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Vitamin D supplementation decreased body weight and body mass index of Iranian type-2 diabetic patients: A randomised clinical trial study

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ABSTRACT

Introduction: Vitamin D as a common deficient micronutrient possibly plays an important role in body weight management. The aim of this study was to assess possible effects of vitamin D supplementation on anthropometric parameters of type-2 diabetic patients. **Methods:** Participants of this randomised controlled trial were 28 type-2 diabetic patients who received 4000 IU/day vitamin D and 30 patients who received placebo for two months. All patients were selected from the Iranian Diabetes Association (IDA), Tehran, Iran. Weight, height, body mass index, waist circumference, hip circumference and waist to hip ratio (WHR) were determined before and after the intervention. Dietary information was obtained using a 3-day food record. **Results:** Results showed a significant decrease in bodyweight (from 75.73±3.09 kg to 74.63±3.04 kg, $p = 0.002$), BMI (from 27.94±0.92 kg/m^2 to 27.544±0.90 kg/m^2, $p = 0.001$); waist circumference (from 92.56±2.33 cm to 91.05±2.27 cm, $p = 0.004$); and hip circumference (from 104.19±1.88 cm to 102.35±1.88 cm, $p = 0.029$) in the vitamin D group. Food record analysis showed that the percent of total calorie intake from dietary carbohydrates increased (from 50.40±1.38% to 53.14±1.53%, $p = 0.023$) and from fat, it decreased (from 38.43±1.30% to 35.22±1.49%, $p = 0.011$) significantly in the vitamin D group at the end of the intervention. **Conclusion:** Supplementation with vitamin D seems to include beneficial effects on bodyweight management in type-2 diabetic patients. However, the percentage of total calorie intake from each macronutrient should be considered.

Keywords: Vitamin D, type-2 diabetes, weight, BMI

INTRODUCTION

Studies have shown that overweight and obesity are the major causes of chronic disorders such as type-2 diabetes, cardiovascular diseases, cancers and other health treating diseases that could result in further morbidity and mortality (Guh *et al.*, 2009). Moreover, studies have demonstrated that serum level of vitamin D decreases in type-2 diabetic patients (Shankar, Sabanayagam & Khalidindi, 2015). Vitamin D plays an important role in glucose homeostasis via regulation of insulin secretion from β-cells (Zeitz *et al.*, 2003). Therefore, vitamin D deficiency is

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possibly associated with impaired insulin secretion and glucose control in diabetic patients. It has been reported that serum 25(OH)D3 concentration, the best indicator of body vitamin D status, has an inverse relationship with bodyweight and the risk of obesity decreases in people with a high concentration of serum 25(OH)D3 (Arunabh *et al.*, 2003).

The presence of vitamin D receptor (VDR) in adipose tissues may suggest that this vitamin possibly plays a role in the control of fat metabolism and is linked to bodyweight management (Sun & Zemel, 2008). A recent meta-analysis has shown that low 25(OH)D3 concentration is independently linked to abdominal obesity and hyper-glycemia (Pittas *et al.*, 2007). One study showed that low circulating levels of calcidiol could predispose individuals to fat accumulation (Grineva *et al.*, 2013), while in another clinical trial, supplementation with calcium and vitamin D did not significantly affect weight of obese women (Holecki *et al.*, 2008). Recently, Kimiagar *et al.* (2010) reported a high rate of vitamin D deficiency in several cities of Iran. However, the effect of vitamin D supplementation on bodyweight and BMI is still conflicting and not clearly explained. Due to the role of vitamin D in insulin function and its possible role in control of bodyweight and due to the widespread rate of vitamin D deficiency in Iran, the current study was carried out to assess the potential effects of vitamin D supplementation on bodyweight loss in type-2 diabetic patients.

METHODS

The participants of this double-blind placebo-controlled randomised clinical trial (RCT) study consisted of 65 type-2 diabetic patients aged 30 to 60 years selected from the Iranian Diabetes Association (IDA), Tehran, Iran. All participants completed an informed consent form. This study was approved by Tehran University of Medical Sciences Ethical Committee (ID: 17112) and registered in *www.clinicaltrial.org* (Reg. No. NCT01876563). Seven of these participants were excluded from the study because they did not consume all supplements; this study was therefore completed with 58 participants (36 women and 22 men). The exclusion criteria included consumption of any supplements having vitamin D within 3 months before the beginning of the study and occurrence of diabetes complications, thyroid disorders and use of insulin, thiazolidindiones or any drugs for treatment of obesity. The antidiabetic drugs used by the participants included metformin and/or glibenclamide. All participants agreed to continue their usual physical activities and not to change their diets during the intervention. Participants of this study were divided randomly into two groups of vitamin D and placebo using random permuted blocks. The vitamin D group received UL level of vitamin D (100 µg/4000 IU) daily and the placebo group received one tablet of the placebo drug daily for two months. Both placebo made from starch and vitamin D were obtained from Minoo Pharmaceutica, Cosmetic and Hygienic. Dietary information was collected in the beginning of intervention and after two months using a 3-day food record and was analysed using Nutritionist 4 Software for calculating the energy, macro-nutrient and micro-nutrient intakes. Blood samples were collected after 12–14 h overnight fasting at the beginning of the study and after two months of supplementation. Sera were separated from the whole blood and stored at -80°C for assessing biochemical parameters.

Height and weight of the participants were measured by a stadiometer (SECA, Germany) and SECA digital scale, respectively. Patients' height and weight

were recorded to the nearest centimetre and kilogram respectively and BMI calculated using "weight divided by the square of height" formula. Waist and hip circumferences were measured at the narrowest part of the torso and in a horizontal plane at the level of the maximal extension of the buttocks, respectively.

Statistical analysis was carried out using SPSS V.18 Software. Data were shown as mean ±SE (standard error). The normality of variables was checked using Kolmogrov-Smirrnof test. For non-normal variables, Wilcoxon test and Mann-Whitney test were used to analyse variables within and between the study groups. Independent sample *t*-test and paired *t*-test were used for the comparison of variables between the study groups before and after the supplementation and within the study groups, respectively. *p* values of ≤ 0.05 were considered statistically significant.

RESULTS

No statistical differences were seen between the two study groups in sex distribution, mean age, disease duration and time of sun exposure at the beginning of the intervention ($p = 0.154$, $p = 0.924$, $p = 0.877$ and $p = 0.580$, respectively). The anthropometric characteristics of the study groups at the beginning of the study and post-intervention are shown in Table 1. As shown in the table, all anthropometric parameters (except WHR) decreased significantly in the vitamin D group.

Table 1. Baseline and post-interventional anthropometric characteristics of study groups

Treatment group		*Vitamin D group (n = 28)*	*Placebo group group (n = 30)*	*p value**
Weight (kg)	Baseline	75.73±3.09	82.32±0.29	0.125
	Post-intervention	74.63±3.04	82.16±2.86	0.076
	Difference	-1.1±0.311	-0.15±2.90	0.035†
	p value**	0.002	0.598	
BMI (kg/m2)	Baseline	27.94±0.92	28.75±0.95	0.541
	Post-intervention	27.544±0.90	28.69±0.92	0.375
	Difference	-0.40±0.11	-0.06±0.10	0.032†
	p value**	0.001	0.557	
Waist circumference (cm)	Baseline	92.56±2.33	96.53±2.23	0.223
	Post-intervention	91.05±2.27	96.47±2.26	0.097
	Difference	-1.51±0.48	-0.05±0.50	0.037†
	p value**	0.004	0.914	
Hip circumference (cm)	Baseline	104.19 ±1.88	106.40 ±1.47	0.356
	Post-intervention	102.35 ±1.88	105.46 ±1.40	0.186
	Difference	-1.84 ±0.80w	-0.93 ±0.43	0.320†
	p value**	0.029	0.036	
WHR	Baseline	0.89±0.014	0.90±0.012	0.348
	Post-intervention	0.89±0.013	0.91±0.014	0.211
	Difference	0.001±0.005	0.008±0.006	0.440†
	p value**	0.841	0.208	

Data are expressed as mean ±SE; *Student *t*-test; **paired *t*-test; †adjusted for total calorie percent from dietary fat and carbohydrate.

Dietary intake and biochemical parameters of the study groups are shown in Table 2 and Table 3, respectively. No significant differences were observed between the two groups in energy, carbohydrate and protein intakes at the beginning and end of the intervention. Although we emphasised that all participants maintain their usual dietary habits during intervention, the mean intakes of dietary carbohydrates and fat and also the percent of total calorie from these nutrients were significantly increased and decreased, respectively in vitamin D group at the end of the intervention. There was no correlation between any anthropometric parameters and dietary intakes of energy, fat, carbohydrate and protein at the beginning of the study and after the 2-month intervention. No significant differences were seen in dietary vitamin D intake between the two groups at the beginning and end of the intervention (data not shown).

DISCUSSION

In general, the results of the current study have revealed that vitamin D supplementation can decrease bodyweight and BMI in diabetic patients. Consistent with our results, Nikooyeh *et al.* (2011) have shown that vitamin D supplementation alone or in combination with calcium could result in a significant decrease in weight, BMI and WC of type-2 diabetic patients. In another study, Rosenblum *et al.* (2012) has shown that vitamin D supplementation can decrease visceral adipose tissues significantly in obese people. In contrast, Mason *et al.* (2014) reported no beneficial effects of vitamin D supplementation on weight reduction in overweight or obese patients. Another study showed that supplementation with 7000 IU/day vitamin D for 26 weeks did not change significantly body fat, percutaneous fat and visceral fat in obese adults (Wamberg *et al.*, 2013). Obesity can decrease bioavailability of vitamin D by trapping it in adipose tissues. In fact, accumulation of vitamin D in adipose tissues can decrease access of the human body to the vitamin for converting it to 25(OH) D3 and the subsequent formation of calcitriol (Heaney *et al.*, 2009).

A possible mechanism for the effects of vitamin D on lowering bodyweight is the suppressing effect of vitamin D on PTH hormone which can promote fat accumulation in adipose tissues by increasing the intracellular level of calcium (Zemel *et al.*, 2000).

Studies have shown that the hormonal form of vitamin D can suppress adipocyte differentiation in pre-adipocytes which can increase the adipogenesis in the absence of VDR (Blumberg *et al.*, 2006) and induce apoptosis in mature 3T3-L1 adipocytes through ca2+-dependent apoptotic proteases, caspase 12 and calpain (Sergeev, 2012).

The VDR can mediate the actions of hormonal form of vitamin D in some body organs including adipose tissue, independent from its classical role in calcium homeostasis (Nagpal Na & Rathnachalam, 2005). Previously, it was revealed that un-coupling proteins such as UCP-1 and UCP-3 were up-regulated in brown adipose tissue of VDR (–/–) mice regardless of their dietary condition (Enerback et al., 1997) and an increase in the gene expression of UCP-1 in white adipose tissue could reduce fat stores in transgenic mice (Kopecky *et al.*, 1995). Experimental studies have shown that energy expenditure, fatty-acid β-oxidation and uncoupling protein (UCP) levels are higher in VDR-deficient mice, in comparison with wild-type counterparts (Narvaez *et al.*, 2009). However, a cross-sectional study has revealed that the rate of REE/ kg of bodyweight is significantly lower

Table 2. Baseline and post-interventional dietary intakes in the study groups

Treatment group		*Vitamin D group (n = 28)*	*Placebo group (n = 30)*	*p value**
Energy	Baseline	2234±69.6	2129±71.9	0.296
	Post-intervention	2225±64.3	2196±68	0.242
	Difference	-10.53±46.21	66.71±54.00	0.535
	p value**	0.637	0.228	
CHO (g/day)	Baseline	270.76±13.42	284.1±13.40	0.488
	Post-intervention	293.33±11.89	276.2±11.0	0.296
	Difference	22.57±10.42	-7.82±8.87	0.032
	p value**	0.039	0.385	
Pro (g/day)	Baseline	72.40±3.85	68.77±3.09	0.469
	Post-intervention	72.44±3.67	68.80±2.65	0.432
	Difference	0.04±3.86	0.04±2.83	0.99
	p value**	0.991	0.990	
Fat (g/day)	Baseline	94.36±4.94	98.41±3.92	0.373
	Post-intervention	83.42±4.66	100.63±3.57	0.011
	Difference	10.94±4.11	2.21±4.13	0.140
	p value**	0.013	0.60	
CHO (%)	Baseline	50.40±1.38	48.21±1.44	0.322
	Post-intervention	53.14±1.53	49.96±1.13	0.104
	Difference	2.74±1.1	1.75±1.49	0.421
	p value**	0.023	0.250	
Pro (%)	Baseline	13.12±0.59	12.91±0.54	0.793
	Post-intervention	12.67±0.49	12.35±0.50	0.654
	Difference	-0.44±0.48	-0.55±0.53	0.882
	p value**	0.362	0.304	
Fat (%)	Baseline	38.43±1.30	39.95±1.35	0.703
	Post-intervention	35.22±1.49	39.15±1.35	0.022
	Difference	-3.21±1.17	-0.8±1.40	0.103
	p value**	0.011	0.571	

Data are expressed as mean ±SE; *Student *t*-test; **paired *t*-test.

in women with vitamin D deficiency, compared to that in women having sufficient levels of vitamin D (Hossein-Nezhad *et al.*, 2013).

In obesity, the volume of adipocytes increases and the cells can secrete significant levels of pro-inflammatory cytokines such as TNF-α and IL-6 as well as IL-1β, which can result in insulin resistance in several organs including liver and skeletal muscles though inhibition of insulin receptor signaling (Hotamisligil, 2006). Effects of nutrients on serum insulin as well as insulin resistance have been shown in previous studies (Rad *et al.*, 2014; Saboori *et al.*, 2016). Vitamin D can regulate glucose-mediated insulin secretion from β-cells and enhance uptake of glucose by skeletal muscles and adipose tissues through glucose transporters and hence is able to improve glycemic control in

Table 3. Fasting biochemical characteristics of study groups at baseline and post-intervention

Treatment group		*Vitamin D group (n = 28)*	*Placebo group (n = 30)*	*p value**
FBS (mg/dl)	Baseline	147.07±10.11	151.23±7.48	0.740
	Post-intervention	147.74±10.16	161.27±7.69	0.288
	Difference	2.70±9.66	10.03±4.61	0.483
	p value**	0.782	0.038	
TG (mg/dl)	Baseline	158.25±12.41	167.43±16.10	0.656
	Post-intervention	145.33±10.28	178.20±14.80	0.080
	Difference	-13.07±13.15	10.76±14.45	0.231
	p value**	0.329	0.462	
TC (mg/dl)	Baseline	201.82±7.91	184.53±6.73	0.100
	Post-intervention	189±7.04	200.87±8.70	0.301
	Difference	-12.88±7.25	16.33±6.93	0.005
	p value**	0.087	0.025	
HDL-C (mg/dl)	Baseline	42.29±1.84	41.17±2.15	0.697
	Post-intervention	49.63±3.28	49±3.03	0.888
	Difference	6.81±3.25	7.83±3.39	0.830
	p value**	0.046	0.028	
LDL-C (mg/dl)	Baseline	88.93±7.23	97.37±7.64	0.427
	Post-intervention	88.37±6.94	98.67±7.22	0.311
	Difference	0.89±7.19	1.30±8.65	0.971
	p value**	0.903	0.882	
HbA1c (%)	Baseline	7.29±0.22	7.84±0.28	0.132
	Post-intervention	6.76±0.18	7.73±0.23	0.002
	Difference	-0.53±0.08	-0.11±0.08	0.001
	p value**	<0.001	0.176	
Insulin (μIU/mL)	Baseline	8.24±0.97	7.49±0.58	0.505
	Post-intervention	6.55±0.28	7.96±0.94	0.171
	Difference	-1.68±0.81	0.47±0.51	0.027
	p value**	0.048	0.367	
MOMA-IR	Baseline	2.50±0.19	2.55±0.16	0.841
	Post-intervention	2.38±0.18	2.78±0.19	0.134
	Difference	-0.14±0.14	0.22±0.13	0.056
	p value**	0.307	0.092	
Calcidiol (ng/ml)	Baseline	15.55±1.91	14.64±2.22	0.759
	Post-intervention	27.50±2.04	15.95±2.20	<0.001
	Difference	11.95±1.44	1.92±0.89	<0.001
	p value**	<0.001	0.040	

Data are expressed as mean ±SE; *Student *t*-test; **paired *t*-test.

obese people (Teegarden & Donkin, 2009).

Results of the present study demonstrate that although the level of energy intake did not change significantly between the study groups, the vitamin D group experienced significant decreases in bodyweight, BMI and WC at the end of the intervention. A possible explanation is that the intake of macronutrients has changed significantly in this group during the study. Although all participants of this study were requested not to change their usual diet during the intervention, the intake of carbohydrates and fat and the percent of total calorie resulting from these two nutrients changed significantly in participants receiving vitamin D at the end of the study. High carbohydrate diets may improve body energy regulation through altering gut microbial composition (Fava *et al.*, 2013). Some studies have revealed that dietary changes for the reduction of diet fat content from 40 to 25–30% of the total calorie can result in 2–4 kg weight loss in people (Bray & Popkin, 1998). Although cross-sectional studies have shown a close relationship between the dietary intakes of carbohydrates and fat and the body fat status (Astrup *et al.*, 1997), longitudinal studies are not able to show the relationship between the reported macronutrient intakes and subsequent weight changes (Kant *et al.*, 1995). It should be noted that the extent of decrease in anthropometric parameters was not clinically significant in the current study. The best explanation probably is that obesity is a consequence of an imbalance between energy intake and its expenditure and as mentioned earlier, the amount of energy intake did not change significantly in our study groups.

One limitation of this study was the short duration of vitamin D supplementation; if the patients had consumed supplements for a longer time, the extent of reduction in anthropometric characteristics could have been clinically significant. Another limitation of this study was the changes in macronutrient distribution in dietary intake of participants, although the energy intake did not change significantly during the intervention.

CONCLUSION

Results from the current study have shown that vitamin D supplementation can significantly decrease anthropometric characteristics of type-2 diabetic patients, although their physical activity level and average energy intake did not change significantly during intervention.

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Authors' contributions

Mahmoud D designed the study; Esmaeil YR and Somayeh S performed the study in the field under the supervision of Mahmoud D; Ebrahim F performed data analysis and interpretation; Esmaeil YR and Somayeh S drafted the manuscript; Ebrahim F and Mahmoud D revised the article for important intellectual content.

Conflict of interest

The authors declare that there is no conflict of interest.

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