

## ORIGINAL ARTICLE

# Oral carnitine supplementation reduces body weight and insulin resistance in women with polycystic ovary syndrome: a randomized, double-blind, placebo-controlled trial

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## Summary

**Objective** Limited data are available for evaluating the effects of oral carnitine supplementation on weight loss and metabolic profiles of women with polycystic ovary syndrome (PCOS). This study was designed to determine the effects of oral carnitine supplementation on weight loss, and glycaemic and lipid profiles in women with PCOS.

**Design, Patients and Measurements** In a prospective, randomized, double-blind, placebo-controlled trial, 60 overweight patients diagnosed with PCOS were randomized to receive either 250 mg carnitine supplements ( $n = 30$ ) or placebo ( $n = 30$ ) for 12 weeks. Fasting blood samples were obtained at the beginning and the end of the study to quantify parameters of glucose homeostasis and lipid concentrations.

**Results** At the end of the 12 weeks, taking carnitine supplements resulted in a significant reduction in weight ( $-2.7 \pm 1.5$  vs  $+0.1 \pm 1.8$  kg,  $P < 0.001$ ), BMI ( $-1.1 \pm 0.6$  vs  $+0.1 \pm 0.7$  kg/m<sup>2</sup>,  $P < 0.001$ ), waist circumference (WC) ( $-2.0 \pm 1.3$  vs  $-0.3 \pm 2.0$  cm,  $P < 0.001$ ) and hip circumference (HC) ( $-2.5 \pm 1.5$  vs  $-0.3 \pm 1.8$  cm,  $P < 0.001$ ) compared with placebo. In addition, compared with placebo, carnitine administration in women with PCOS led to a significant reduction in fasting plasma glucose ( $-0.38 \pm 0.36$  vs  $+0.11 \pm 0.97$  mmol/l,  $P = 0.01$ ), serum insulin levels ( $-14.39 \pm 25.80$  vs  $+3.01 \pm 37.25$  pmol/l,  $P = 0.04$ ), homeostasis model of assessment-insulin resistance ( $-0.61 \pm 1.03$  vs  $+0.11 \pm 1.43$ ,  $P = 0.04$ ) and

dehydroepiandrosterone sulphate ( $-3.64 \pm 7.00$  vs  $-0.59 \pm 3.20$   $\mu$ mol/l,  $P = 0.03$ ).

**Conclusions** Overall, 12 weeks of carnitine administration in PCOS women resulted in reductions in weight, BMI, WC and HC, and beneficial effects on glycaemic control; however, it did not affect lipid profiles or free testosterone.

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## Introduction

Polycystic ovary syndrome (PCOS) is often characterized by obesity and impaired insulin function and affects about 7–10% of women during reproductive age.<sup>1</sup> Obesity may play a role in the aetiology of PCOS, and weight loss has been found to improve some of the clinical aspects of PCOS including menses regularity and fertility,<sup>2</sup> as well as many cardiovascular (CV) risk markers associated with PCOS such as insulin resistance (IR) and dyslipidaemia.<sup>3</sup> IR affects approximately 65% of women with PCOS<sup>4</sup> and can potentially increase the prevalence of impaired glucose tolerance, type 2 diabetes mellitus (T2DM)<sup>5</sup> and metabolic syndrome (MetS).<sup>6</sup>

Carnitine plays a substantial role in weight loss, glucose tolerance, insulin function and fatty acid metabolism. The potential mechanisms include increasing mitochondrial efflux of excess acyl groups from insulin-responsive tissues and facilitating transportation of the long-chain free fatty acids into the mitochondrial matrix.<sup>7–9</sup> Some studies have reported that circulating levels of free and total L-carnitine were significantly lower in PCOS women.<sup>8,10</sup> In addition, in a study by Ismail *et al.*,<sup>11</sup> combined L-carnitine and clomiphene citrate significantly improved both ovulation and cumulative pregnancy rates in patients with clomiphene-resistant PCOS. Taking intravenous L-carnitine

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(4 g/day) among patients with MetS for 7 days resulted in weight loss, improved fasting-induced hunger and cholesterol abnormalities.<sup>12</sup> Carnitine supplementation in human studies and animal models has indicated an improvement in parameters of glucose homeostasis, particularly during an insulin-resistant state.<sup>13,14</sup> However, carnitine administration did not affect weight loss outcomes in valproate-treated bipolar patients consuming an energy-restricted and low-fat diet.<sup>15</sup>

Although there is some evidence to indicate that carnitine administration may help to reduce body weight and improve markers of insulin metabolism and lipid profiles in individuals diagnosed with PCOS, the beneficial effects of carnitine supplementation on these markers has not yet been studied. This study aimed to determine the effect of carnitine administration on weight loss, parameters of glucose homeostasis and lipid profiles of overweight and/or obese women diagnosed with PCOS. We hypothesized that carnitine administration might reduce body weight and improve the metabolic status of PCOS patients.

## Subjects and methods

### Participants

This randomized, double-blind, placebo-controlled clinical trial was conducted from July 2015 to September 2015 among the women with PCOS referred to Taleghani Clinic, affiliated to Arak University of Medical Sciences (AUMS), Arak, Iran. The diagnosis of PCOS was made according to the Rotterdam criteria<sup>16</sup>: those with the two of the following criteria were considered as having PCOS: oligo- and/or anovulation (defined as delayed menses >35 days or <8 spontaneous haemorrhagic episodes/year), clinical (hirsutism using modified Ferriman–Gallwey score of  $\geq 8$ ) and/or biochemical signs of hyperandrogenism (total testosterone >1.7 nmol/l) and polycystic ovaries (12 or more follicles in each ovary measuring 2–9 mm in diameter and/or increased ovarian volume >10 ml<sup>3</sup>). PCOS women aged 18–40 years, BMI >25 kg/m<sup>2</sup> with phenotypes A (oligo/anovulation + hyperandrogenism + polycystic ovary morphology) and D (oligo/anovulation + polycystic ovary morphology), were included in the study. Exclusion criteria were as follows: subjects with hyperprolactinaemia, diabetes mellitus (DM), thyroid disease, subjects following a special diet or consuming drugs with an effect on hormonal profile like oral contraceptives (OCP), ovulation induction agents and anti-obesity therapies in the last 3 months before enrolment.<sup>17</sup> The primary outcome variable was homeostasis model of assessment–insulin resistance (HOMA-IR). To calculate sample size, we used the standard formula suggested for clinical trials by considering a type one error ( $\alpha$ ) of 0.05 and type two error ( $\beta$ ) of 0.20 (power = 80%). Based on the study of Molfino *et al.*,<sup>18</sup> we used 0.5 as SD and 0.4 as the difference in mean (d) of HOMA-IR as key variable. Based on this, we reached 25 subjects in each group. To allow for five dropouts in each group, the final sample size was determined to be 30 patients per group. The current study was approved by the Institutional Review Board at AUMS and was confirmed by the Ethics Committee of AUMS before beginning the trial. In addition, written informed

consent was taken from all participants prior to their enrolment. The trial was registered at the Iranian Clinical Trial Registry (IRCT code: IRCT201508025623N49).

### Study design

At the onset of the study, patients were matched according to BMI (25–29.9 and  $\geq 30$  kg/m<sup>2</sup>), age (<30 and  $\geq 30$  years) and phenotypes A (12 patients in each group) and D (18 patients in each group) of PCOS. Sixty patients with PCOS were randomized to receive either 250 mg carnitine supplements ( $N = 30$ ) or placebo per day ( $N = 30$ ) for 12 weeks. Randomization was performed by the use of computer-generated random numbers. Carnitine supplements and placebo (cellulose) were manufactured by AVECINA (Tehran, Iran) and Barij Essence Pharmaceutical Company (Kashan, Iran), respectively. The placebo was matched to carnitine capsules for shape, size and colour (Barij Essence Pharmaceutical Company, Kashan, Iran). Quality control of carnitine supplements was performed in the laboratory of Food and Drug Administration in Tehran, Iran, by high-performance liquid chromatography method. Following quality control, we found that the amount of carnitine in the prescribed capsules was at the range of 237–275 mg. Patients and researchers were masked to treatment randomization. Based on guidelines, all patients were taking metformin tablets at the initial dose of 500 mg, which was increased in a stepwise manner during the first 3 weeks to a total of 1500 mg/day.<sup>19</sup> The study patients were instructed not to change their usual diet and levels of physical activity throughout the study period as well as not to take any medications that might affect their weight and metabolic status during the 12-week intervention. To assess compliance, patients were asked to bring the medication containers. Compliance was checked through counting unused capsules. To increase compliance, all patients were received short reminders on their cell phones to take the supplements each day. In all subjects, both dietary and physical activity records were provided at week 3, 6 and 9 of treatment. To obtain macro- and micronutrient intakes of patients with PCOS based on these three-day food diaries, we used Nutritionist IV software (First Databank, San Bruno, CA, USA) modified for Iranian foods.

### Assessment of anthropometric measures

Height and weight (Seca, Hamburg, Germany) were recorded by standard protocols without shoes. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m<sup>2</sup>). Waist circumference (WC) was quantified at the midpoint level between the iliac crest and the lowest rib at the end of expiration. Hip circumference (HC) was determined at the maximum protuberance of the buttocks. All anthropometric measures were performed by a trained midwife.

### Outcomes

In the current study, primary outcomes were markers of insulin metabolism and secondary outcomes were body weight, BMI,

WC, HC, fasting plasma glucose (FPG), lipid profiles and androgenic hormones.

### Biochemical assessment

Ten millilitre fasting blood samples were collected at the beginning and end of the study at Arak reference laboratory in the early morning after a 12-h overnight fast. Samples were immediately centrifuged (Hettich D-78532, Tuttlingen, Germany) at  $1465 \times g$  for 10 min to separate serum. FPG and lipid concentrations were determined on the day of blood collection. Samples were then kept at  $-80^{\circ}\text{C}$  until the day of analysis at the AUMS reference laboratory. Commercial kits (Pars Azmun, Tehran, Iran) were used to assay FPG, serum triglycerides, VLDL, total, LDL and HDL cholesterol concentrations. All inter- and intra-assay CVs for FPG and lipid profiles measurements were less than 5%. Serum insulin concentrations were determined using enzyme-linked immunosorbent assay by ELISA kit (Monobind, California, USA) with intra- and interassay CVs of 2.6 and 4.7%, respectively. HOMA-IR, homeostatic model assessment-beta cell function (HOMA-B) and the quantitative insulin sensitivity check index (QUICKI) were calculated based on suggested formulas.<sup>20</sup> Serum-free testosterone and dehydroepiandrosterone sulphate (DHEAS) concentrations were measured using commercial kits (Monobind) with inter- and intra-assay CVs less than 5%.

### Statistical methods

Normal distribution of variables was assessed using Kolmogorov–Smirnov test. We performed the analysis according to the intention-to-treat (ITT) principle. To detect differences in general characteristics and dietary intakes between the two groups, we used independent samples Student's *t*-test.

Paired-samples *t*-test was used to detect within-group changes. To determine the effects of carnitine intake on glycaemic control and lipid concentrations, one-way repeated-measures ANOVA was used to evaluate the between-group changes in variables during the study. To assess some confounding variables including baseline biochemical indicators, age and baseline BMI, we used analysis of covariance (ANCOVA). *P* values of  $<0.05$  were considered as statistically significant. All statistical analyses were performed using the Statistical Package for Social Science version 17 (SPSS Inc., Chicago, IL, USA).

### Results

Three subjects in the carnitine group [withdrawn due to personal reasons ( $N = 3$ )] and three in the placebo group [withdrawn due to personal reasons ( $N = 3$ )] did not complete the intervention (Fig. 1). However, as the analysis was conducted based on ITT principle, all 60 patients with PCOS were included in the final analysis. On average, the rate of compliance was high, such that more than 90% of capsules were consumed throughout the study in both groups. No side effects were recorded following the administration of carnitine supplements in patients with PCOS throughout the study.

Mean age, height, weight at baseline and end-of-trial of study subjects were not statistically different between carnitine and placebo groups (Table 1). At the end of the 12 weeks, taking carnitine supplements resulted in a significant reduction in weight ( $-2.7 \pm 1.5$  vs  $+0.1 \pm 1.8$  kg,  $P < 0.001$ ), BMI ( $-1.1 \pm 0.6$  vs  $+0.1 \pm 0.7$  kg/m<sup>2</sup>,  $P < 0.001$ ), WC ( $-2.0 \pm 1.3$  vs  $-0.3 \pm 2.0$  cm,  $P < 0.001$ ) and HC change ( $-2.5 \pm 1.5$  vs  $-0.3 \pm 1.8$  cm,  $P < 0.001$ ) compared with placebo.

Based on the three-day dietary records obtained pre-intervention and throughout the intervention, no statistically significant

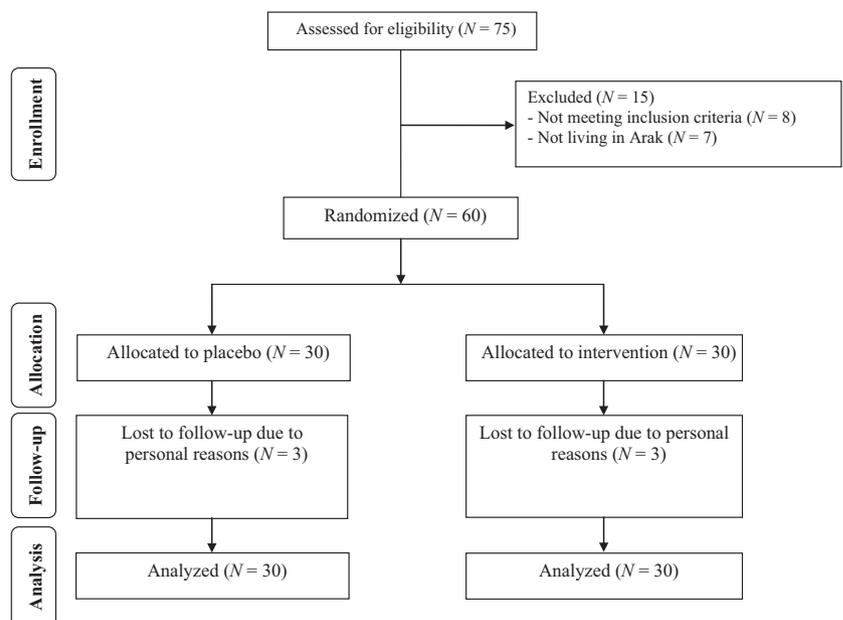


Fig. 1 Summary of patient flow.

**Table 1.** General characteristics of study participants

	Placebo group (N = 30)	Carnitine group (N = 30)	P†
Age (years)	25.5 ± 5.7	24.8 ± 5.5	0.61
Height (cm)	158.6 ± 3.8	157.3 ± 5.2	0.28
Weight at study baseline (kg)	72.9 ± 11.0	72.2 ± 10.2	0.78
Weight at end-of-trial (kg)	73.0 ± 11.8	69.5 ± 10.0	0.21
Weight change (kg)	0.1 ± 1.8	-2.7 ± 1.5	<0.001
BMI at study baseline (kg/m <sup>2</sup> )	28.9 ± 3.9	29.1 ± 3.4	0.85
BMI at end-of-trial (kg/m <sup>2</sup> )	29.0 ± 4.2	28.0 ± 3.3	0.33
BMI change (kg/m <sup>2</sup> )	0.1 ± 0.7	-1.1 ± 0.6	<0.001
WC at study baseline (cm)	89.3 ± 8.4	90.2 ± 12.2	0.70
WC at end-of-trial (cm)	89.0 ± 9.2	88.2 ± 12.0	0.79
WC change (cm)	-0.3 ± 2.0	-2.0 ± 1.3	<0.001
HC at study baseline (cm)	102.6 ± 8.9	101.3 ± 9.8	0.59
HC at end-of-trial (cm)	102.3 ± 9.6	98.8 ± 9.5	0.16
HC change (cm)	-0.3 ± 1.8	-2.5 ± 1.5	<0.001

Data are means ± SDs.

BMI, body mass index; HC, hip circumference; WC, waist circumference.

†Obtained from independent *t*-test.

change was seen between the two groups in terms of macro- and micronutrient intakes (Table 2).

Compared with the placebo, carnitine administration in women with PCOS led to a significant decrease in FPG ( $-0.38 \pm 0.36$  vs  $+0.11 \pm 0.97$  mmol/l,  $P = 0.01$ ), serum insulin levels ( $-14.39 \pm 25.80$  vs  $+3.01 \pm 37.25$  pmol/l,  $P = 0.04$ ), HOMA-IR ( $-0.61 \pm 1.03$  vs  $+0.11 \pm 1.43$ ,  $P = 0.04$ ) and DHEAS ( $-3.64 \pm 7.00$  vs  $-0.59 \pm 3.20$  µmol/l,  $P = 0.03$ ) (Table 3). We did not find any significant effect of carnitine supplementation on lipid profiles or free testosterone.

## Discussion

In the current study, for the first time, we assessed the effects of 250 mg of carnitine supplementation on weight loss, glycaemic and lipid concentrations among women diagnosed with PCOS. We found that taking carnitine supplements reduced body weight, BMI, WC and HC and improved glycaemic control in PCOS patients, but did not affect lipid profiles or free testosterone.

Patients with PCOS are susceptible to impaired carbohydrate and lipid metabolism.<sup>21</sup> We found that carnitine supplementation for 12 weeks among women with PCOS resulted in a significant reduction in body weight, BMI, WC and HC compared with placebo. Some previous studies have reported that carnitine supplementation among patients without PCOS may reduce body weight and BMI. Supporting our findings, taking 4 g/day of intravenous L-carnitine among patients with MetS resulted in a significant decrease in body weight and WC.<sup>12</sup> In experimental models, L-carnitine administration led to an increase of total energy expenditure in obese rats with insulin resistance<sup>7</sup> and weight loss in calorie-restricted cats receiving 40% of their main-

**Table 2.** Dietary intakes of study participants throughout the study

	Placebo group (N = 30)	Carnitine group (N = 30)	P†
Energy (kcal/day)	2517 ± 169	2462 ± 204	0.25
Carbohydrates (g/day)	344.1 ± 38.5	341.3 ± 46.7	0.80
Protein (g/day)	88.0 ± 10.0	84.2 ± 15.3	0.25
Fat (g/day)	92.5 ± 13.1	88.4 ± 17.8	0.32
SFAs (g/day)	27.0 ± 5.0	25.1 ± 6.7	0.21
PUFAs (g/day)	29.7 ± 8.4	29.1 ± 8.1	0.80
MUFAs (g/day)	25.0 ± 4.5	24.6 ± 7.9	0.80
Cholesterol (mg/day)	199.3 ± 90.9	202.9 ± 125.1	0.89
TDF (g/day)	19.8 ± 4.6	18.4 ± 4.9	0.24
Magnesium (mg/day)	320.3 ± 81.7	299.0 ± 92.9	0.33
Zinc (mg/day)	11.1 ± 2.1	10.1 ± 3.6	0.19
Manganese (mg/day)	2.5 ± 0.7	2.3 ± 0.6	0.35

Data are means ± standard deviations.

SFAs, saturated fatty acids; PUFAs, polyunsaturated fatty acids; MUFAs, monounsaturated fatty acids; TDF, total dietary fibre.

†Obtained from independent *t*-test.

tenance energy requirement.<sup>22</sup> However, carnitine administration (15 mg/kg/day) among valproate-treated bipolar patients receiving an energy-restricted and low-fat diet for 26 weeks did not affect weight loss outcomes.<sup>15</sup> In addition, our findings differ from previous studies, which indicated that oral L-carnitine supplementation had no effect on weight loss among patients with impaired glucose metabolism<sup>18</sup> or in obese women.<sup>23</sup> Carnitine intake may decrease body weight, BMI, WC and HC through increasing β-oxidation of fatty acids<sup>24</sup> and basal metabolic rates.<sup>25</sup>

This study demonstrated that carnitine intake for 12 weeks among women with PCOS led to a significant decrease in FPG, serum insulin levels and HOMA-IR compared with placebo, but unchanged HOMA-B and QUICKI. Some studies have reported the beneficial effects of carnitine supplementation on glucose homeostasis parameters. This is similar to the findings of a study by Molino *et al.*<sup>18</sup> which demonstrated that supplementation with 2 g oral L-carnitine (twice per day) for 10 days led to a significant decrease in insulin levels and insulin resistance. Furthermore, intravenous infusion of L-carnitine with a priming dose (3 mmol) followed by a constant rate (1.7 pmol/min) for 3 h in insulin-resistant subjects with T2DM reduced insulin resistance.<sup>26</sup> Similar findings were seen following supplementation with oral acetyl-L-carnitine (1 g/twice per day) for 24 weeks in participants at increased risk of cardiovascular disease.<sup>27</sup> Insulin resistance is common in patients with PCOS, and it is likely to be explained by a high rate of obesity among patients. However, taking carnitine supplements (2 g/day) did not affect glycaemic parameters among patients with T2DM for 6 months.<sup>28</sup> L-carnitine supplementation might improve parameters of insulin metabolism by modulating the expression of glycolytic and gluconeogenic enzymes,<sup>29</sup> improving mitochondrial glucose oxidation, and acting as a transport molecule for free fatty acids or as an important acetyl-group donor in high-energy metabolism situation.<sup>30</sup>

**Table 3.** The effect of carnitine administration on glycaemic control and lipid profiles

	Placebo group (N = 30)			Carnitine group (N = 30)			P†
	Baseline	End-of-trial	Change	Baseline	End-of-trial	Change	
FPG (mmol/l)	4.84 ± 0.36	4.95 ± 0.83	0.11 ± 0.97	5.26 ± 0.57	4.88 ± 0.56	-0.38 ± 0.36	0.01
Insulin (pmol/l)	74.40 ± 37.20	77.41 ± 45.62	3.01 ± 37.25	66.00 ± 31.82	51.61 ± 24.05	-14.39 ± 25.80	0.04
HOMA-IR	2.71 ± 1.52	2.82 ± 1.73	0.11 ± 1.43	2.62 ± 1.34	2.01 ± 1.04	-0.61 ± 1.03	0.04
HOMA-B	47.22 ± 23.81	49.23 ± 30.51	2.01 ± 25.32	38.21 ± 21.44	29.02 ± 15.43	-9.19 ± 18.02	0.05
QUICKI	0.33 ± 0.02	0.34 ± 0.03	0.01 ± 0.03	0.33 ± 0.02	0.35 ± 0.02	0.02 ± 0.02	0.19
Triglycerides (mmol/l)	1.16 ± 0.42	1.31 ± 0.53	0.15 ± 0.46	1.50 ± 0.77	1.42 ± 0.60	-0.07 ± 0.48	0.06
VLDL cholesterol (mmol/l)	0.53 ± 0.19	0.59 ± 0.24	0.06 ± 0.21	0.68 ± 0.35	0.64 ± 0.27	-0.04 ± 0.22	0.06
Total cholesterol (mmol/l)	4.39 ± 0.83	4.26 ± 0.81	-0.13 ± 1.00	4.59 ± 1.02	4.64 ± 0.79	0.05 ± 0.83	0.44
LDL cholesterol (mmol/l)	2.59 ± 0.69	2.35 ± 0.74	-0.24 ± 0.85	2.75 ± 0.80	2.68 ± 0.77	-0.07 ± 0.79	0.42
HDL cholesterol (mmol/l)	1.26 ± 0.31	1.31 ± 0.49	0.05 ± 0.42	1.15 ± 0.25	1.31 ± 0.27	0.16 ± 0.11	0.16
Free testosterone (pmol/l)	5.41 ± 4.12	5.69 ± 4.19	0.28 ± 1.76	4.68 ± 1.94	4.37 ± 2.32	-0.31 ± 1.76	0.18
DHEAS (µmol/l)	11.34 ± 5.32	10.75 ± 6.19	-0.59 ± 3.20	11.18 ± 6.21	7.54 ± 7.14	-3.64 ± 7.00	0.03

All values are means ± SDs.

DHEAS, dehydroepiandrosterone sulphate; FPG, fasting plasma glucose; HOMA-IR, homoeostasis model of assessment-estimated insulin resistance; HOMA-B, homoeostasis model of assessment-estimated b-cell function; QUICKI, quantitative insulin sensitivity check index.

†Obtained from independent *t*-test.

Our study showed that carnitine administration in women with PCOS for 12 weeks did not influence lipid concentrations compared with placebo. This is similar to findings of a study by Derosa *et al.*<sup>28</sup> which showed there was no significant change in lipid profiles after taking 1 g L-carnitine daily for 6 months among hypercholesterolaemic patients with T2DM. Furthermore, six months of L-carnitine infusions (20 mg/kg) postdialysis in haemodialysis patients did not affect lipid profiles.<sup>31</sup> However, two other studies showed different findings: administration of 2 g oral L-carnitine per day for 3 months among patients with diabetes was associated with an improvement in lipid profiles<sup>32</sup> and oral supplementation of 1 g L-carnitine for 16 weeks in haemodialysis patients was associated with decreased triglycerides and total cholesterol with increased HDL cholesterol levels.<sup>33</sup> Different study designs, different dosages of carnitine used in our study, the short duration of intervention and the characteristics of study patients might provide some reasons for these discrepant findings.

The current study had some limitations. Firstly, the rate of compliance was higher than 90% throughout the study in both groups. However, due to limited funding, we did not evaluate the compliance through quantification of carnitine concentrations. Secondly, we did not assess biomarkers of inflammation, oxidative stress and sex hormone-binding globulin. Thirdly, the sample size was small. Fourthly, we used 4 g carnitine supplementation per day to provide statistical power for our study. This may result in decreasing the power calculation used and the reduction of FPG in the carnitine group may have been caused by regression to the mean. Future studies with crossover design, longer duration of intervention and bigger sample size are needed to confirm the validity of our findings. Moreover, the comparison of carnitine intake in patients with and without PCOS would be of interest, as a possible topic for future study. In the current study, FPG alone was obtained rather than

performing the area under the curve following an oral glucose tolerance test for glucose and insulin at baseline and after 12 weeks of experiment. As PCOS is primarily a postprandial insulin-resistant state, these data would have strengthened the study. Nevertheless, the HOMA-IR changes seen in the study have shown some interesting results. It must be kept in mind that in previous studies investigating the beneficial effects of carnitine supplementation on metabolic status in relation to other diseases doses of 500–4000 mg were used.<sup>18,34</sup> However, due to the limited data about the appropriate dosage of carnitine for women with PCOS, to be safe, we used a dose of a 250 mg. Further long-term studies with higher doses of carnitine are needed to examine the effect of carnitine supplementation on markers of insulin resistance, lipid profiles and hormonal outcomes in PCOS patients.

Overall, 12 weeks of carnitine administration among women diagnosed with PCOS had beneficial effects on weight, BMI, WC, HC and glycaemic control; however, it did not affect lipid profiles or free testosterone.

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## Authors' contributions

ZA contributed in conception, design, statistical analysis and drafting of the manuscript. MS, MJ, FA-E, MR and BT contributed in data collection and manuscript drafting. ZA supervised the study. All authors approved the final version for submission.

## Conflicts of interest

Nothing to declare.

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