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The Effect of Flaxseed Enriched Yogurt on the Glycemic Status and Cardiovascular Risk Factors in Patients with Type 2 Diabetes Mellitus: Randomized, Open-labeled, Controlled Study

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ABSTRACT

Flaxseed is one of the rich sources of α-linolenic acid and lignan. Flaxseed and its components have antioxidant, hypolipidemic and hypoglycemic effects. The study aimed to investigate the effect of flaxseed enriched yogurt on glycemic control, lipid profiles and blood pressure in patients with type 2 diabetes. A randomized, open-labeled, controlled clinical trial was conducted on 57 patients with type 2 diabetes. Participants were assigned to receive 200 g 2.5% fat yogurt containing 30-g flaxseed or plain yogurt daily for 8 weeks. Anthropometrics and biochemical parameters were evaluated at the beginning and end of the study. After 8 weeks of supplementation, Hemoglobin A1c was significantly decreased in the intervention group compared to control ($p = 0.007$). Also, at the end of the study, significant differences were seen between the flaxseed enriched yogurt and control groups in triglycerides and total cholesterol concentrations ($p = 0.04$ and $p = 0.01$), systolic blood pressure and diastolic blood pressure ($p = 0.02$ and $p = 0.002$, respectively). However, we did not find any difference between 2 groups in low-density lipoprotein, high-density lipoprotein, body weight and waist circumference ($p > 0.05$). Our results showed that the addition of flaxseed to yogurt can be effective in the management of type 2 diabetes.

Trial Registration: ClinicalTrials.gov Identifier: NCT02436369

Keywords: Type 2 diabetes; Flaxseed; Yogurt; Blood pressure

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Conflict of Interest

The authors declare that they have no competing interests.

INTRODUCTION

According to the International Diabetes Federation statistics, presently every seven seconds someone is estimated to die from diabetes or its complications, with 50% of those deaths (4 million in total per year) occurring under the age of 60 years [1]. The prevalence of type 2 diabetes is increasing globally, especially in developing countries [2]. In Iran, 11.4% of adults have been estimated to suffer from type 2 diabetes [3]. Type 2 diabetes is related to various clinical features, including central obesity, hyperglycemia, dyslipidemia, low-density lipoproteins (LDL) oxidation, inflammation, hypertension, and the prothrombotic state [4]. Diabetic patients have a high risk of developing the cardiovascular disease due to insulin resistance and dyslipidemia [5]. Dyslipidemia is the most important modifiable risk factor for atherosclerosis in diabetic patients. Glycemic control and reduced insulin resistance can moderate diabetes-related dyslipidemia and improve the endothelial function [6,7]. Fat accumulation, especially abdominal obesity and visceral fat play an important role in insulin resistance and pathogenesis of various metabolic diseases related to type 2 diabetes [8]. A recent meta-analysis study showed that AIP was a direct correlation with lipid profiles in patients with type 2 diabetes [9].

Recently, flaxseed (*Linum usitatissimum*) has received substantial attention for the potential health benefits about many metabolic disorders such as diabetes. Flaxseed is a rich source of plant omega-3 fatty acid α-linolenic acid (ALA), soluble fibers, and lignan phytoestrogen. Most beneficial effects of flaxseed are due to the high contents of omega-3 fatty acids. Several studies reported flaxseed antioxidant activity against various diseases, including diabetes, atherosclerosis, and hypertension, in addition to anti-inflammatory and anticarcinogenic effects [10-13].

Dairy products are rich in important nutrients such as vitamins (A, D [in fortified products], riboflavin, B_{12} and Phylloquinone), minerals (calcium, magnesium, and potassium), and high-quality protein. On the other hand, dairy products are significant sources of saturated fats, and evidence indicates that high intake of saturated fat is linked to an increased risk of cardiovascular disease, mainly by increasing blood LDL cholesterol [14]. It is a notable fact that yogurt is commonly consumed by Iranian people [15]; so, if flaxseed enriched yogurt can improve diabetes management, it may be suggested as a potential agent in medical nutrition therapy for patients with type 2 diabetes.

Since some studies have found a decreased risk of type 2 diabetes associated with higher intake of dairy products, but other studies suggested no association [16]; therefore, this study aimed to evaluate the possible effect of flaxseed enriched yogurt on clinical manifestation of diabetes. It was assumed that flaxseed can modify the relation between dairy products and type 2 diabetes and participants in the intervention group, compared to control group, would show an improvement in their glycemic status and anthropometric indices.

METHODS

Trial design

This trial was registered at ClinicalTrials.gov as NCT02436369. The study was a randomized, double-blind, parallel controlled trial. The protocol was approved by the Institutional Review Board of the Urmia University of Medical Sciences (No. 92-01-32-1125). Written informed consent was obtained from participants.

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Participants

The study was conducted between August 2015 and November 2017. Subjects were recruited from patients registered at Urmia Diabetes Association. Eligible subjects had been newly diagnosed within the past 3 months according to the diagnostic criteria of type 2 diabetes [17]. The inclusion criteria were body mass index (BMI) within the range of 20 ≤ BMI ≤ 35 kg/m^2; treated with oral hypoglycemic agents (Metformin or Glibenclamide); no insulin injection; fasting blood glucose ≥ 126 (but less than 400 mg/dL) or 2 hours blood sugar ≤ 200 mg/dL before taking blood lowering drugs; no pregnancy and lactation; not taking omega-3, antioxidants and fiber supplements and phytoestrogens within past 3 months. Exclusion criteria included the history of gastrointestinal disease, history of allergy or high consumption (greater than one serving per day) of nuts, flaxseed, or sesame seeds; food allergies or intolerances; malignancies; renal failure; liver, endocrine, or inflammatory disorders; smoking; and using alcohol.

Interventions

The eligible subjects were randomly assigned into 2 groups (intervention and control). Subjects in the intervention group were provided with 200 g 2.5% fat yogurt containing 30-g flaxseed, and the control group received 200 g/day plain yogurt per day for 8 weeks. Nutrition facts in serving size (1 cup) of 2.5% fat yogurt including: calories = 154 kcal, total fat = 3.6 g, saturated fatty acid= 2.3 g, carbohydrate = 17.25 g and protein = 12.86 g. The participants were asked to maintain their usual dietary and exercise patterns.

Measurement of anthropometric parameters and blood pressure (BP)

Anthropometric indices and blood pressure were evaluated at the beginning and end of the intervention. Height was measured without shoes to the nearest 0.5 cm, and weight was measured without shoes and with light clothing using Seca scale (Seca, Hamburg, Germany). BMI was also calculated by dividing each participant's weight in kg by his or her height in m^2. Waist circumference (WC) was measured by a tape measure, while the patients were at the end of breathing out, at the midpoint of lower rib and iliac crest. All the measurements were taken by the same person to decrease the error rate. A standardized mercury sphygmomanometer 160 was used to measure resting BP in a quiet room by well-trained physicians on 2 occasions, before and after sitting for a 15 minutes period, using a manual BP cuff; the mean BP reading was used for the present analysis [18].

Assessment of dietary intake and physical activity

Dietary intake was assessed at the beginning and end of the study by a 3-day, 24-hour recall questionnaire. Food scales and models were also used to enhance portion size. Dietary intakes were analyzed by Nutritionist IV software (version 3.5.2; The Hearst Corporation, San Bruno, CA, USA). Physical activity was also assessed using the metabolic equivalent of task (MET) questionnaire [19] at weeks 0 and 8.

Measurement of biochemical parameters

A blood sample was taken at the beginning and end of the study after 12 hours fasting to measure biochemical parameters and serum lipid profile, fasting blood sugar levels, and hemoglobin A1c (HbA1c) levels were determined. Fasting plasma glucose concentration and HbA1c were measured by an enzymatic colorimetric method with commercial kits from Pars Azmun Inc. (Tehran, Iran). Serum triacylglycerol (TAG), high-density lipoprotein (HDL), and total cholesterol (TC) levels were determined using a photometric assay (Reckon, New South Wales, Australia), while LDL cholesterol levels were determined using the following equation: LDL = TC − HDL − 0·16(TAG).

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Statistical analyses

All data were expressed as mean ± standard deviation. Kolmogorov–Smirnov test was used to determine the normality of studied variables. Independent sample t-test was used for comparing the differences in variables between the 2 groups. Within-group, analyses were conducted using paired samples t-test based on a change from baseline. To eliminate the effects of confounding factors, the analysis of covariance (ANCOVA) test was used. All ANCOVA models were adjusted for age, sex, and calorie intake. The $p < 0.05$ was considered significant.

RESULTS

Characteristics of the participants

Seventy participants were randomly assigned to the intervention and control group. At the end of the study, 28 subjects in the control group and 29 patients in the intervention group entered the final analysis. Patient screening, enrollment, and retention by treatment group are shown in **Figure 1**. The baseline characteristics of study participants are shown in **Table 1**. Comparisons showed that subjects were not significantly different regarding age, sex, and physical activity levels. Statistical analysis of energy, macronutrients, and dietary intake of polyunsaturated fatty acid, monounsaturated fatty acid, and saturated fatty acid showed no significant differences between two groups and within each group at the beginning and end of the study (**Table 2**). The anthropometric measurements of the participants before and after intervention are shown in **Table 3**. At the end of the study, WC declined significantly more in the intervention group compared to the control group ($p < 0.001$). However, the significance did not remain after adjusting the results for age, sex, and calorie intake ($p = 0.2$) (**Table 4**). Changes in weight between the 2 groups were not significant ($p = 0.8$).

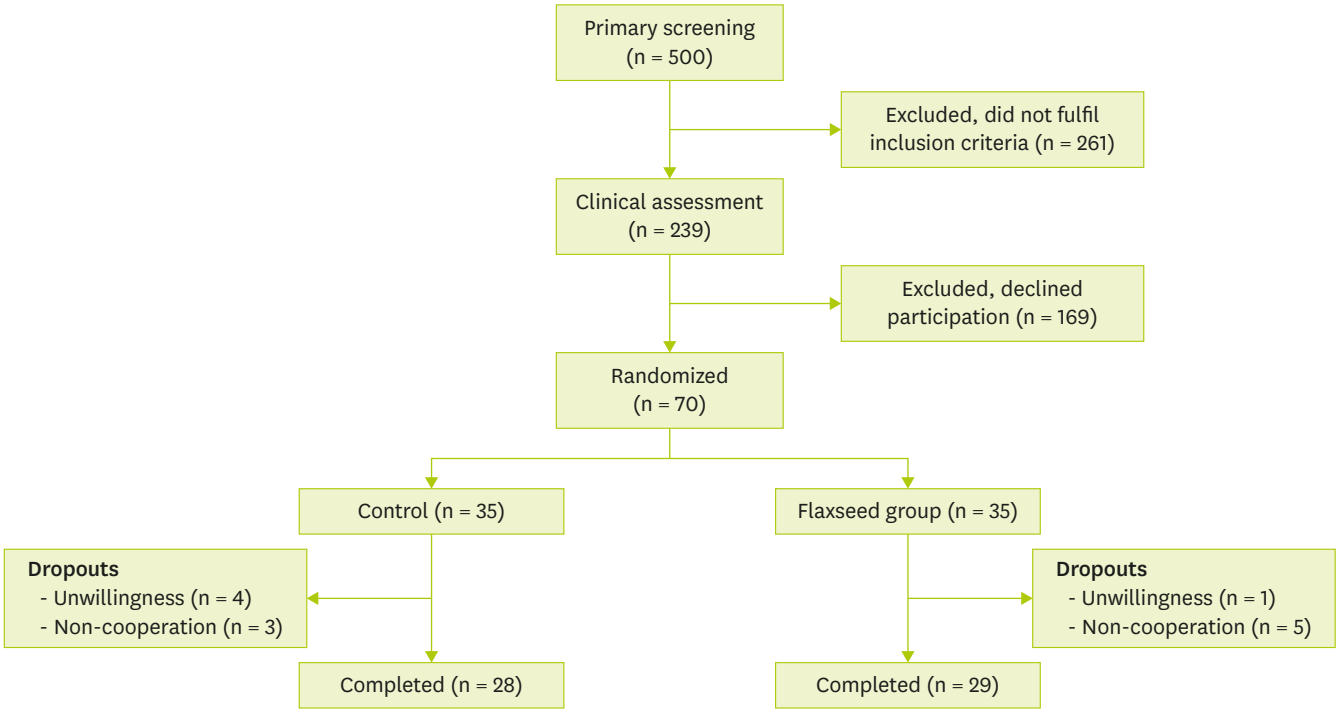

Figure 1. Flow chart of participant's enrollment in the study.

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Table 1. The characteristics of the study participants

Variables	Groups		p value
	Intervention (n = 29)	Control (n = 28)	
Age (yr)	54.18 ± 5.41	52.59 ± 6.01	0.30
Height (cm)	167.22 ± 12.22	163.34 ± 9.23	0.26
Weight (kg)	75.17 ± 14.33	77.25 ± 10.21	0.53
Sex			0.54
Male	10 (34)	11 (40)	
Female	19 (66)	18 (60)	
Physical activity level (before)			0.99
Light	13 (44.8)	13 (46.4)	
Moderate	12 (41.4)	11 (39.3)	
Severe	4 (13.8)	4 (14.3)	
Physical activity level (after)			0.97
Light	11 (39.3)	11 (37.9)	
Moderate	12 (42.9)	13 (44.8)	
Severe	5 (17.9)	5 (17.2)	

Values are means ± standard deviation or number (%).

Table 2. Dietary intakes of subjects at baseline and post intervention

Variables	Groups		p value
	Intervention (n = 29)	Control (n = 28)	
Energy (kcal)			
Before	2,244.75 ± 262.88	2,142.70 ± 155.54	0.08
After	2,221.93 ± 241.08	2,128.87 ± 186.71	0.11
p value	0.05	0.57	
Carbohydrate (g)			
Before	275.32 ± 71.06	246.41 ± 44.82	0.07
After	209.63 ± 67.85	235.94 ± 45.83	0.09
p value	< 0.001	0.10	
Protein (g)			
Before	65.93 ± 12.18	62.94 ± 8.75	0.29
After	68.91 ± 14.91	65.05 ± 12.93	0.30
p value	0.17	0.07	
SFA (g)			
Before	14.58 ± 3.13	14.24 ± 2.93	0.67
After	15.97 ± 3.51	15.79 ± 3.68	0.85
p value	0.06	0.05	
MUFA (g)			
Before	13.45 ± 0.57	13.08 ± 0.43	0.49
After	13.8 ± 0.59	13.30 ± 0.88	0.52
p value	0.66	0.83	
PUFA (g)			
Before	12.98 ± 3.17	12.76 ± 3.64	0.81
After	14.24 ± 2.93	12.40 ± 4.26	0.06
p value	0.006	0.69	

Values are means ± standard deviation.
SFA, saturated fatty acid; MUFA, monounsaturated fatty acid; PUFA, polyunsaturated fatty acid.

Table 3. Anthropometric measurements, FBS, HbA1c, lipid profile, and BP of the patients at the beginning and at the end of the study

Variables	Groups		p value*
	Intervention (n = 29)	Control (n = 28)	
Weight (kg)			
Before	75.17 ± 14.33	77.25 ± 10.21	0.53
After	74.34 ± 13.77	76.36 ± 8.69	0.51
Change	−0.83 ± 1.54	−0.89 ± 2.49	0.91
p value†	0.008	0.06	
WC (cm)			
Before	91.00 ± 10.37	92.35 ± 8.26	0.59
After	87.18 ± 10.21	91.24 ± 6.45	0.08

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Table 3. (Continued) Anthropometric measurements, FBS, HbA1c, lipid profile, and BP of the patients at the beginning and at the end of the study

Variables	Groups		p value*
	Intervention (n = 29)	Control (n = 28)	
Change	−4.14 ± 2.20	−1.28 ± 3.24	< 0.001
p value	< 0.001	0.04	
BMI (kg/m^2)			
Before	29.32 ± 3.42	29.54 ± 3.94	0.83
After	28.92 ± 3.30	28.99 ± 3.53	0.88
Change	−0.40 ± 0.52	−0.55 ± 1.66	0.30
p value	< 0.001	0.02	
WHR			
Before	0.57 ± 0.05	0.57 ± 0.06	0.95
After	0.55 ± 0.05	0.56 ± 0.06	0.009
Change	−0.02 ± 0.013	−0.01 ± 0.016	0.009
p value	< 0.001	< 0.001	
FBS (mg/dL)			
Before	114.14 ± 33.63	139.38 ± 50.14	0.03
After	118.36 ± 26.72	154.11 ± 52.55	0.004
Change	4.21 ± 26.03	25.27 ± 36.66	0.02
p value	0.39	0.002	
HbA1c (%)			
Before	6.4 ± 1.1	7.32 ± 1.59	0.01
After	6.10 ± 1.02	7.17 ± 1.59	0.005
Change	−0.3 ± 0.36	−0.15 ± 1.01	0.47
p value	< 0.001	0.44	
SBP (mmHg)			
Before	12.32 ± 1.28	12.86 ± 1.06	0.09
After	11.86 ± 1.25	12.76 ± 1.23	0.008
Change	−0.46 ± 1.11	−0.10 ± 1.11	0.23
p value	0.04	0.62	
DBP (mmHg)			
Before	7.32 ± 0.99	7.79 ± 0.9	0.03
After	7.23 ± 0.90	7.98 ± 0.74	0.004
Change	−0.09 ± 0.94	0.19 ± 0.78	0.66
p value	0.62	0.20	
TC (mg/dL)			
Before	166.96 ± 31.81	164.62 ± 37.61	0.96
After	164.21 ± 38.90	177.41 ± 38.40	0.065
Change	−2.75 ± 17.02	12.79 ± 26.39	0.042
p value	0.40	0.07	
TG (mg/dL)			
Before	145.00 ± 68.4	156.52 ± 72.47	0.43
After	134.78 ± 47.21	152.96 ± 51.10	0.17
Change	−10.22 ± 38.15	−3.56 ± 36.29	0.52
p value	0.83	0.48	
HDL (mg/dL)			
Before	47.37 ± 10.07	44.47 ± 9.2	0.25
After	48.99 ± 9.48	46.10 ± 8.79	0.24
Change	1.59 ± 3.13	1.81 ± 5.76	0.86
p value	0.01	0.11	
LDL (mg/dL)			
Before	78.91 ± 21.93	80.79 ± 23.18	0.75
After	82.69 ± 26.92	87.40 ± 26.73	0.51
Change	3.77 ± 11.05	6.61 ± 21.96	0.54
p value	0.08	0.12	

Values are presented as mean ± standard deviation.
FBS, fasting blood sugar; HbA1c, hemoglobin A1c; BP, blood pressure; WC, waist circumference; BMI, body mass index; WHR, waist to height ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; TG, triglycerides; HDL, high-density lipoprotein; LDL, low-density lipoprotein.
*Calculated by independent sample t-test; †calculated by paired t-test.

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Table 4. Results of analysis of covariance after adjusting for baseline values and dietary intakes

Variables	Groups	Model 1*		Model 2†	
		Mean ± SE	p value	Mean change ± SE	p value
Weight (kg)	Intervention	75.31 ± 0.33	0.80	−0.89 ± 0.41	0.32
	Control	75.43 ± 0.33		−0.87 ± 0.39	
WC (cm)	Intervention	87.45 ± 0.48	< 0.001	−4.18 ± 0.58	0.20
	Control	90.48 ± 0.48		−1.25 ± 0.55	
BMI (kg/m^2)	Intervention	29.01 ± 0.22	0.30	−0.41 ± 0.25	0.12
	Control	28.70 ± 0.21		−0.56 ± 0.24	
FBS (mg/dL)	Intervention	123.96 ± 5.93	0.007	5.51 ± 6.22	0.09
	Control	148.07 ± 6.16		25.42 ± 6.34	
HbA1c (%)	Intervention	6.47 ± 0.14	0.13	−0.29 ± 0.15	0.007
	Control	6.78 ± 0.14		−0.16 ± 0.15	
SBP (mmHg)	Intervention	12.02 ± 0.19	0.04	−0.39 ± 0.21	0.02
	Control	12.59 ± 0.19		−0.12 ± 0.21	
DBP (mmHg)	Intervention	7.45 ± 0.13	-	−0.14 ± 0.16	0.002
	Control	7.85 ± 0.13		0.21 ± 0.16	
TC (mg/dL)	Intervention	167.15 ± 4.20	0.39	−0.15 ± 4.05	0.04
	Control	172.23 ± 4.13		9.72 ± 3.90	
TG (mg/dL)	Intervention	138.82 ± 6.42	0.26	−12.50 ± 7.60	0.01
	Control	148.99 ± 6.31		5.75 ± 7.32	
HDL (mg/dL)	Intervention	47.70 ± 0.83	0.79	1.42 ± 0.92	0.087
	Control	47.39 ± 0.83		1.95 ± 0.90	
LDL (mg/dL)	Intervention	83.55 ± 3.31	0.51	1.65 ± 3.22	0.06
	Control	86.56 ± 3.25		8.03 ± 3.11	

SE, standard error; WC, waist circumference; BMI, body mass index; FBS, fasting blood sugar; HbA1c, hemoglobin A1c; SBP, systolic blood pressure; DBP, diastolic blood pressure; TC, total cholesterol; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein.
*Baseline value adjusted for post-intervention; †variable changes adjusted for age, sex, and calorie intake.

Primary outcome

Eighty-one percent of patients completed eight weeks of treatment. Results showed that fasting blood sugar (FBS) levels increased in both groups; however, the increase in the intervention group was significantly lower than the control group (p = 0.02). After adjustment for confounding factor, differences in serum FBS did not remain significant (p = 0.09).

Secondary outcomes

The mean reduction of HbA1c concentration was −0.29 ± 0.36 in the flaxseed group and −0.15 ± 1.01 in the control group, which was significant (p = 0.035). Even after adjustment for confounding factor, the difference remained significant (p = 0.007). At the end of the 8-week treatment period, a significant improvement in triglycerides (TGs) concentration was seen in the flaxseed group compared to control after the adjustment for age, sex, and calorie intake (p = 0.01). Also, adjusting model had shown that the consumption of flaxseed enriched yogurt significantly reduced TC rather than the control group (p = 04). However, the HDL level in the control group showed a significant increase compared to the intervention group (p = 0.003). Furthermore, the mean changes in the LDL and HDL cholesterol concentrations were not significant between the 2 groups (p = 0.06 and p = 0.087, respectively). In the adjusted model, systolic blood pressure (SBP) and diastolic blood pressure (DBP) in the flaxseed enriched yogurt were reduced significantly compared to the control group (p = 0.02 and p = 0.002, respectively). None of the patients completing the study had any serious adverse events, which indicated tolerance to the treatment.

DISCUSSION

To our knowledge, this is the first randomized, open-labelled clinical trial study that examined the effects of flaxseed enriched yogurt on glycemic status and cardiovascular risk

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factors in patients with type 2 diabetes mellitus. In this study, daily consumption of 200-g flaxseed enriched yogurt for 8 weeks resulted in a significant decrease in HbA1c, TG, and TC. Weight, WC, and BMI was not significantly decreased in the intervention group rather than control at the end of the study.

Previous animal studies have shown that flaxseed oil, which is a rich source of omege-3 fatty acids improve glycemic profile [20]. In the current study, the consumption of flaxseed enriched yogurt resulted in improved HbA1c. However, FBS did not change significantly. Ghazanfari et al. [21] showed that the HbA1c was a relatively strong predictor in diabetic subjects. Fasting blood glucose is affected by the patient's recent diet, but Hb1c is average blood glucose levels for the last 2 to 3 months [22]. The flaxseed component that may have positive effects on glycemic indices is the secoisolariciresinol diglucoside (SDG), the main flaxseed lignin, which has been reported to improve glycemic control [23]. Additionally, it has been found that SDG isolated from flaxseed is effective in retarding the development of diabetes in Zucker diabetic rats [24]. Also, several studies have shown that soluble and insoluble fiber in flaxseed improves glycemic profiles [25]. Yari et al. [26] have shown that flaxseed supplementation in patients with metabolic syndrome decreased insulin resistance and FBS level. Flaxseed consumption can reduce the speed of glucose absorption and the need for insulin production [27].

The findings of the current study showed that after adjusting for confounding variables, flaxseed enriched yogurt reduced TG and TC levels significantly compared to the control group. Our results confirmed the previous data [28], which showed that flaxseed powder consumption desirably reduced serum lipids. Similar to our results, a recent study reported that flaxseed intake is associated with a significant improvement in lipid profile [29]. Cornish et al. found only reduction in serum TG and not the cholesterol level of patients with metabolic syndrome who were supplemented with flaxseed lignin [30]. Flaxseed is a rich source of dietary fiber (28% by weight), and 25% of its fiber is soluble. Dietary soluble fibers have shown cholesterol-lowering effects [11,31]. Also, flaxseed is nature's most concentrated source of ALA, which can reduce the TG level [11]. Moreover, it has been proposed that increased bile acid synthesis is one of the important cholesterol-lowering mechanisms of flaxseed [32]. It has been reported that flaxseed SDG lignan may play an important role in improving the lipid profile [23]. We not found any significant difference between two groups in concentrations of HDL cholesterol. This can be due to the short duration of the intervention in our study.

In this trial, we showed significant effects of flaxseed enriched yogurt on BP. Similar to our findings, Paschos et al. observed a hypotensive effect of ALA-rich flaxseed oil (8 g/day) in the 12-week intervention [33]. The mechanisms for the antihypertensive effects of flaxseed are not completely clear, but the evidence proposes that ALA may exert most of the antihypertensive action of flaxseed. Circulating levels of ALA showed significant correlation with SBP and DBP [34]. ALA may reduce the activity of soluble epoxide hydrolase, the target enzyme for antihypertensive treatment; this enzyme produces oxylipins, which results in loss of vasodilation and promote inflammation [35]. Another component related to flaxseed effects on BP is vitamin E; flaxseed contains considerable amounts of vitamin E, primarily as γ-tocopherol. It has been stated that γ-tocopherol increases sodium excretion in the urine, which may help lower BP [36].

In the current study, both intervention and control group showed a non-significant reduction in the body weight and BMI. In the crude model, intervention group has shown a more

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reduction in the WC rather than the control group, but, it did not remain significant after adjusting the results for confounding factors, which was contradicted previous findings [37].

Previous studies reported that the flaxseed could induce satiety and reduce energy intake due to its high content of fiber [38]. Similarly, Kapoor et al. [39] suggested that fiber derived from flaxseed increases transit time and delays gastric emptying. In another study, flaxseed decreased the consumption of energy-rich foods in the menopausal female with diabetes. In addition, body weight was noticeably decreased after 12 months of flaxseed intake [40]. Also, it has been suggested that regular intake of flaxseed fiber in a low-energy beverage prior to each meal may aid to maintain or lose weight [38]. Another reason may be due to the impacts of isoflavones and lignans on energy metabolism; in fact, phytoestrogens have been shown to inhibit the activity of several enzymes involved in cell-signalling pathways and nuclear mechanisms such as cell proliferation and differentiation [41]. Mohammadi et al. [37] in a meta-analysis showed that using whole flaxseed in doses > 30 g/day, longer-term interventions (≥ 12 weeks) and studies including participants with higher BMI (≥ 27 kg/m^2) had positive effects on body composition. Therefore, it seems that in order to observe the effects of flaxseed on body composition, it is necessary to have a higher dose and longer duration of the intervention.

One limitation of our study was the open-labelled design because an inert placebo for flaxseed was not known [42]. Also, the duration of the intervention was also short.

The current study had several strengths including: i) designing as a controlled clinical trial, ii) combining the flaxseed with yogurt which the consumption of yogurt among Iranians is high, and iii) using whole-grain flaxseed instead of flaxseed oil or lignan.

CONCLUSIONS

In conclusion, the addition of flaxseed to the diet can reduce some risk factors in patients with type 2 diabetes mellitus. Hence, it is suggested consumption of flaxseed enriched yogurt may be useful as an alternative preventive approach and decrease diabetes complications. Additional studies with a more prolonged period of intervention, larger sample size and the various dose of flaxseed are needed to verify these effects.

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