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Effects of Chromium and Carnitine Co-supplementation on Body Weight and Metabolic Profiles in Overweight and Obese Women with Polycystic Ovary Syndrome: a Randomized, Double-Blind, Placebo-Controlled Trial

Mehri Jamilian[1] · Fatemeh Foroozanfard[2] · Elham Kavossian[2] · Mersedeh Kia[3] · Esmat Aghadavod[4] · Elaheh Amirani[4] · Zatollah Asemi[4]

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Abstract

The primary aim of our study was to determine the influence of taking chromium plus carnitine on insulin resistance, with a secondary objective of evaluating the influences on lipid profiles and weight loss in overweight subjects with polycystic ovary syndrome (PCOS). In a 12-week randomized, double-blind, placebo-controlled clinical trial, 54 overweight women were randomly assigned to receive either supplements (200 μg/day chromium picolinate plus 1000 mg/day carnitine) or placebo (27/each group). Chromium and carnitine co-supplementation decreased weight (− 3.6 ± 1.8 vs. − 1.0 ± 0.7 kg, $P < 0.001$), BMI (− 1.3 ± 0.7 vs. − 0.3 ± 0.3 kg/m^2, $P < 0.001$), fasting plasma glucose (FPG) (− 5.1 ± 6.0 vs. − 1.1 ± 4.9 mg/dL, $P = 0.01$), insulin (− 2.0 ± 1.4 vs. − 0.2 ± 1.2 μIU/mL, $P < 0.001$), insulin resistance (− 0.5 ± 0.4 vs. − 0.04 ± 0.3, $P < 0.001$), triglycerides (− 18.0 ± 25.2 vs. + 5.5 ± 14.4 mg/dL, $P < 0.001$), total (− 17.0 ± 20.3 vs. + 3.6 ± 12.0 mg/dL, $P < 0.001$), and LDL cholesterol (− 13.3 ± 19.2 vs. + 1.4 ± 13.3 mg/dL, $P = 0.002$), and elevated insulin sensitivity (+ 0.007 ± 0.005 vs. + 0.002 ± 0.005, $P < 0.001$). In addition, co-supplementation upregulated peroxisome proliferator-activated receptor gamma ($P = 0.02$) and low-density lipoprotein receptor expression ($P = 0.02$). Overall, chromium and carnitine co-supplementation for 12 weeks to overweight women with PCOS had beneficial effects on body weight, glycemic control, lipid profiles except HDL cholesterol levels, and gene expression of PPAR-γ and LDLR. Clinical trial registration number: http://www.irct.ir: IRCT20170513033941N38.

Keywords Carnitine · Chromium · Body weight · Metabolic profiles · Polycystic ovary syndrome

Introduction

Polycystic ovary syndrome (PCOS) is the most prevalent endocrine disorder among women of reproductive age [1]. It is associated with increased infertility, gestational diabetes mellitus (GDM), type 2 diabetes mellitus (T2DM), non-alcoholic fatty liver disease, and cardiovascular disease (CVD) [2–4]. Hyperinsulinemia increases ovulatory disruption among PCOS patients [5]. Prior evidence showed that dyslipidemia including low HDL cholesterol, high LDL cholesterol, and triglycerides levels are common among women with PCOS [6], as well as reduction in mRNA levels LDL receptor (LDLR) reduction within adipose tissue of these patients [7]. The lifestyle modifications including weight loss and dietary changes are essential for better management of PCOS [5]. Recently, the beneficial effects of dietary supplementation including zinc and vitamin D among patients with PCOS are documented in several studies [8, 9].

Carnitine transports free fatty acids into mitochondria; therefore, it involves in fat metabolism and energy expenditure [10]. In addition, chromium may play an important role in decreasing insulin resistance and lipid abnormalities, as well as weight loss process in the body [11]. Many studies examined the effects of chromium and carnitine supplementation on

✉ Zatollah Asemi
asemi_r@yahoo.com

[1] Traditional and Complementary Medicine Research Center, Arak University of Medical Sciences, Arak, Iran

[2] Gametogenesis Research Center, Kashan University of Medical Sciences, Kashan, I.R., Iran

[3] Department of Midwifery, Gorgan Branch, Islamic Azad University, Gorgan, Iran

[4] Research Center for Biochemistry and Nutrition in Metabolic Diseases, Kashan University of Medical Sciences, Kashan, I.R., Iran

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the metabolic profiles [12–14]. Earlier, it was reported that chromium supplementation to women with PCOS and diabetic patients significantly improved glucose homeostasis parameters and some lipids profiles [12, 15], although several studies did not report lipid- and glucose-lowering effects of chromium supplements [16]. Previous evidence showed that carnitine supplementation had beneficial effects on weight loss, glycemic control, and lipid profiles in patients with CVD and PCOS [17, 18].

Previous studies have demonstrated that joint supplementation of chromium and carnitine is much more efficient in influencing metabolic profiles that single chromium or carnitine supplementation. Several animal studies evaluated the effects of chromium and carnitine co-supplementation on metabolic profiles. In a study conducted by Zhou et al. [19], yeast chromium (300 μg/kg diet) and/or L-carnitine (100 mg/kg diet) for 8 weeks in sheep reduced insulin levels, while glucose levels did not change. In addition, Wang et al. [20] found a significant synergistic effect of chromium and carnitine on blood glucose in chicken. This evidence suggests that chromium and carnitine may have better effects on glycemic control and lipid profiles. Therefore, we conducted this study to determine the effects of carnitine and chromium co-supplementation on body weight, metabolic and genetic profiles in overweight and obese women with PCOS.

Subjects and Methods

In a 12-week randomized, double-blind, placebo-controlled clinical trial, 54 overweight women were randomly assigned to receive either supplements (200 μg/day chromium picolinate plus 1000 mg/day carnitine) or placebo (starch) (27/each group). Carnitine, chromium, and the placebo were manufactured by Avecinna (Tehran, Iran), twenty-first century (Arizona, USA), and Barij Essence (Kashan, Iran), respectively. The appearance of the placebo, carnitine, and chromium capsules, such as color, shape, size, and packaging, were totally similar. Randomization and allocation to intervention groups were blinded from the researcher and subjects until the main analyses were completed. At the clinic, a midwife conducted the randomized allocation sequence, and enrolment and assignment of the participants to the groups. During the study, use of carnitine, chromium supplements, and the placebos was examined by asking subjects to return the medication containers and through brief daily cell phone reminders to take the supplements. Study protocol was published in the Iranian website for registration of clinical trials (www.irct.ir; no: IRCT20170513033941N38). Eligible study participants were overweight and obese women (BMI > 25 kg/m^2) with PCOS diagnosed based on the Rotterdam criteria [21], aged 18–40 years who referred to the outpatient Teleghani Clinic in Arak, Iran, between June and November 2018. The study protocol was approved by the Ethics Committee of Arak University of Medical Sciences (AUMS). Written informed consent was obtained from all participants prior to the intervention. Exclusion criteria were as follows: pregnancy, adrenal hyperplasia, androgen-secreting tumors, hyperprolactinemia, thyroid dysfunction, and diabetes prior to study inclusion. All subjects completed 3-day diet recall form at weeks 0, 3, 6, 9, and 12 of the intervention. Dietary intakes of macro- and micronutrients were calculated by nutritionist IV software (First Databank, San Bruno, CA). Physical activity was defined as metabolic equivalents (METs) in hours per day. To calculate the METs for each participant, we multiplied the times (in hour per day) reported for each physical activity by its related METs coefficient by standard tables [22].

Anthropometric Measures

A midwife took anthropometric measurements at the clinic at baseline and the end of the intervention. Height and weight (Seca, Hamburg, Germany) were measured light clothing with shoes removed. Body mass index (BMI) was calculated as weight (in kilograms) divided by height (in meters) squared.

Assessment of Outcomes

Glycemic control was considered as the primary outcome. Lipid profiles, peroxisome proliferator-activated receptor gamma (PPAR-γ), low-density lipoprotein receptor (LDLR), and glucose transporter 1 (GLUT-1) expression were recognized as the secondary outcomes.

Biochemical Assessment

Fasting blood samples were collected from participants (15 mL) at weeks 0 and 12 of the intervention. To determine fasting plasma glucose (FPG) and lipid profiles, enzymatic kits (Pars Azmun, Tehran, Iran) with inter- and intra-assay coefficient variances (CVs) lower than 5% were used. Serum insulin values were assessed using an ELISA kit (Monobind, California, USA) with the intra- and inter-assay CVs lower than 6%. The homeostatic model assessment for insulin resistance (HOMA-IR), homeostatic model assessment-beta cell function (HOMA-B), and the quantitative insulin sensitivity check index (QUICKI) were determined according to the suggested formulas [23].

RNA Extraction and Real-Time PCR

PPAR-γ, GLUT-1, and LDLR expression were evaluated by quantitative RT-PCR, using the LightCycler technology (Roche Diagnostics, Rotkreuz, Switzerland) with SYBR green detection and Amplicon Kit (Table 1).

Table 1 Specific primers used for real-time quantitative PCR

Gene	Primer	Product size (bp)	Annealing temperature (C)
GAPDH	F: AAGCTCATTTCCTGGTATGACAACG R: TCTTCCTCTTGTGCTCTTGCTGG	126	61.3
PPAR-γ	F: ATGACAGACCTCAGACAGATTG R: AATGTTGGCAGTGGCTCAG	210	54
GLUT-1	F: TATCTGAGCATCGTGGCCAT R: AAGACGTAGGGACCACACAG	238	62.1
LDLR	F: ACTTACGGACAGACAGACAG R: GGCCACACATCCCATGATTC	223	57

GAPDH, glyceraldehyde-3-phosphate dehydrogenase; GLUT-1, glucose transporter 1; LDLR, low-density lipoprotein receptor; PPAR-γ, peroxisome proliferator-activated receptor gamma

Statistical Analyses

In the present study, we used sample size estimation formula for randomized clinical trials where type one (α) and type two errors (β) were 0.05 and 0.20 (power = 80%), respectively. According to a previous study [18], we used 1.03 as SD and 0.83 as the change in mean (d) of HOMA-IR as a main variable. Based on the formula, we needed 25 patients in each group; after considering 20% dropout in each group, the final sample size was 30 participants in each group.

The Kolmogorov-Smirnov test was done to determine the normality of variables. Independent sample t test was done to compare changes in anthropometric values and dietary intakes between the two groups. Two-way analysis of variance (ANOVA) for repeated measures was done to compare changes in gene expression related to PPAR-γ, GLUT-1, and LDLR between the two groups. To determine the effects of chromium and carnitine co-administration on metabolic profiles, we used two-way ANOVA for repeated measures. In these analyses, the treatments (placebo and chromium plus carnitine groups) were regarded as between-subject factors and time was considered as within-subject factor. Significance of the treatment effects was presented as the mean differences with 95% confidence interval. P values < 0.05 were considered statistically significant.

Results

During the treatment, six participants dropped out of the study due to personal reasons ($n = 3$ each group) (Fig. 1). Finally, 54 participants [placebo ($n = 27$) and supplements ($n = 27$)] completed the trial.

The mean age, height, BMI, and weight of patients at baseline and after the 12-week intervention were not significant (Table 2).

The mean intake of macro- and micronutrients during the treatment was not significant between the two groups (Table 3).

Chromium and carnitine co-supplementation decreased weight (− 3.6 ± 1.8 vs. − 1.0 ± 0.7 kg, $P < 0.001$), BMI (− 1.3 ± 0.7 vs. − 0.3 ± 0.3 kg/m^2, $P < 0.001$) (Table 2), FPG (− 5.1 ± 6.0 vs. − 1.1 ± 4.9 mg/dL, $P = 0.01$), insulin (− 2.0 ± 1.4 vs. − 0.2 ± 1.2 μIU/mL, $P < 0.001$), HOMA-IR (− 0.5 ± 0.4 vs. − 0.04 ± 0.3, $P < 0.001$), triglycerides (− 18.0 ± 25.2 vs. + 5.5 ± 14.4 mg/dL, $P < 0.001$), total (− 17.0 ± 20.3 vs. + 3.6 ± 12.0 mg/dL, $P < 0.001$) and LDL cholesterol (− 13.3 ± 19.2 vs. + 1.4 ± 13.3 mg/dL, $P = 0.002$), and elevated QUICKI (+ 0.007 ± 0.005 vs. + 0.002 ± 0.005, $P < 0.001$) (Table 4). In addition, co-supplementation upregulated PPAR-γ ($P = 0.02$) and LDLR expression ($P = 0.02$) (Fig. 2).

Discussion

In the current study, we investigated the effects of chromium and carnitine co-supplementation for 12 weeks on body weight, glycemic control, lipid concentrations, and gene expression related to insulin and lipid among women with PCOS. We found that taking combined chromium and carnitine supplements by women with PCOS significantly improved body weight, BMI, glycemic control, lipid parameters except HDL cholesterol levels, and gene expression related to insulin and lipid.

Effects on Body Weight and BMI

PCOS patients due to hyperinsulinemia and androgen excess are susceptible to an increased body weight which is associated with further reduction in insulin sensitivity and elevated the risk of metabolic syndrome [4]. We found that chromium and carnitine co-supplementation for 12 weeks to women with PCOS significantly decreased weight and BMI. Our findings were in consistent with the results of some meta-analyses

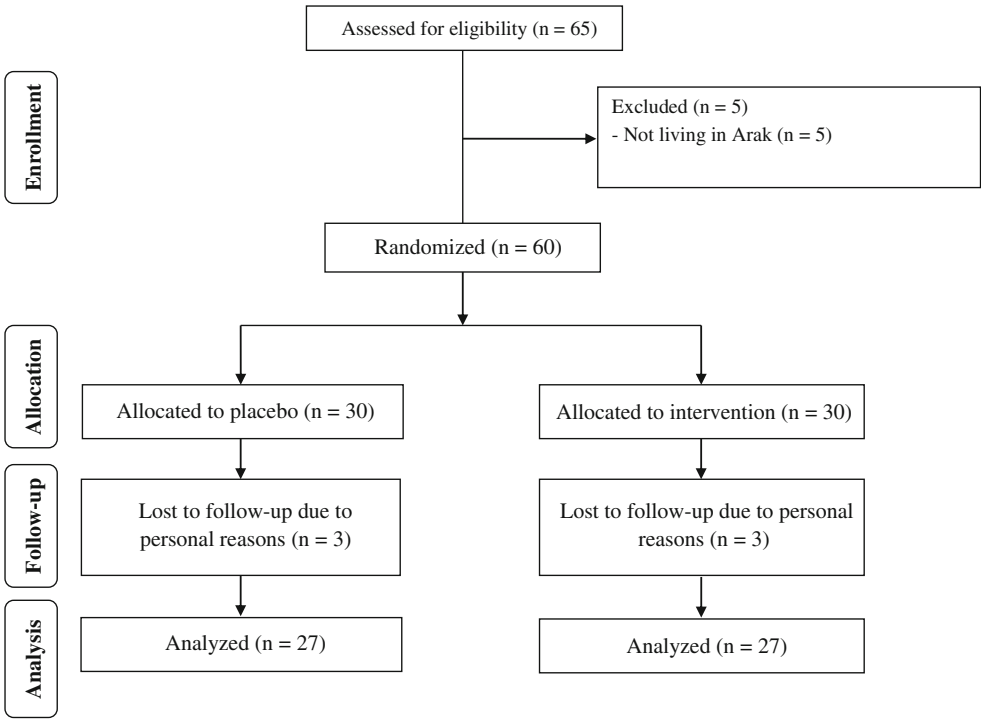

Fig. 1 Summary of patient flow diagram

evaluating the effects of chromium or carnitine supplementation on weight and BMI [24–26]. However, in a meta-analysis, chromium supplementation did not reduce BMI among women with PCOS [27]. The controversial findings might be mediated by different study designs, baseline values of measured variables, baseline levels of chromium and carnitine, different dosages, and type of chromium and carnitine used as well as different participants' characteristics. Chromium may reduce body weight by insulin sensitizing effects, stimulating thermogenesis, and suppressing the appetite through stimulating insulin-sensitive glucoreceptors in the brain [21]. Carnitine contributes to activation of the glycolytic pathways and has an important role in fat metabolism by transformation of long-chain fatty acids across the mitochondrial membrane and due to the effects on glucose and lipid metabolism increase energy expenditure which in turn may help weight loss [24].

Effects on Glycemic Control

In the current study, we observed the favorable effects of chromium and carnitine co-supplementation on glycemic status for 12 weeks among PCOS subjects. On the basis of available researches, chromium supplementation may have some

Table 2 General characteristics of study participants[1]

	Placebo group ($n = 27$)	Chromium and carnitine group ($n = 27$)	P^2
Age (y)	27.4 ± 5.3	29.6 ± 4.3	0.15
Height (cm)	164.7 ± 6.0	163.0 ± 4.9	0.25
Weight at study baseline (kg)	75.6 ± 5.9	77.6 ± 9.1	0.41
Weight at end-of-trial (kg)	74.8 ± 6.0	73.9 ± 8.7	0.67
Weight change (kg)	− 1.0 ± 0.7	− 3.6 ± 1.8	< 0.001
BMI at study baseline (kg/m^2)	28.0 ± 2.3	29.1 ± 2.8	0.10
BMI at end-of-trial (kg/m^2)	27.6 ± 2.2	27.8 ± 2.7	0.78
BMI change (kg/m^2)	− 0.3 ± 0.3	− 1.3 ± 0.7	< 0.001
MET-h/day at study baseline	26.2 ± 1.8	26.7 ± 1.6	0.27
MET-h/day at end-of-trial	26.5 ± 1.8	27.1 ± 1.5	0.19
MET-h/day change	0.3 ± 0.5	0.4 ± 0.4	0.53

[1] Data are means ± SDs

[2] Obtained from independent *t* test. METs, metabolic equivalents

Table 3 Mean dietary intakes of study participants throughout the study

	Placebo group ($n = 27$)	Chromium and carnitine group ($n = 27$)	P^1
Energy (kcal/d)	2252 ± 172	2327 ± 277	0.24
Carbohydrates (g/d)	307.9 ± 50.1	318.8 ± 59.6	0.47
Protein (g/d)	85.5 ± 15.1	83.5 ± 13.6	0.61
Fat (g/d)	78.5 ± 13.5	83.4 ± 16.7	0.23
SFAs (g/d)	23.0 ± 6.2	16.4 ± 2.1	0.47
PUFAs (g/d)	26.3 ± 5.3	28.1 ± 7.4	0.31
MUFAs (g/d)	20.4 ± 5.2	21.6 ± 5.8	0.42
Cholesterol (mg/d)	202.0 ± 125.9	223.4 ± 132.6	0.54
TDF (g/d)	16.9 ± 4.4	17.8 ± 5.5	0.49
Chromium (μg/d)	34.1 ± 10.7	36.5 ± 10.9	0.65
Magnesium (mg/d)	260.4 ± 43.7	270.7 ± 66.5	0.50
Zinc (mg/d)	9.6 ± 2.7	9.6 ± 2.5	0.97
Manganese (mg/d)	2.0 ± 0.7	2.1 ± 0.8	0.55

Data are means ± SDs

[1] Obtained from independent *t* test

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

beneficial effects on glycemic control. Suksomboon et al. [12] demonstrated that chromium supplementation significantly reduced FPG and HbA1c in diabetic patients. In addition, Samimi et al. [18] indicated that carnitine supplementation led to a significant reduction in FPG, insulin levels and HOMA-IR, but did not affect QUICKI. However, Fazelian et al. [26] reported that taking chromium by PCOS women did not influence FPG. Moreover, Tang et al. [27] found that chromium supplementation to women with PCOS decreased HOMA-IR and had no beneficial effects on other parameters of glycemic control, including FPG, insulin, and QUICKI. Decreased glucose levels by chromium is accompanied by an increase in hepatic glucose uptake, the activity of glycolytic enzymes, including glucokinase, phosphofructokinase, and pyruvate kinase [28]. In consistent with our study, the results of a meta-analysis by Xu et al. [29] revealed that carnitine could improve HOMA-IR. Vidal-Casariego et al. [30] also reported that carnitine supplementation in T2DM patients reduced FPG, but did not influence HbA1c. In addition, a 12-month treatment with orlistat plus carnitine compared with orlistat alone resulted in a better improvement in FPG, HbA1C, and HOMA-IR among uncontrolled T2DM subjects [31]. However, Derosa et al. [32] did not find any significant effect of 6-month carnitine consumption on fasting glucose, HbA1C and insulin levels in hypercholesterolemic T2DM subjects. Suggested mechanisms supporting favorable effects of carnitine on glucose metabolism include increasing mitochondrial oxidation of long-chain fatty acids, which in turn reduce insulin resistance, modulating pyruvate dehydrogenase complex activity, regulating gene expression of glycolytic and gluconeogenic enzymes, stimulating of insulin and insulin-like growth factor-1 cascade [10].

Our findings showed that chromium and carnitine co-supplementation increased gene expression of PPAR-γ, but did not affect gene expression of GLUT-1. Several studies have evaluated the effects of chromium or carnitine supplementation to these genetic responses. Administration of chromium picolinate alone or in combination with biotin increased PPAR-γ expression in type 2 diabetic rat model [33]. Animal experiments have reported that carnitine or its derivatives supplementation improved glucose and lipid metabolism by regulation of PPAR-γ, GLUT-2 and GLUT-4 mRNA expression [34, 35]. In addition, in vitro treatment of rat sertoli cell with carnitine and L-acetylcarnitine alone and in combination increased mRNA expression of GLUT-1 and decreased of insulin-like growth factor binding protein-4 mRNA [36]. PPAR-γ mainly expresses in adipose tissue and involves in the reduction of insulin resistance, modification of glucose metabolism and lipid storage [37, 38]. It may regulate early stages in gene expression of GLUT-4 [39].

Effects on Lipid Parameters

In the current trial, we observed beneficial effects of chromium and carnitine co-administration on lipid profiles except HDL cholesterol levels among women with PCOS. Currently, we demonstrated that chromium administration for 8 weeks to PCOS women candidate for in vitro fertilization significantly decreased total, VLDL cholesterol, and triglycerides levels, but did not affect other lipid profiles [40]. Although in some studies, chromium supplementation did not affect lipid parameters among patient with metabolic diseases [15, 41]. Chromium may improve lipid metabolism through altering the activity of lipoprotein lipase and

Table 4 Metabolic profiles at baseline and after the 12-week intervention in women with polycystic ovary syndrome that received either carnitine plus chromium supplements or placebo[1]

	Placebo group ($n = 27$)			Chromium and carnitine group ($n = 27$)			P^2
	Wk0	Wk12	Change	Wk0	Wk12	Change	
FPG (mg/dL)	93.5 ± 6.4	92.4 ± 7.3	− 1.1 ± 4.9	94.1 ± 10.4	89.0 ± 8.4	− 5.1 ± 6.0	0.01
Insulin (μIU/mL)	11.5 ± 2.2	11.3 ± 2.7	− 0.2 ± 1.2	12.5 ± 2.1	10.5 ± 1.7	− 2.0 ± 1.4	< 0.001
HOMA-IR	2.6 ± 0.5	2.6 ± 0.6	− 0.04 ± 0.3	2.9 ± 0.6	2.4 ± 0.4	− 0.5 ± 0.4	< 0.001
HOMA-B	141.2 ± 40.1	144.3 ± 44.5	3.1 ± 17.7	190.5 ± 200.3	181.7 ± 142.7	− 8.8 ± 128.9	0.63
QUICKI	0.33 ± 0.01	0.33 ± 0.01	0.002 ± 0.005	0.32 ± 0.009	0.33 ± 0.008	0.007 ± 0.005	< 0.001
Triglycerides (mg/dL)	159.0 ± 35.1	164.5 ± 33.2	5.5 ± 14.4	154.4 ± 49.7	136.4 ± 38.4	− 18.0 ± 25.2	< 0.001
VLDL cholesterol (mg/dL)	31.8 ± 7.0	32.9 ± 6.5	1.1 ± 2.9	30.9 ± 9.9	27.3 ± 7.7	− 3.6 ± 5.0	< 0.001
Total cholesterol (mg/dL)	192.0 ± 35.1	195.6 ± 34.6	3.6 ± 12.0	202.9 ± 45.6	185.9 ± 36.0	− 17.0 ± 20.3	< 0.001
LDL cholesterol (mg/dL)	109.8 ± 38.5	111.2 ± 39.2	1.4 ± 13.3	123.9 ± 39.9	110.6 ± 33.1	− 13.3 ± 19.2	0.002
HDL cholesterol (mg/dL)	50.4 ± 6.0	51.4 ± 8.7	1.0 ± 7.3	48.0 ± 10.9	48.0 ± 9.3	− 0.03 ± 5.8	0.56
Total-/HDL cholesterol ratio	3.8 ± 0.9	3.9 ± 1.0	0.1 ± 0.6	4.3 ± 1.0	4.0 ± 0.8	− 0.3 ± 0.7	0.01

[1] All values are means ± SDs

[2] *P* values represent the time × group interaction (computed by analysis of the repeated measures ANOVA)

FPG, fasting plasma glucose; HOMA-IR, homeostasis model of assessment-insulin resistance; homeostasis model of assessment-estimated b cell function; HDL cholesterol, high density lipoprotein cholesterol; LDL cholesterol, low-density lipoprotein cholesterol; QUICKI, quantitative insulin sensitivity check index; VLDL cholesterol, very low density lipoprotein cholesterol

inhibiting the transcription of rate limiting enzymes in the fat synthesis pathway, such as fatty acid synthetase and acetyl-coA carboxylase [19]. A number of animal and human studies have evaluated the effects of carnitine on lipid profiles. Similar to our findings, Lee et al. [17] reported that a 12-week supplementation with carnitine decreased triglycerides. Moreover, carnitine supplementation for 12 weeks in diabetic patients led to a significant reduction in total, LDL cholesterol, and triglycerides, while HDL cholesterol levels were increased [42]. A meta-analysis conducted by Vidal-Casariego et al. [30], it was observed that carnitine supplementation to T2DM patients reduced total and LDL cholesterol, but did not affect triglycerides and HDL cholesterol levels. However, Derosa et al. [32] did not report any significant improvement in total, LDL, and HDL cholesterol and triglycerides levels following the supplementation of carnitine in hypercholesterolemic people with T2DM. The different findings might be explained by kind of chromium used, baseline circulating levels of chromium and carnitine, different study designs as well as different participants of the study. Carnitine facilitates transportation of activated long-chain fatty acids from the cytosol to mitochondria. It also changes the triglycerides synthesis and esterification toward the formation of acetylcarnitines, which in turn decrease the plasma concentration of triglycerides and VLDL cholesterol values [43].

In the present study, we found that LDL-R expression increased in response to the intervention. There are few studies investigating the effects of chromium and carnitine on gene

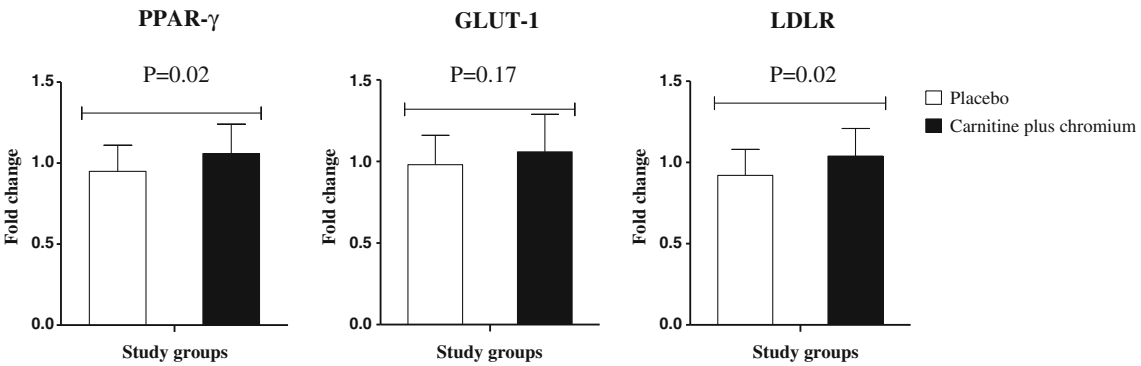

Fig. 2 Fold change (means ± SDs) in gene expression levels of PPAR-γ, GLUT-1, and LDLR in women with polycystic ovary syndrome receiving chromium plus carnitine supplements and placebo. *P* value was obtained from two-way ANOVA for repeated measures. $N = 27$ in each group. GLUT-1, glucose transporter 1; LDLR, low-density lipoprotein receptor; PPAR-γ, peroxisome proliferator-activated receptor gamma; PCOS, polycystic ovary syndrome

expression related to lipid metabolism. Lee et al. [44] reported that adding chromium picolinate to intravenous glucose infusion in Korean native steers improved lipid metabolism and gene expression of fatty acid synthase and stearoyl-CoA desaturase-1. In addition, a 10-week hypocaloric high-protein diet and carnitine supplementation increased expression of LDLR and lipoprotein lipase mRNA [45]. Some animal studies investigating combined chromium and carnitine found an interactive effect on several lipid parameters. Wang et al. [20] found a significant interactive effect between chromium and L-carnitine on total, HDL cholesterol, triglycerides and free fatty acids in chicken. Another animal investigation reported that chromium picolinate in combination with carnitine reduced non-esterified fatty acids concentrations which could reflect improved fatty acids utilization [46]. Although, the authors observed that combined supplementation had no significant effect on triglyceride levels [46].

Conclusions

Overall, chromium and carnitine co-supplementation for 12 weeks to overweight women with PCOS had beneficial effects on body weight, glycemic control, lipid profiles except HDL cholesterol levels, and gene expression of PPAR-γ and LDLR. This suggests consumption of chromium plus carnitine supplements may confer advantageous therapeutic potential for overweight women with PCOS. Further studies are needed in other patients and with longer periods to determine the beneficial effects of chromium and carnitine co-supplementation.

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Authors' Contributions ZA contributed in conception, design, statistical analysis, and drafting of the manuscript. MJ, FF, EK, EA, MK, EA, and AM contributed in data collection and manuscript drafting. All authors approved the final version for submission. ZA supervised the study.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

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