska-Krepa E, Sobczak A, Bartoszewicz Z, Nauman J, Stańczak K, Langfort J. Long-term consumption of a carbohydrate-restricted diet does not induce deleterious metabolic effects. Nutr Res 2008;28:825-33.
    PUBMED | CROSSREF

12. Bazzano LA, Hu T, Reynolds K, Yao L, Bunol C, Liu Y, Chen CS, Klag MJ, Whelton PK, He J. Effects of low-carbohydrate and low-fat diets: a randomized trial. Ann Intern Med 2014;161:309-18.
    PUBMED | CROSSREF

13. Krieger JW, Sitren HS, Daniels MJ, Langkamp-Henken B. Effects of variation in protein and carbohydrate intake on body mass and composition during energy restriction: a meta-regression 1. Am J Clin Nutr 2006;83:260-74.
    PUBMED | CROSSREF

14. Jesus F, Nunes CL, Matias CN, Francisco R, Carapeto B, Macias H, Müller D, Cardoso M, Valamatos MJ, Rosa G, Sardinha LB, Martins P, Minderico CS, Silva AM. Are predictive equations a valid method of assessing the resting metabolic rate of overweight or obese former athletes? Eur J Sport Sci 2020;20:1225-34.
    PUBMED | CROSSREF

15. Sajjadi SF, Mirzababaei A, Abdollahi A, Shiraseb F, Mirzaei K. The association between deficiency of nutrient intake and resting metabolic rate in overweight and obese women: a cross-sectional study. BMC Res Notes 2021;14:179.
    PUBMED | CROSSREF

16. Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412-9.
    PUBMED | CROSSREF

17. Katz A, Nambi SS, Mather K, Baron AD, Follmann DA, Sullivan G, Quon MJ. Quantitative insulin sensitivity check index: a simple, accurate method for assessing insulin sensitivity in humans. J Clin Endocrinol Metab 2000;85:2402-10.
    PUBMED | CROSSREF

18. Azadbakht L, Haghighatdoost F, Feizi A, Esmaillzadeh A. Breakfast eating pattern and its association with dietary quality indices and anthropometric measurements in young women in Isfahan. Nutrition 2013;29:420-5.
    PUBMED | CROSSREF

19. Ghaffarpour M, Houshiar-Rad A, Kianfar H. The manual for household measures, cooking yields factors and edible portion of foods, Vol. 7. Tehran: Nashre Olume Keshavarzy; 1999. p. 213.

20. Wolever TM, Yang M, Zeng XY, Atkinson F, Brand-Miller JC. 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 2006;83:1306-12.
    PUBMED | CROSSREF

21. Eslamian G, Mirmiran P, Asghari G, Hosseini-Esfahani F, Yuzbashian E, Azizi F. Low carbohydrate diet score does not predict metabolic syndrome in children and adolescents: Tehran Lipid and Glucose Study. Arch Iran Med 2014;17:417-22.
    PUBMED

22. Craig CL, Marshall AL, Sjöström M, Bauman AE, Booth ML, Ainsworth BE, Pratt M, Ekelund U, Yngve A, Sallis JF, Oja P. International physical activity questionnaire: 12-country reliability and validity. Med Sci Sports Exerc 2003;35:1381-95.
    PUBMED | CROSSREF

23. Kyle RG, Wills J, Mahoney C, Hoyle L, Kelly M, Atherton IM. Obesity prevalence among healthcare professionals in England: a cross-sectional study using the Health Survey for England. BMJ Open 2017;7:e018498.
    PUBMED | CROSSREF

24. Erlanson-Albertsson C, Mei J. The effect of low carbohydrate on energy metabolism. Int J Obes 2005;29 Suppl 2:S26-30.
    PUBMED | CROSSREF

25. Tay J, Luscombe-Marsh ND, Thompson CH, Noakes M, Buckley JD, Wittert GA, Yancy WS Jr, Brinkworth GD. Comparison of low- and high-carbohydrate diets for type 2 diabetes management: a randomized trial. Am J Clin Nutr 2015;102:780-90.
    PUBMED | CROSSREF

26. Dreon DM, Fernstrom HA, Campos H, Blanche P, Williams PT, Krauss RM. Change in dietary saturated fat intake is correlated with change in mass of large low-density-lipoprotein particles in men. Am J Clin Nutr 1998;67:828-36.
    PUBMED | CROSSREF

27. Shirani F, Esmaillzadeh A, Keshteli AH, Adibi P, Azadbakht L. Low-carbohydrate-diet score and metabolic syndrome: an epidemiologic study among Iranian women. Nutrition 2015;31:1124-30.
    PUBMED | CROSSREF

28. Hirsch KR, Smith-Ryan AE, Blue MN, Mock MG, Trexler ET. Influence of segmental body composition and adiposity hormones on resting metabolic rate and substrate utilization in overweight and obese adults. J Endocrinol Invest 2017;40:635-43.
    PUBMED | CROSSREF

29. Dulloo AG, Jacquet J, Solinas G, Montani JP, Schutz Y. Body composition phenotypes in pathways to obesity and the metabolic syndrome. Int J Obes 2010;34 Suppl 2:S4-17.
    PUBMED | CROSSREF

30. Hirsch KR, Smith-Ryan AE, Blue MN, Mock MG, Trexler ET. Influence of segmental body composition and adiposity hormones on resting metabolic rate and substrate utilization in overweight and obese adults. J Endocrinol Invest 2017;40:635-43.
    PUBMED | CROSSREF

31. Bosy-Westphal A, Müller MJ, Boschmann M, Klaus S, Kreymann G, Lührmann PM, Neuhäuser-Berthold M, Noack R, Pirke KM, Platte P, Selberg O, Steiniger J. Grade of adiposity affects the impact of fat mass on resting energy expenditure in women. Br J Nutr 2009;101:474-7.
    PUBMED | CROSSREF

32. Shook RP, Hand GA, Paluch AE, Wang X, Moran R, Hébert JR, Jakicic JM, Blair SN. High respiratory quotient is associated with increases in body weight and fat mass in young adults. Eur J Clin Nutr 2016;70:1197-202.
    PUBMED | CROSSREF

33. Hirsch KR, Smith-Ryan AE, Blue MN, Mock MG, Trexler ET. Influence of segmental body composition and adiposity hormones on resting metabolic rate and substrate utilization in overweight and obese adults. J Endocrinol Invest 2017;40:635-43.
    PUBMED | CROSSREF

34. Brehm BJ, Seeley RJ, Daniels SR, D'Alessio DA. A randomized trial comparing a very low carbohydrate diet and a calorie-restricted low fat diet on body weight and cardiovascular risk factors in healthy women. J Clin Endocrinol Metab 2003;88:1617-23.
    PUBMED | CROSSREF

35. Ebbeling CB, Swain JF, Feldman HA, Wong WW, Hachey DL, Garcia-Lago E, Ludwig DS. Effects of dietary composition on energy expenditure during weight-loss maintenance. JAMA 2012;307:2627-34.
    PUBMED | CROSSREF

36. Gillingham LG, Robinson KS, Jones PJ. Effect of high-oleic canola and flaxseed oils on energy expenditure and body composition in hypercholesterolemic subjects. Metabolism 2012;61:1598-605.
    PUBMED | CROSSREF

37. Jones PJ, Jew S, AbuMweis S. The effect of dietary oleic, linoleic, and linolenic acids on fat oxidation and energy expenditure in healthy men. Metabolism 2008;57:1198-203.
    PUBMED | CROSSREF

38. Kien CL, Bunn JY, Tompkins CL, Dumas JA, Crain KI, Ebenstein DB, Koves TR, Muoio DM. Substituting dietary monounsaturated fat for saturated fat is associated with increased daily physical activity and resting energy expenditure and with changes in mood. Am J Clin Nutr 2013;97:689-97.
    PUBMED | CROSSREF

39. Casas-Agustench P, López-Uriarte P, Bulló M, Ros E, Gómez-Flores A, Salas-Salvadó J. Acute effects of three high-fat meals with different fat saturations on energy expenditure, substrate oxidation and satiety. Clin Nutr 2009;28:39-45.
    PUBMED | CROSSREF

40. Moradi S, Mirzaei K, Abdurahman AA, Keshavarz SA. Adipokines may mediate the relationship between resting metabolic rates and bone mineral densities in obese women. Osteoporos Int 2017;28:1619-29.
    PUBMED | CROSSREF

41. Astrup A, Buemann B, Christensen NJ, Madsen J, Gluud C, Bennett P, Svenstrup B. The contribution of body composition, substrates, and hormones to the variability in energy expenditure and substrate utilization in premenopausal women. J Clin Endocrinol Metab 1992.74:279-86.
    PUBMED

42. Wright TG, Dawson B, Jalleh G, Guelfi KJ. Influence of hormonal profile on resting metabolic rate in normal, overweight and obese individuals. Ann Nutr Metab 2015;66:162-7.
    PUBMED | CROSSREF

43. Weyer C, Bogardus C, Pratley RE. Metabolic factors contributing to increased resting metabolic rate and decreased insulin-induced thermogenesis during the development of type 2 diabetes. Diabetes 1999;48:1607-14.
    PUBMED | CROSSREF

44. Moradi S, Mirzaei K, Abdurahman AA, Keshavarz SA, Hossein-Nezhad A. Mediatory effect of circulating vaspin on resting metabolic rate in obese individuals. Eur J Nutr 2016;55:1297-305.
    PUBMED | CROSSREF

45. Kim Y, Keogh JB, Clifton PM. Differential effects of red meat/refined grain diet and dairy/chicken/nuts/whole grain diet on glucose, insulin and triglyceride in a randomized crossover study. Nutrients 2016;8:E687.
    PUBMED | CROSSREF

46. Fontvieille AM, Lillioja S, Ferraro RT, Schulz LO, Rising R, Ravussin E. Twenty-four-hour energy expenditure in Pima Indians with type 2 (non-insulin-dependent) diabetes mellitus. Diabetologia 1992;35:753-9.
    PUBMED | CROSSREF

47. Hussain Z, Khan JA. Food intake regulation by leptin: mechanisms mediating gluconeogenesis and energy expenditure. Asian Pac J Trop Med 2017;10:940-4.
    PUBMED | CROSSREF