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ORIGINAL ARTICLE

The effect of oral consumption of sesame oil on anthropometric, metabolic and oxidative stress markers of patients with type 2 diabetes: a double-blind, randomized controlled trial

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Abstract

Background Sesame oil has several polyphenols with possible anti-diabetic and antioxidative characteristics. However, there is limited evidence for the efficacy of sesame oil on type 2 diabetes mellitus (T2DM) consequences.

Objective We aimed to investigate the effects of sesame oil as one of the safest edible oils and whether it could be an adjuvant therapy to medications in T2DM patients.

Methods Fifty-six patients with T2DM were enrolled in the study. They were randomly divided into two groups, receiving sesame oil (SO) as the intervention group ($n=28$) or sunflower seeds oil (SSO) as the placebo group ($n=28$) for 8 weeks. We assessed anthropometric, lipid profile, oxidative stress, and inflammatory indicators. We used paired sample *t*-test/Wilcoxon test and an unpaired *t*-test to examine the differences among and between the arms.

Results The results showed that malondialdehyde (MDA) was reduced significantly in the SO group (1.33 ± 1.26, $p=0.001$). However, the results in the SSO group were vice versa, and MDA noticeably increased in the control group after eight weeks (-2.08 ± 1.01, $p<0.001$). Despite the in-group changes, no significant differences were spotted in anthropometric, glycemia, lipid profile, and inflammatory indicators between both intervention and control groups.

Conclusion Our findings suggest a possible antioxidative effect of sesame oil by decreasing MDA with no effects on other measured outcomes. This is a safe and effective option for relieving oxidative stress, as the supplementation with sesame oil for 8 weeks was not associated with harmful effects.

Keywords Glycemia · Inflammatory markers · Lipid profile · Oxidative stress markers · Sesame oil · Type 2 diabetes

Introduction

T2DM is one of the most prevalent chronic diseases known as non-insulin-dependent. In 2017, approximately 4 million adults passed away due to diabetes, and nearly half of them were younger than 60 years old [1]. Ninety-five percent of diabetes patients suffer from T2DM [1]. The pathogenesis of T2DM is the combination of insulin insensitivity in organs that typically should respond to adequate insulin doses and the inability of pancreatic B cells to release enough insulin [2]. T2DM is mainly characterized by instability in insulin levels [3], hypoglycemia [1], and high hemoglobin A1C levels [4]. Glucose dysregulation in T2DM patients could lead to many microvascular and macrovascular complications, such as neuropathy, nephropathy, cardiomyopathy, sarcopenia, and retinopathy [5–7]. Almost all people are in danger of T2DM, but some risk factors significantly increase this possibility. Diabetes family history [8], obesity [9], and sedentary lifestyle [10] are amongst the most critical risk factors of T2DM. Until today, no reliable cure for T2DM has been introduced; however, many treatments help control blood glucose levels and other diabetes complications [11–14]. Oral medications are the first option for controlling T2DM

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or even preventing pre-diabetes from turning into T2DM. Metformin is the most used anti-diabetic medication. Also, there are injectional medications such as insulin and GLP-1 agonist [15]. Despite all the advantages of anti-diabetic drugs, some unavoidable side effects might appear in some consumers. These medications' most common side effects include hypoglycemia, nausea, dizziness, and unwanted changes in weight [16]. Finding new and more effective treatments for curing or preventing T2DM is always one of the significant concerns of practitioners and investigators, who are constantly striving to find a novel and promising target for the prevention and treatment of the disease [17, 18]. Improving chronic diseases through dietary and natural treatment has always been popular, especially among chronic patients [19, 20]. Recent studies have discussed that various new agents and natural compounds may improve symptoms of different chronic conditions [21, 22] including diabetes complications [23, 24]. Nowadays, more studies are being done on dietary factors to reduce the risk of T2DM and modulate its complications.

Fats could have advantages for T2DM patients as well as disadvantages. Studies have shown that dietary oils can modulate the gut microbiota by increasing the abundance of beneficial bacteria [25, 26]. Moreover, it has been shown that altering the composition of the gut microbiota can lead to improvements in symptoms and quality of life in patients with chronic or critical conditions [27–30]. Evidence suggests that free fatty acids in plasma modulate insulin activity and insulin-dependent glucose uptake [31]. Based on recent publications, Sesame oil has shown some positive effects on insulin sensitivity and glucose tolerance among T2DM patients [32, 33]. Sesame oil, as one of the current accessible oils for dietary usage and oral intake, is known to have a significant amount of PUFAs (poly-unsaturated fatty acids) and MUFAs (mono-unsaturated fatty acids) [34]. Sankar et al. reported that sesame oil had indicated positive effects on plasma glucose levels and lipid profiles in diabetic patients [35]. In addition to fatty acids, sesame oil, and sesame seeds, several polyphenols carry sesame's main antioxidative properties. sesamin, sesamol, episesamin, and sesamolin are the main phenols of sesame [36]. A significant part of sesame oil's anti-diabetic, anti-hyperlipidemic, and antioxidative effects is on these polyphenols and lignans [37–40]. Sesame oil also has a considerable vitamin E content as a fat-soluble vitamin and antioxidative agent [34].

In this study, we aimed to investigate sesame oil's anti-diabetic and antioxidative effects as one of the safest edible oils and whether it could be an adjuvant therapy to medications in T2DM patients.

Materials and methods

Subjects

T2DM patients were recruited from the Akhavan special clinic of Kashan University of medical sciences. All participants were between 30 and 70 years of age, non-smokers, and nonalcoholic. Eligible patients had HbA1C between 7 and 9% in the last 3 months, had not regularly consumed sesame oil (more than once per week) in the last 3 months, had not shown allergic reactions to sesame, and had not changed the dose of their anti-diabetic drugs at least 4 weeks before the study started. Criteria for exclusion were severe cardiovascular disorder, infection, kidney disease, hyperthyroidism or hypothyroidism, pregnancy, and lactation phase. During the study, participants who had a significant change in their physical activity, type, and doses of anti-diabetic drugs, started taking omega-3 supplements, showed any allergic reaction, or had not used the oils abided by prior orders were excluded from the study.

Study design

This study was a randomized, double-blind control trial. Subjects, investigators, staff, and statisticians were blinded to intervention during the study. The sample size was measured based on the information obtained from a similar article [41], considering the power of the test is 80%, and the type 1 error is 0.05. The required number of people in each group was calculated as 28 patients, considering that 10% of the participants may drop out during the study. Fifty-six patients with T2DM were enrolled in the study via the simple random sampling method. www.sealedenvelope.com was used for randomization to establish sesame oil and placebo group [42]. They were randomly divided into two groups, receiving sesame oil (SO) as the intervention group ($n = 28$) or sunflower seeds oil (SSO) as the placebo group ($n = 28$). Randomization was done using computer-generated randomization in blocks of four. Kashan Research Center for Biochemistry and Nutrition in Metabolic Diseases was responsible for labeling the bottles of oils with the subject's randomization number. The bottles provided to both groups were similar in appearance and packaging. For 8 weeks, the patients were instructed to prepare salads or cook with about 15 g of oil per individual each day. A short questionnaire was used at the first appointment to record demographic characteristics

and smoking and alcohol consumption. At the first visit and after the intervention period, venous blood samples of patients were collected to measure FBS level, insulin level, lipid profile, CRP (C-reactive protein) as an inflammatory biomarker, and oxidative stress biomarkers (GSH, TAC, and MDA). Anthropometric factors also were assessed before and after the intervention period.

Participants were asked to keep their routine diet and physical activity until the end of the intervention. In order to evaluate individual intakes of energy and nutrient intakes including proteins, carbohydrates, lipids, and other nutrients, patients have been asked to fill a 3-day food diary on three consecutive days (two weekdays and one weekend day) at the first and eighth weeks of the study. The 3-day food diary was analyzed using Nutritionist 4 software. Moreover, the IPAQ (international physical activity questionnaire) was filled out for each participant at the intervention's beginning and end to indicate if their physical activity had changed throughout the study. The validity and reliability of the Iranian version of IPAQ have been assessed [43]. Patients received a phone call weekly for a follow-up check and a personal meeting in the fourth week to minimize withdrawal and ask about possible complications.

Laboratory procedures

Sample collection and plasma preparation

On the mornings of the first appointment and the day at the end of the study, research nurses collected 10 ml of 12 h-fasting venous blood of patients between 7:00 and 9:00 a.m. Venous blood samples were collected into plain tubes, and after storage for 30 min at room temperature, a blood clot formed, and plasma samples were separated by centrifugation at 3000 g for 10 min and stored in clean microtubes in a − 70° C freezer until analysis.

Fasting blood sugar (FBS), insulin plasma level, and insulin indexes

FBS level was assessed by glucose oxidase/peroxidase method with commercial kits (Pars Azmun, Iran). The insulin level was measured by sandwich ELISA method using IBT kits (Infinitum Biotech, the Netherlands).

The HOMA-IR and QUICKI are two indexes to measure insulin sensitivity. HOMA-IR (homeostatic model assessment for insulin resistance) is calculated as fasting insulin (μU/ml) × fasting glucose (mmol/l)/22.5 (normal young people insulin resistance = 1). QUICKIE (quantitative insulin-sensitivity check index) is calculated by the formula 1/(log fasting insulin [μU/ml] + log glucose [mg/dl]) [44, 45].

Lipid profile blood tests

Automated enzymatic procedures were used for the measurement of total cholesterol, HDL (high-density lipoprotein), LDL (low-density lipoprotein), and TG (triglyceride) levels with BT3000 autoanalyzer (Biotecnica, Italy) and commercial kits (Pars Azmun, Iran).

Oxidative stress and inflammatory biomarkers

The ferric-reducing antioxidant power (FRAP) method was used to measure the plasma's total antioxidant capacity (TAC). This approach used FeSO4.7H2O solution as standard (0.1–1 mmol/L) to measure TAC by reducing a ferric tripyridyltriazine (Fe3 + -TPTZ) complex to Fe2 + form. The reduced glutathione (GSH) concentration in plasma was measured using DTNB, which forms a yellow color complex with GSH. Additionally, the MDA of plasma was measured using a colorimetric assay, this compound's reaction with TBA, and the development of a pink color complex. A 540-nm wavelength was used to measure the complex's optical absorption. Using quantitative CRP kits, we used the ELISA approach to evaluate CRP as an inflammatory biomarker. (Pars Azmun, Karaj, Iran).

Anthropometric factors

Anthropometric factors, including weight, BMI (body mass index), SMM (skeletal muscle mass), BFM (body fat mass), PBF (percent body fat), and WHr (waist-to-hip ratio), were assessed before and after the intervention period by InBody 770 body analyzer (InBody, South Korea). The measurement of anthropometric factors was conducted according to the body analyzer manufacturer's instructions.

Statistical analysis

The obtained data were analyzed by SPSS (Statistical Package for the Social Sciences) 22 version. We used the Kolmogorov–Smirnov test to determine the normality of variable distribution. Paired sample *t*-test and Wilcoxon test were used for the changes in anthropometric and biochemical parameters in patients before and after the intervention. Moreover, an unpaired *t*-test was used to examine the differences between the arms.

Result

From 56 volunteers who had recruited for the study between August and September 2020, 39 patients completed the trial. No adverse effects were reported during or after the intervention. However, after 8 weeks, a total of seventeen patients were lost to follow-up. Seven patients from the intervention group and ten patients from the control group were unable to continue participating in the study. This was due to various reasons, including four patients being unable to follow the study protocol, 11 patients expressing personal desire to discontinue, and two patients experiencing changes in their clinical condition (Fig. 1).

Of all the participants, 21 were male (53.8%) and 18 were female (46.2%). The average age in the SO group was 50 ± 8.31, and in the SSO group was 55.38 ± 8.34. No statistically significant difference was found across the group for any of the characteristics of subjects at baseline except for age and TAC (Table 1).

Dietary intake

Dietary intake of baseline and after 8 weeks of intervention in both groups are presented in Table 2. No significant difference has been spotted in baselines of macronutrients and micronutrients in both groups; however, slight changes in mean dietary intakes occurred after the intervention period (Table 2).

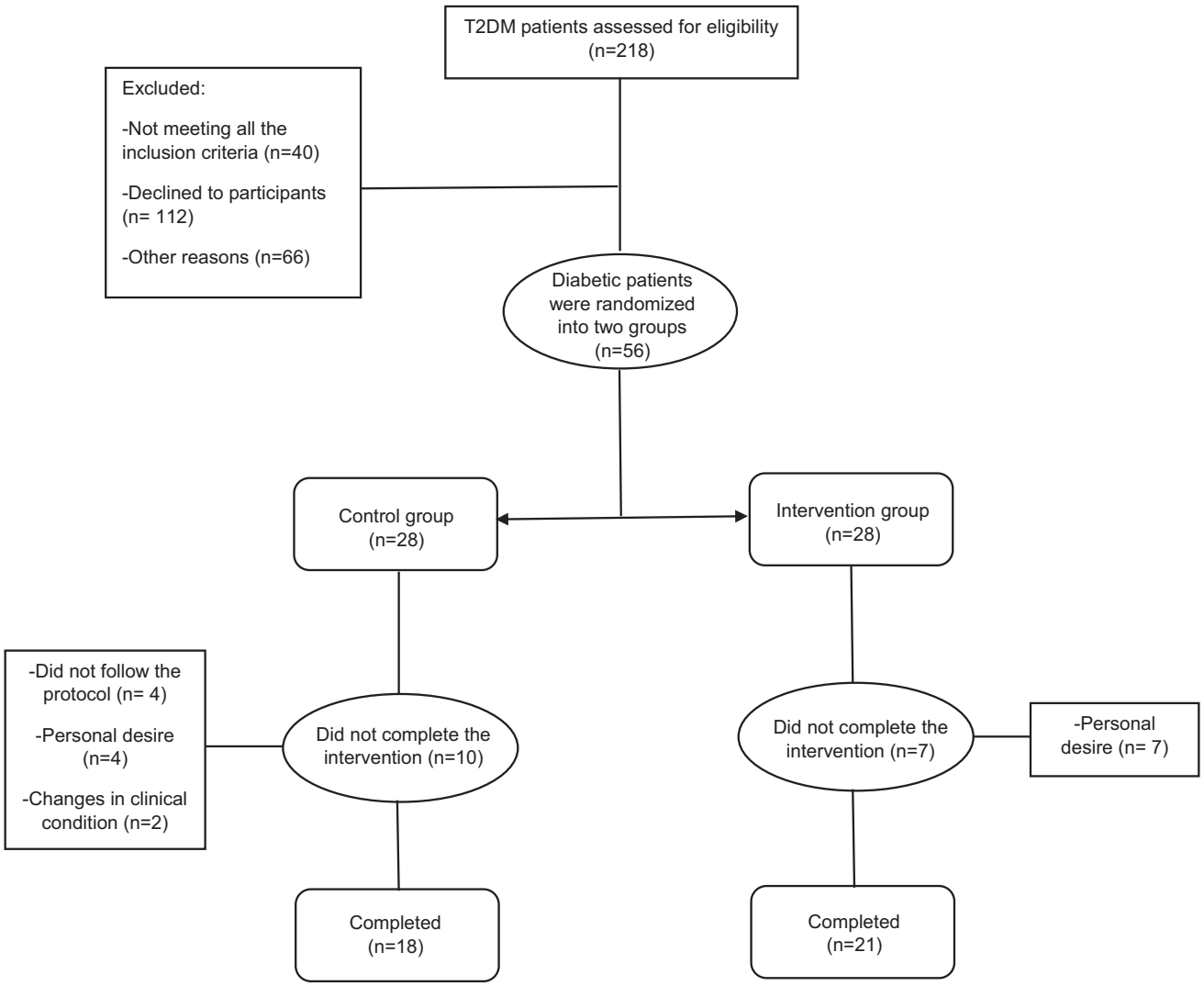

Fig. 1 Trial profile

Table 1 Baseline characteristics in the sesame oil group and sunflower seed oil group

Baseline characteristic	Intervention group ($n=21$)	Control group ($n=18$)	p value
Age (year)	50.0 ± 8.31	55.38 ± 8.34	0.041
Sex			
Male	12 (57.1%)	9 (50%)	
Female	9 (42.9%)	9 (50%)	
Height (cm)	165.23 ± 11.50	164.27 ± 8.87	0.77
GSH	40.2 ± 7.25	41.24 ± 8.23	0.68
MDA*	8.12 ± 0.81	7.69 ± 0.61	0.056
TAC	1119.1 ± 173.68	972.2 ± 126.3	0.00
CRP*	1.95 ± 1.20	2.61 ± 2.63	0.602
Chol	122.0 ± 41.1	138.5 ± 46.8	0.249
LDL	63.6 ± 24.9	74.3 ± 3 2.0	0.248
HDL*	37.1 ± 11.5	42.6 ± 12.1	0.076
TG*	151.2 ± 100.4	136.5 ± 84.3	0.746
FBS*	151.2 ± 60.3	153.4 ± 69.5	0.789
Insulin*	10.61 ± 5.71	9.02 ± 3.01	0.535
HOMA-ir*	4.32 ± 3.42	3.54 ± 2.40	0.573
Quicki*	0.32 ± 0.04	0.32 ± 0.03	0.573
Weight (kg)	80.07 ± 13.9	74.9 ± 16.1	0.291
InBody® score*	71.1 ± 10.08	69.6 ± 9.2	0.394
SMM	29.8 ± 6.19	27.8 ± 6.6	0.352
BFM*	26.1 ± 12.3	24.0 ± 11.1	0.545
BMI* (kg/m^2)	29.6 ± 6.11	27.7 ± 6.1	0.181
PBF	32.0 ± 10.9	31.6 ± 9.0	0.921
Whr	0.92 ± 0.06	0.93 ± 0.06	0.628

Values are mean ± SD or number

*Nonparametric variants

GSH (glutathione), MDA (malondialdehyde), TAC (total antioxidant capacity), CRP (C-reactive protein), Chol (cholesterol), LDL (low-density lipoprotein), HDL (high-density lipoprotein), TG (triglyceride), FBS (fasting blood sugar), HOMA-ir (homeostatic model assessment for insulin resistance), Quicki (quantitative insulin sensitivity check index), InBody ® Score (it is a number in the body composition report indicating fat mass and lean body mass values compared to healthy average ranges), SMM (skeletal muscle M ass), BFM (body fat mass), BMI (body mass index), PBF (percent body fat), Whr (waist-to-hip ratio)

Table 2 Dietary intake before and after the intervention period in the sesame oil group and sunflower seed oil group

Daily intake	Sesame oil ($n=21$)		Sunflower seed oil ($n=18$)	
	Before	After	Before	After
Energy (Kcal)	1842.71 ± 277.99	1938.01 ± 245.87*	1712.38 ± 286.62	1653.12 ± 305.58**
Protein (g)	72.62 ± 15.65	78.96 ± 17.94*	65.29 ± 12.41	61.13 ± 12.15**
Carbohydrate (g)	215.25 ± 42.94	229.90 ± 25.55	203.33 ± 37.28	199.52 ± 46.96**
Lipid (g)	81.64 ± 13.25	83.28 ± 13.13	75.66 ± 17.39	72.36 ± 18.24
SAF (g)	22.16 ± 6.293	22.06 ± 5.346	19.76 ± 5.84	20.05 ± 6.82
MUFA (g)	30.87 ± 4.67	31.37 ± 4.74	29.33 ± 7.01	26.97 ± 7.72
PUFA (g)	21.97 ± 2.82	23.20 ± 3.31*	20.94 ± 6.78	19.27 ± 6.38**
Fiber (g)	36.78 ± 15.15	34.00 ± 15.24	33.29 ± 22.82	30.49 ± 19.73

Values are mean ± SD

*Significantly different from baseline ($p<0.05$) (based on the results of paired sample t-test)

**Significantly different from intervention group ($p<0.05$) (based on the results of independent sample t-test)

SAF (saturated fatty acid), MUFA (mono-unsaturated fatty acid), PUFA (poly-unsaturated fatty acid)

Fasting blood sugar (FBS), insulin plasma level, and insulin indexes

Insulin levels in the SO group were decreased after the intervention (1.60 ± 6.84), although these changes were not significant. QUICKI and HOMA-IR did not show a significant change by the intervention. FBS insignificantly increased in both groups (SO group: − 1.65 ± 96.85, $p = 0.274$; SSO group: − 7.77 ± 53.05, $p = 0.446$); nevertheless, this increment has been more in the SSO group (Table 3). Eight weeks of intervention did not significantly influence FBS ($p = 0.746$) and insulin ($p = 0.844$) values between the two arms. Additionally, HOMA-IR and QUICKI did not show significant differences between intervention and control groups ($p = 0.324$ and $p = 0.398$, respectively) (Table 3).

Oxidative stress and inflammatory biomarkers

No significant change has been observed in GSH levels before and after intervention duration in the SO group (− 0.06 ± 9.31, $p = 0.974$) nor the SSO group (− 0.35 ± 9.25, $p = 0.872$). No significant change was detected in TAC in the SO group (− 72.58 ± 162.25, $p = 0.054$); however, a significant enhancement of TAC was indicated in the SSO group (− 88.25 ± 126.10, $p = 0.009$). CRP levels were increased insignificantly in both groups (SO − 0.38 ± 1.35, $p = 0.242$ and SSO − 0.38 ± 2.59, $p = 0.797$). MDA was reduced significantly after intervention in the SO group (1.33 ± 1.26, $p = 0.001$). However, the results in the SSO group were vice versa, and MDA noticeably increased in the control group after eight weeks (− 2.08 ± 1.01, $p < 0.001$). Although GSH, TAC, and CRP did not indicate a significant difference between intervention and placebo groups ($p = 0.923$ and $p = 0.370$, respectively), MDA was significantly lower in the SO group compared with the SSO group ($p = 0.000$) (Table 3).

Lipid profile

As it is shown in Table 4, total cholesterol, LDL-C, and HDL-C slightly increased after intervention in both the SO and the SSO groups. In the SO group, some non-significant changes were observed in total cholesterol (− 7.28 ± 42.45, $p = 0.441$), LDL-C levels (− 4.71 ± 28.41, $p = 0.456$), and HDL-C levels (− 3.04 ± 8.35, $p = 0.238$). TG levels decreased after the intervention in the SO group (22.85 ± 114.0, $p = 0.881$), while in the control group, a non-significant increase in TG level was observed (− 21.11 ± 84.07, $p = 0.794$). Despite all these in-group changes, no significant differences were spotted in total cholesterol ($p = 0.974$), LDL cholesterol ($p = 0.845$), HDL cholesterol ($p = 0.309$), and triglyceride ($p = 0.778$) between both intervention and control groups (Table 4).

Anthropometric factors

After 8 weeks of intervention, the patients' mean weight for the sesame oil (SO) group had not changed compared to the baseline. The mean weight for the sunflower seed oil (SSO) group slightly increased. However, none of these changes were statistically significant; no significant change in body mass index (BMI) was observed before and after the intervention in the SO group (0.03 ± 0.57, $p = 0.663$) or SSO group (− 0.06 ± 0.77, $p = 0.079$). The mean of InBody®

Table 3 Differences in antioxidants and FBS, and insulin before and after 8 weeks of intervention

Variable	Baseline (mean ± SD)		8weeks (mean ± SD)		Difference (mean ± SD)		*p* value within *groups*[a]		*p* value between *groups*[b]
	Intervention ($n = 21$)	Placebo ($n = 18$)	Intervention ($n = 21$)	Placebo ($n = 18$)	Intervention ($n = 21$)	Placebo ($n = 18$)	Intervention ($n = 21$)	Placebo ($n = 18$)	
GSH	40.2 ± 7.25	41.24 ± 8.23	40.3 ± 6.92	41.60 ± 6.51	− 0.06 ± 9.31	− 0.35 ± 9.25	0.974	0.872	0.923
MDA*	8.12 ± 0.81	7.6 ± 0.61	6.79 ± 0.74	9.77 ± 0.93	1.33 ± 1.26	− 2.08 ± 1.01	0.001	0.00	0.00
TAC	1119.1 ± 173.68	972.2 ± 126.3	1191.7 ± 158.11	1060.4 ± 185.7	− 72.58 ± 162.25	− 88.25 ± 126.10	0.054	0.009	0.741
CRP*	1.95 ± 1.20	2.61 ± 2.63	2.33 ± 1.39	3.0 ± 4.48	− 0.38 ± 1.35	− 0.38 ± 2.59	0.242	0.797	0.393
FBS*	151.2 ± 60.3	153.4 ± 69.5	152.9 ± 92.7	161.2 ± 54.3	− 1.65 ± 96.85	− 7.77 ± 53.05	N.S	0.586	0.746
Insulin*	10.61 ± 5.71	9.02 ± 3.01	9.00 ± 8.33	9.14 ± 5.61	1.60 ± 6.84	− 0.11 ± 4.61	0.274	0.446	0.844
HOMA-ir*	4.32 ± 3.42	3.54 ± 2.40	3.92 ± 5.08	3.93 ± 3.60	0.395 ± 3.72	− 0.39 ± 3.33	0.394	0.586	0.324
Quicki*	0.32 ± 0.04	0.32 ± 0.03	0.35 ± 0.06	0.32 ± 0.03	− 0.027 ± 0.66	− 0.00 ± 0.02	0.159	0.774	0.398

[a]The results of the paired-*t*-test for parametric variables and *Wilcoxon test for nonparametric variables

[b]The results of the independent *t*-test for parametric variables and *Mann–Whitney *U* test for nonparametric variables

*Significant *p*-value considered less than 0.05 and the confidence interval percentage at 95%

GSH (glutathione), MDA (malondialdehyde), TAC (total antioxidant capacity), CRP (C-reactive protein), FBS (fasting blood sugar)

Table 4 Differences in anthropometric characteristics and lipid profile before and after 8 weeks of intervention

Variable	Baseline (mean ± SD)		8weeks (mean ± SD)		Difference (mean ± SD)		*p* value within *groups*[a]		*p* value between *groups*[b]
	Intervention ($n=21$)	Placebo ($n=18$)	Intervention ($n=21$)	Placebo ($n=18$)	Intervention ($n=21$)	Placebo ($n=18$)	Intervention ($n=21$)	Placebo ($n=18$)	
Weight	80.07 ± 13.9	74.9 ± 16.1	80.07 ± 13.8	75.2 ± 15.6	0.00 ± 1.78	− 0.28 ± 1.90	N.S	0.526	0.628
InBody® score*	71.1 ± 10.08	69.6 ± 9.2	70.7 ± 10.33	69.7 ± 9.8	0.42 ± 3.57	− 0.05 ± 2.01	0.930	0.949	0.988
SMM	29.8 ± 6.19	27.8 ± 6.6	29.7 ± 6.24	27.9 ± 6.7	0.12 ± 1.13	− 0.05 ± 0.84	0.610	0.804	0.586
BFM*	26.1 ± 12.3	24.0 ± 11.1	26.5 ± 12.4	24.4 ± 10.7	− 0.31 ± 1.54	− 0.43 ± 1.76	0.602	0.395	0.693
BMI*	29.6 ± 6.11	27.7 ± 6.1	29.6 ± 6.2	27.8 ± 5.8	0.03 ± 0.57	− 0.06 ± 0.77	0.663	0.079	0.693
PBF	32.0 ± 10.9	31.6 ± 9.0	32.4 ± 11.1	31.9 ± 9.1	− 0.44 ± 2.28	− 0.31 ± 1.48	0.380	0.387	0.829
Whr	0.92 ± 0.06	0.93 ± 0.06	0.94 ± 0.05	0.95 ± 0.06	− 0.01 ± 0.03	− 0.01 ± 0.02	0.046	0.005	0.705
Chol	122.0 ± 41.1	138.5 ± 46.8	129.3 ± 35.1	146.2 ± 37.6	− 7.28 ± 42.45	− 7.72 ± 38.90	0.441	0.411	0.974
LDL	63.6 ± 24.9	74.3 ± 3 2.0	68.3 ± 22.7	77.4 ± 27.0	− 4.71 ± 28.41	− 3.05 ± 23.26	0.456	0.585	0.845
HDL*	37.1 ± 11.5	42.6 ± 12.1	40.1 ± 7.8	43.5 ± 8.6	− 3.04 ± 8.35	− 0.83 ± 10.15	0.238	0.694	0.309
TG*	151.2 ± 100.4	136.5 ± 84.3	128.3 ± 58.0	157.6 ± 129.1	22.85 ± 114.0	− 21.11 ± 84.07	0.881	0.794	0.778

[a]The results of the paired *t*-test for parametric variables and *Wilcoxon test for nonparametric variables

[b]The results of the independent *t*-test for parametric variables and *Mann–Whitney *U* test for nonparametric variables

*Significant *p*−value considered less than 0.05 and the confidence interval percentage at 95%

score, skeletal muscle mass (SMM), percent body fat (PBF), and body fat mass (BFM) after intervention did not show any significant difference compared with the baseline values in both the SO group and SSO group. Nevertheless, after 8 weeks of intervention, the waist-to-hip ratio (WHr) has significantly increased in both groups compared to their baseline values (SO group − 0.01 ± 0.03, $p=0.046$; SSO group: − 0.01 ± 0.02, $p=0.005$). None of the measured anthropometric characteristics showed any significant difference between the SO and SSO groups (Table 4).

Discussion

T2DM is a complex metabolic disorder that affects numerous physiological processes related to glucose metabolism, oxidative stress, inflammation, and lipid profile. High blood glucose levels, oxidative stress, and inflammatory markers are commonly observed in individuals with T2DM. In light of these multiple pathophysiological abnormalities, effective therapy for T2DM should address not only glycemic control but also other related factors such as oxidative stress, inflammation, and lipid metabolism. Oxidative stress and inflammatory responses are key components of the pathophysiology of diabetes and its complications. These can lead to damage to various tissues and organs, including the nervous system. In particular, oxidative stress-induced damage to nerve cells and the surrounding microvasculature is a major contributor to the development of diabetic or non-diabetic neuropathies [46, 47].

Previous investigations have examined the effects of sesame oil on glycemia indices in patients with T2DM, but the findings have been controversial. Majid Mohammad Shahi et al. suggested that after 8 weeks, sesamin supplementation in T2DM patients was associated with a decrease in FBS and hemoglobin A1C [48]. In another study, Aslam et al. revealed that after 90 days of sesame oil consumption, FBS was significantly decreased in the sesame oil group compared to the control group, while insulin level was significantly increased [49]. However, Raeisi-Dehkordi et al. revealed no significant difference between sesame oil and control groups in FBS, insulin, HOMA-IR, and QUICKI levels [50]. In the current study, FBS had slightly increased in both groups after the intervention duration, although this enhancement was more in the sunflower seed oil group. At the same time, insulin level indicated controversial results; its amount had decreased in the sesame oil group and increased in the sunflower seed oil group. However, similar to the Raeisi-Dehkordi et al. study, all these results were statistically non-significant between groups.

Regarding oxidative stress and inflammatory markers, 8 weeks of intervention with 15 g/day of sesame oil was not related to any significant changes in the GSH and CRP, while interestingly, results showed that MDA had been significantly reduced by sesame oil intervention. At the same time, its increment in the control group has been observed. Regarding MDA's significant decrease in the sesame group,

put together with MDA's significant increase in the control group, sesame oil could have antioxidative effects and reduce the unwanted metabolite of fatty acids peroxidation.

During prolonged oxidative stress, MDA levels in the body rise [51]. One of the main results of free-radical-mediated injury is lipid peroxidation, which affects membranes directly and produces several secondary products, including aldehydes like MDA, which is the most prevalent individual aldehyde [52]; free radicals start a chain reaction that results in cell death, DNA damage, lipid peroxidation, and neurological issues. Sesame oil lignans, including sesamin or sesamol, may help explain why sesame oil has antioxidative properties. However, it is still unclear which active ingredients are responsible for sesame oil's significant antioxidant action [53].

In an oxidative stress situation, total antioxidant capacity (TAC) decreases [54], and it is measured as an indicator of oxidative stress [54]. In our study, no significant alteration of TAC levels was observed in the sesame oil group. Although the amount of TAC was significantly increased among the sunflower seed oil group (control group), it did not indicate a significant difference between the intervention and placebo groups. This marginal effect may be because sunflower seed oil is a source of phenols and tocopherols [55]. The coagulation score and several oxidative and inflammatory indicators were examined by Akrami et al. in participants with MetS who consumed flaxseed oil and sunflower oil. There was no discernible difference between the two groups coagulation scores and serum TAC levels after 7 weeks, which agreed with the current study [56].

Fasting hypertriglyceridemia and increased lipemia are common in T2DM patients [57, 58]. Therefore, a diet that lowers lipid concentrations might help to prevent cardiovascular problems linked to T2DM, such as atherosclerosis [59]. Mono-unsaturated fatty acids (MUFA) and poly-unsaturated fatty acids (PUFA) reduce those risks via altering lipids, whereas dietary saturated fatty acids (SFA) raise the risk for cardiovascular disease [60]. Around 50% of sesame seeds are oil, whereas 20% are protein, and up to 1.5% of them are lignans, including sesamin and sesamolin [61]. The sesame seed's potential to lower plasma cholesterol is due to its high dietary fiber and linoleic acid content [38, 62]. Despite no alteration in lipid profile items observed in our study, the TG level in the sesame oil group had been reduced through the intervention and increased in the control group. In other studies, results regarding the effect of sesame oil on lipid profile and blood glucose indices are controversial. In a study, the sesame oil group had lower TC and LDL-C values, although these reductions were not statistically significant. However, compared to the control group, the sesame oil group's TC to HDL-C ratio was significantly lower [63]. In a related study, Khajehdehi et al. examined kidney-affected individuals who consumed sesame oil for eight weeks to support these findings. They revealed that the levels of TC, TG, and LDL-C were unaltered at the end of the intervention. [64]. Namayandeh et al. indicated that after sesame oil consumption, cholesterol, TG, and LDL-C were significantly decreased [65]. Sankar et al. reported that sesame oil can decrease TG levels among T2DM patients. Their study was 60 days, and each patient in the sesame oil group was supplemented with 35 g of sesame oil per day [53]. Golzarand et al. reported that grounded sesame seed consumption in diabetic patients decreased TG significantly, but no significant changes were seen in FBS. Farajbakhsh et al. indicated that in individuals who consumed sesame oil enriched with or without vit E, there were significant reductions in serum total cholesterol (TC), triglycerides (TG), FBS, HOMA-IR, and MDA [66].

Almost all anthropometric items, including weight, InBody score, BMI, BMF, SMM, and PBF, showed no noticeable change after eight weeks of intervention with either sesame or sunflower seed oil. The only anthropometric item that was affected was WHr (waist-to-hip ratio). WHr was significantly increased in both groups; even so, the between groups results had not shown any significant difference between the sesame and sunflower oil. There are limited investigations on sesame seed and sesame oil's effect on body composition. Sankar et al. study reported a significant reduction in WHr, BMI, and weight in diabetic samples after 45 days of intervention with sesame oil; however, after 45 days of intervention withdrawal, almost all of these amounts returned to their pre-intervention levels. This article suggested sesame oil's "PUFA" content as the leading cause of this result [35]. On the contrary, another recent study indicated no significant change in anthropometric parameters among intervention and placebo after sesamin supplementation [48].

Such discrepancies between the results of different studies could be attributed to several limitations of our study. Firstly, differences in the characteristics of the study populations, such as the participant's age, gender, ethnicity, and disease duration, could have played a role in the observed differences. Secondly, variations in the dosages of sesame oil used across different studies could have affected the outcomes, as the optimal dosages of sesame oil for glycemic control in patients with T2DM remain unclear. Thirdly, differences in the duration of treatment could have influenced the results, as longer treatment durations may be needed to observe significant effects on glycemic indices. Finally, the specific study designs used to assess the effects of sesame oil on glycemic indices also contributed to the discrepancies between the findings of different studies. Therefore, it is essential to consider these factors when interpreting the results of studies investigating the effects of sesame oil on glycemic indices in patients with T2DM. Further research using standardized methodologies and larger sample sizes is

needed to clarify the potential benefits of sesame oil in the management of T2DM and to identify the optimal dosages and durations of treatment.

Conclusion

In conclusion, the results of our study reveal possible antioxidative effects of sesame oil by decreasing MDA. Eight weeks of supplementation with sesame oil did not leave any harmful effect on the intervention group, so this could be considered a safe and effective option for relieving oxidative stress, as the supplementation with sesame oil for 8 weeks was not associated with any harmful effects. However, further research should investigate the potential mechanisms of action by which sesame oil exerts its antioxidative effects. Understanding these mechanisms will enhance our knowledge of the underlying processes involved and could potentially lead to the development of targeted therapies. Studies could also explore the optimal dosage and duration of sesame oil supplementation to achieve the maximum antioxidative effects. Different dosages and durations should be tested to determine the most effective regimen. Future studies could also explore the potential synergistic effects of combining sesame oil with other anti-diabetic medications or lifestyle interventions. This could help determine whether sesame oil can enhance the efficacy of existing therapies or interventions for T2DM. The effect of sesame oil on specific subgroups of T2DM patients should be investigated, such as those with different levels of disease severity or comorbidities. This could help identify potential beneficiary populations who may respond more favorably to sesame oil supplementation.

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Author contributions SJ, NA, and MT conceived the trial and designed the experiment. NA, MT, MS, and SJ assisted with the conduction of the study and wrote the manuscript. SJ supervised the study. All study authors read and approved the final version of the manuscript.

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Data Availability The data supporting the findings of this study will be made available upon reasonable request in a scientifically sound manner.

Declarations

Competing interests The authors declare no competing interests.

Ethical approval and Consent of patient The study protocol was registered at the Iranian Registry of Clinical Trials (IRCT20150920024103N2) and was approved by the Ethics Committee of Kashan University of Medical Sciences (IR.KAUMS.MEDNT.REC.1398.055). At the first clinical visit, the risks and benefits of the study were described in detail, and all participants provided written informed consent. All patients gave oral informed consent before enrolment in the study after fully describing the study procedure to them.

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