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# The effect of Okra (Abelmoschus esculentus) on lipid profiles and glycemic indices in type 2 diabetic adults: randomized double blinded trials

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**Running title**: Okra and Type 2 Diabetes

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**Abstract**:

**Background:** The aim of the present study was to investigate the effect of okra consumption on serum levels of lipid profiles and glycemic indices in Type 2 Diabetic (T2D) patients.

**Methods**: The present study was a randomized, double-blinded clinical trial, carried out in Kerman, Iran. Sixty T2D patients were randomized into intervention and control groups, and received 10 g okra powder blended in 150 g conventional yogurt or conventional yogurt alone, along with dinner and lunch, for 8-weeks. Glycemic markers and lipid profile were assessed, as well as anthropometric measures, at the beginning and end of study.

**Results**: The findings showed that 8-weeks okra consumption resulted in a significant decrease in fasting plasma glucose (-15.61±19.44 vs -3.40±24.78; P=0.02), HOMA-IR (-1.17±1.61 vs -0.14±1.64; P=0.01), QUICKI (0.01±0.007 vs 0.00±0.01; P=0.004), triacylglycerol (-22.30±32.46 vs -3.86±30.57; P=0.001), total-cholesterol (-10.23±10.36 vs -2.03±13.94; P=0.004), LDL-C (-8.15±10.01 vs -2.31±9.37; P=0.02) and LDL-C/ HDL-C ratio (-0.28±0.37 vs -0.08±0.24; P=0.01). No significant difference was observed between groups in HDL-C, HbA1C, fasting insulin levels and anthropometric measures.

**Conclusion**: The present study suggests that okra consumption can elicit improvements in lipid profile, as well as glycemic markers, among T2D patients.

**KEYWORD**: Abelmoschus esculentus, diabetics mellitus, lipid profiles, glycemic indices, trials.

**Introduction**:

Type 2 diabetes (T2D) is one of the most prevalent non-communicable diseases worldwide, responsible for morbidity and early mortality, and can be characterized by hyperglycemia and insulin resistance (DeFronzo et al., 2015). The etiology of T2D is related to both genetic and environmental factors, including dietary pattern and physical activity (Zheng, Ley, & Hu, 2018). In addition to the negative health consequences of T2D, those affected are at an increased risk for stroke, cardiovascular disease, kidney disease, and retinopathy (Al-Saeed et al., 2016; Bril & Cusi, 2016; Pourmasoumi, Hadi, Najafgholizadeh, Joukar, & Mansour-Ghanaei, 2019); which is attributable to associated abnormal metabolic factors, such as hyperlipidemia and hyperglycemia (Group, 2010). Furthermore, T2D can lead to a reduction in quality of life and impose a huge economic burden on patients and health care systems (Seuring, Archangelidi, & Suhrcke, 2015). In addition, the typically prescribed anti-diabetic pharmacological agents often result in unfavorable side effects, which can limit the compliance of patients (Chaudhury et al., 2017; Hasani et al., 2019). In this regard, finding an effective approach or adjuvant remedy, which could reduce the cost of treatment, as well as decrease the adverse effects of pharmacological agents, remains of paramount importance (Karimian, Hadi, Pourmasoumi, Najafgholizadeh, & Ghavami, 2019). In contemporary research and practice, medicinal plants have garnered interest, largely due to their purported beneficial effects on diseases, along with their overall lower cost and side effects in comparison to synthetic drugs (Clark, Ghaedi, Arab, Pourmasoumi, & Hadi, 2019; Pourmasoumi, Ghiasvand, et al., 2019).

Okra (Abelmoschus esculentus (L.) (Moench)), also called lady's finger, belonging to the Malvaceae family, is an annual plant which grows wildly in many countries worldwide (Amin, 2011). The components of okra are mainly carbohydrates, minerals, and vitamins, whilst also rich in bioactive ingredients, such as flavones, alkaloids, pectin, polysaccharides, and linoleic acid (C. Zhang et al., 2018). It has been shown that okra possesses anti-tumor and anti-inflammatory properties and may confer a protective effect on the liver (Freitas et al., 2016; Z. Liao et al., 2019; Monte et al., 2014). Recently, experimental studies have shown the potential anti-diabetic role of okra in animal models (Sabitha, Ramachandran, Naveen, & Panneerselvam, 2011; Z. H. Tian et al., 2015). Although the

animal studies have indicated a promising effect of okra on diabetic metabolic abnormalities (Huang, Wang, Lin, Lin, & Peng, 2017; Peng, Lin, Lin, Wang, & Huang, 2019), there is a paucity of data from human studies. Therefore, the present study was performed to investigate the effect of okra consumption on serum levels of lipid profile and glycemic indices in T2D patients.

**Methods**:

***Participants***

The present study was a double-blinded, single center, randomized clinical trial which was performed between 2015 to 2016. Participants were recruited from T2D diagnosed patients who were referred to Ali-Ebn-e-Abitalb, Kerman, Iran. T2D was diagnosed by an endocrinologist, in accordance with the American Diabetes Association Criteria (Association, 2013); fasting plasma glucose (FPG) ≥ 126 mg/dl, two-hour plasma glucose (2h-post prandial glucose) ≥ 200 mg/dl, and glycated hemoglobin (HbA1C) ≥ 6.5%. T2D patients aged 30 to 75 years old, with a Body mass index (BMI) between 20-30, and duration of disease less than 5 years were included to the study. The patients were excluded if they also had an additional diagnosed chronic disease, such as cardiovascular disease, chronic kidney disease, non-alcoholic fatty liver, pulmonary disease, chronic inflammatory disease, or if they used insulin, were pregnant or lactating, were smokers, were following a specially prescribed diet, underwent herbal and/or medical treatment and/or supplemented with nutrients, antioxidants or omega 3 in the last 6 months, or had a drastic change in their weight during the last 6 months. The sample size was computed by using a formula which was suggested for clinical trial studies; based on data from previous studies (Erfani Majd, Tabandeh, Shahriari, & Soleimani, 2018; Li et al., 2017), and assuming type I error ($\alpha$)=0.5, power 80%, and considering 20% attrition, the sample size was estimated to be 30 patients in each group.

The present study was conducted in accordance with the Declaration of Helsinki and the study protocol was approved by the Ethical Committee of Isfahan University of Medical science (Ethical code: IR.MUI.REC.1396.2.121). It was also registered at the Iranian registry of clinical trials (register number: IRCT20190701044064N1).

### *Study design*

Eligible participants were entered into a 2-week run-in period to refrain from possible medicinal herb intake. Firstly, the aim and nature of the study was explained, and informed consent was obtained from all participants. Subsequently, participants were matched, based on age and gender, and randomly assigned to the intervention or control group, to receive yogurt plus okra or yogurt alone, respectively, for 8-weeks. Randomization was performed by using a computer-generated random number sequence, and the process of randomization and allocation of participants was blinded from the researchers and participants until when the final data were analyzed. Participants were instructed to consume okra powder blended in yogurt or yogurt alone with dinner and lunch-time meals for 8-weeks. The okra was purchased from an herbal drug market and its' main ingredients are presented in **Table 1**. The okra group consumed 10 g okra powder (approximately equal to 100g fresh okra) blended in 150 g yogurt, whilst the placebo was given yogurt plus a consumable color, which made it similar in appearance to the okra plus yogurt. The intervention and control yogurt were similar as flavor and color, and were packaged and encoded by manufactures; therefore, blinding of researchers and participants was guaranteed until study cessation. Participants received their yogurt packs at the beginning of each week, and were also asked to bring back the empty packs to monitor participant adherence. In addition, participants were monitored through periodic phone interviewing to enhance the compliance of the intervention, as well as explore possible side-effects due to consuming okra.

### *Assessment of dietary intake and physical activity*

In order to assess dietary intake, the participants were asked to record a 3-nonconsecutive-days (two week-days and one weekend) food record at the beginning and end of the trial. Daily macro nutrient intake of participants was analyzed from food record information by using Nutritionist IV software (First Databank, San Bruno, CA), modified for Iranian diets. Physical activity was also evaluated using the metabolic equivalent of task (MET) questionnaire (Ainsworth et al., 2000) at the beginning and end of the trial. In addition, participants were requested to maintain their habitual dietary and physical activity practices during the study.

***Assessment of biochemical and anthropometrics variables***

Anthropometric measures were evaluated, following an overnight fast, at the beginning and end of study. Weight and height of participants was measured by a digital scale (Seca, Hamburg, Germany) and a non-stretch tape measure (Seca, Hamburg, Germany), respectively, while they were wearing minimal cloths and unshod. BMI was estimated by dividing weight (Kg) by height squared ($m^2$). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were also assessed using a mercury sphygmomanometer (ALPK2, Zhejiang, China; Datis Co, Tehran, Iran), twice for each person, and following 15 minutes of rest. The mean of the two measurements was recorded and entered for final analysis.

After 12 hours overnight fasting, a 10-milliliter blood sample was obtained from all participants at study commencement and after 8-weeks intervention. FPG, HbA1C, triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were evaluated by an enzymatic method (Pars Azmun, Tehran. Iran). Fasting serum insulin levels were determined by ELISA methods (DiaMetra, Milano, Italy). Homeostatic model of assessment for insulin resistance (HOMA-IR) and the quantitative insulin sensitivity check index (QUICKI) were estimated in accordance with the suggested formulas (Matthews et al., 1985; Pisprasert, Ingram, Lopez-Davila, Munoz, & Garvey, 2013).

***Statistical analysis***

All statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS) software version 20 (SPSS Inc., Chicago, USA). Kolmogorov-Smirnov test was applied to examine the normality of data distribution. The intention-to-treat (ITT) analysis was performed for all participants who randomly assignment into the intervention and control groups. The method of 'Last Observation Carried Forward' was used to dealing with missing data from dropout participants. To investigate the differences in general participant's characteristics and dietary intake between intervention and control groups, independent samples t-test was used. A paired T-Test was applied to determine the changes in each main outcome from baseline measures, in both intervention and control groups. In addition,

analysis of covariance (ANCOVA), adjusted for age, sex, participant's baseline energy intake, physical activity, and body weight, was performed to compare the difference between change of outcomes of interest between the 2 groups during the study. Data were presented as mean ± standard deviation (SD) and a P value <0.05 was considered statistically significant.

**Results**:

**Figure 1** demonstrates the participant's selection, the reasons for withdrawal, and number of patients included in the final analysis. Briefly, 60 T2D patients met the inclusion criteria and were randomly assigned to intervention and control groups. During follow-up, 5 patients from the okra group [unwilling to continue the study (n=2), lost to follow-up (n=3)] and 7 patients from the control group [unwilling to continue the study (n=4), lost to follow-up (n=2), migration (n=1)] were lost to attrition.

However, as ITT approach which was applied for dealing with information, data from all participants were analyzed.

There were no significant differences in baseline characteristics, including anthropometrics measures, age, gender, duration of disease, blood pressure, glycemic indices, and physical activity, between the intervention and control groups **(Table 2)**. In addition, according to information obtained from the 3-days food record, daily energy intake as well as dietary macro and micronutrient ingredients were similar between intervention and control groups **(Table 3)**. No adverse effect related to okra consumption was reported by patients throughout the study.

We found that the consumption of okra significantly reduced FPG (-15.61±19.44 vs -3.40±24.78; P=0.02), HOMA-IR (-1.17±1.61 vs -0.14±1.64; P=0.01), QUICKI (0.01±0.007 vs 0.00±0.01; P=0.004), TG (-22.30±32.46 vs -3.86±30.57; P=0.001), TC (-10.23±10.36 vs -2.03±13.94; P=0.004), LDL-C (-8.15±10.01 vs -2.31±9.37; P=0.02) and LDL-C/ HDL-C ratio (-0.28±0.37 vs -0.08±0.24; P=0.01) in comparison to the control group. However, we did not find any significant effect of okra intake on body weight (-0.50±2.04 vs -0.03±1.44; P=0.24), BMI (-0.17±0.95 vs -0.01±0.72; P=0.29), HDL-C (0.34±1.09 vs 0.31±2.32; P=0.76), HbA1C (-0.16±0.77 vs -0.14±1.12; P=0.55), SBP (-

192 1.20±5.08 vs -0.53±5.29; P=0.41), DBP (-0.68±5.91 vs -0.23±3.98; P=0.78), and fasting insulin levels
193 (-0.66±1.98 vs -0.02±1.65; P=0.23), when compared with the control group **(Table 4)**.

195 The present study suggests that okra consumption for 8-weeks can be beneficial in modulating FPG,
196 HOMA-IR, QUICKI as well as TG, TC, LDL-C, LDL-C/HDL-C ratio in diabetic patients. However,
197 okra did not elicit any significant influence on HDL-C, HbA1C, blood pressure, fasting insulin levels,
198 and body weight.

199 Despite the innumerable strategies that are administrated for the prevention and treatment of T2D, it
200 remains one of the most highly prevalent chronic diseases, globally. Herbal medicine, which has a
201 historical background in many countries, such as those in the middle east and east Asian geographies,
202 can be used as an alternative or adjuvant remedy for T2D. Indeed, the present study demonstrated that
203 okra consumption yielded improvements in FPG, QUICKI and HOMA-IR. In accordance with these
204 results, previous empirical studies have indicated that okra can improve the glucose and lipid metabolic
205 parameters in T2D. A study conducted on diabetes -induced mice, via high-fat diet feeding combined
206 with streptozotocin intraperitoneal injection, showed that 8-weeks administration with 200 or 400
207 mg/kg body weight of a polysaccharide isolated from okra, resulted in an improved oral glucose
208 tolerance test (Zhengzheng Liao et al., 2019). Another study revealed that oral glucose absorption in 24
209 hours fasted rats reduced after okra intake (M. Khatun, Rahman, Biswas, & Islam, 2010). In the present
210 study, a large reduction was observed in HOMA-IR and QUICKI; however, the decrease in fasting
211 insulin levels did not reach significance. A study conducted by Fan et al. (Fan et al., 2014) showed that
212 okra can significantly improve both insulin levels and HOMA-IR. It is conceivable that the discrepancy
213 in our findings, vs. that of previous significant findings in insulin concentrations, are attributable to the
214 model (human vs rodent) and varying okra intake. The quantity of okra used in the animal study was
215 nearly 30g per body weight per day, which is equal to 1500 g/day okra consumption in a 50-kg person;
216 which is drastically higher than the amount that used in this study (10 g/day), and indeed infeasible to
217 be consumed in such quantities in humans. In addition to beneficial effects of okra on T2D; glycemic

and lipid metabolic markers are shown to be improved in gestational diabetic rats (Z.-H. Tian et al., 2015).

The mechanism mediating the influence of okra in regulating glycemic status relevant markers is not well-understood. Okra may improve glucose tolerance and insulin sensitivity in T2D through ameliorating insulin signaling and increasing glucose utilization, as well as suppressing oxidative stress (L.-Y. Wang et al., 2012). It has also been demonstrated that okra polysaccharide can downregulate glucose and lipid levels through preventing PPAR signaling and liver X receptors (Wu, Shi, Wang, & Wang, 2016). In addition, okra contains abscisic acid, which has a hypoglycemic effect and might be capable of improving blood glucose levels (Daliu, Annunziata, Tenore, & Santini, 2019). Moreover, the anti-diabetic activity of okra could be attributed to its' bioactive components, which might contribute to inhibiting pancreatic islet cell apoptosis (Y. Zhang et al., 2014), suppressing α-amylase and α-glucosidase (Cui, Gu, Wang, Ouyang, & Wang, 2015; J.-L. Hu, Nie, Li, & Xie, 2013), anti-inflammatory activity (J. Hu, Pang, Bai, Zheng, & Wu, 2013), activating the cAMP-PKA pathway (H. Wang, Shi, Bao, Li, & Wang, 2015), insulin enhancement (Ren et al., 2015), or even through pathways independent from insulin such as decreasing glucose uptake (Tang, Chen, Wang, & Sun, 2015), and downregulating glycogen phosphorylase mRNA expression (Xiao et al., 2012).

The present study demonstrated a significant improvement in TG, TC, and LDL-C following 8-weeks okra administration. In line with our results, Ngoc et al. (Ngoc, Ngoc, Tran, & Phung, 2008) reported that okra, administered in doses of 30 g/kg body weight, decreased TC and TG concentration in hyperlipidemia induced mice. Another study indicated that okra (in ethanol extract form) can improve serum lipid levels in diet induced obese mice by its flavonoids, isoquercitrin and quercetin 3-O-gentiobioside (Fan et al., 2014). The lipid management effect of okra might be attributed to decreases in lipid peroxidation through binding with bile-acid, prevention of bile acid reabsorption, and reducing liver cholesterol biosynthesis (Kahlon, Chapman, & Smith, 2007; Ngoc et al., 2008). The flavonoid content of okra can suppress the expression of nuclear receptor transcription factor PPARγ, which acts as an important regulator in lipid and glucose homeostasis, respectively (Fan et al., 2014). In addition, the active components in okra can also reduce ROS and MDA, through enhancing the levels of SOD,

GSH-Px, and CAT, which are considered the most important enzymes involved with the enzymatic antioxidant system (Wu et al., 2016).

No adverse side-effects related with okra consumption were reported by patients throughout the duration of the study. Okra is a generally safe food which is widely used in cooking, although it is considered safe when used in food, there is lack of knowledge on the contraindication of okra intake in critically ill patients; therefore, it should be used with caution in clinical environments. Okra intake, when concurrent with metformin, might reduce the metformin absorption, thus, the food-drug interaction should be taken into account when okra is considered as an adjuvant treatment along with conventional pharmacological remedy (H. Khatun, Rahman, Biswas, & Islam, 2011). In addition, further studies are needed to examine minimum and maximum amount of okra needed to obtain optimal results on metabolic outcomes.

To the best of the authors knowledge, the present study is the first clinical trial investigating the effect of okra on metabolic parameters in T2D adults. However, this study has several limitations which should be considered. First, the duration of study was relatively short, and it is possible that 8-weeks administration was not sufficient to yield significant beneficial effects in some variables, such as HbA1C and fasting insulin levels. However, even in this short duration, we could demonstrate a positive influence on lipid profiles and glycemic markers, which represents a promising finding for the treatment of T2D. Second, we were not able to independently analyze the all okra active components and only its main nutrients were analyzed; however, the all doses of okra were obtained from the same medical herb company, in batch form. The amount of bioactive components in okra, like other plant foods and herbs, are affected by the condition, storage, and cultivar, and therefore, these potential discrepancies must be considered. Third, although we tried to limit the effect of confounders, such as diet, it is possible that there were other variables, out of the control of the study, which may have influenced the results.

**Conclusion**:

The present study suggests that okra consumption can improve lipid profiles, as well as insulin resistance and FPG concentration, among T2D patients. Okra consumption appears safe, with several

therapeutic properties, with no reports of adverse effects in this study. Given these promising results, it is evident that okra may represent a viable adjuvant therapy in the treatment or management of T2D; however, further work is required to affirm the veracity of our findings.

**Authorship**:

A. Moradi and S. M. Safavi equally contributed to the conception and design of the research; M. Shafiepour and M. J. Tarrahi contributed to the design of the research; A. Moradi contributed to the acquisition and analysis of the data; M. J. Tarrahi contributed to the interpretation of the data; A. Moradi, S. Ghasempour and C. Clark drafted the manuscript. All authors critically revised the manuscript, agree to be fully accountable for ensuring the integrity and accuracy of the work, and read and approved the final manuscript.

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**Conflict of interest**:

The authors declare no conflict of interest

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**Table 1.** Okra powder nutrient components per 100 g

| **Nutrients** | **Amount** |
|---|---|
| Moisture | 8.46 g |
| Carbohydrate | 50.24 g |
| Protein | 18.25 g |
| Ash | 10.57 g |
| Crude Fiber | 12.97 g |
| Fat | 2.49 g |
| Flavonoid | 2.6 mg |
| Phenol | 11.2 mg |

ndent samples t-test (2-tailed).
-Abbreviations; BMI: Body Mass Index, F: Female, M: Male; HbA1C: Glycated hemoglobin; MET: Metabolic Equivalent of Task.

| riables | Intervention group (N=30) | Control group (N=30) | P-value |
|---|---|---|---|
| e (year) | 54.26±7.62 | 53.33±7.35 | 0.63 |
| (M/F) | 9/21 | 7/23 | 0.78 |
| ly weight (kg) | 71.46±11.33 | 72.80±10.63 | 0.64 |
| II ($kg/m^2$) | 24.90±3.94 | 25.65±3.46 | 0.30 |
| ration of disease | 4.68±2.07 | 3.96±2.83 | 0.26 |
| G (mg/dl) | 182.54±51.11 | 183.36±58.43 | 0.94 |
| A1C (%) | 7.99±0.87 | 7.62±1.39 | 0.22 |
| tolic blood pressure (mm Hg) | 119.34±9.80 | 121.43±11.05 | 0.46 |
| stolic blood pressure (mm Hg) | 80±7.42 | 76±7.47 | 0.10 |
| sical activity (MET-h/day) at baseline | 30.91±6.88 | 29.13±5.86 | 0.28 |
| sical activity (MET-h/day) at end of trial | 31.57±5.72 | 29.68±4.73 | 0.16 |

**Table 3.** Dietary intakes of study participants throughout the study.

| Variables | | Intervention group (N=35) | Control group (N=35) | P-value |
|---|---|---|---|---|
| **Energy (Kcal/day)** | **Before** | 2316±281 | 2274±314 | 0.58 |
| | **After** | 2188±274 | 2146±265 | 0.54 |
| **Carbohydrate (gr/day)** | **Before** | 334±84 | 297±101 | 0.12 |
| | **After** | 309±79 | 282±76 | 0.18 |
| **Protein (gr/day)** | **Before** | 81.1±11.7 | 78.2±10.7 | 0.30 |
| | **After** | 82.7±12.3 | 79.6±11.4 | 0.31 |
| **Fat (gr/day)** | **Before** | 68.7±10.6 | 70.8±10.7 | 0.44 |
| | **After** | 66.3±9.8 | 68.2±9.6 | 0.45 |
| **MUFA (gr/day)** | **Before** | 21.5±5.1 | 22.2±6.4 | 0.64 |
| | **After** | 21.2±4.6 | 21.8±5.9 | 0.63 |
| **PUFA (gr/day)** | **Before** | 18.1±7.4 | 17.4±6.9 | 0.70 |
| | **After** | 19.7±5.8 | 19.1±5.5 | 0.68 |
| **Vitamin E (mg/day)** | **Before** | 6.8±3.1 | 6.3±3.9 | 0.58 |
| | **After** | 6.7±3.7 | 6.4±3.7 | 0.75 |
| **Zinc (mg/day)** | **Before** | 9.7±1.8 | 9.1±2.5 | 0.34 |
| | **After** | 9.8±1.8 | 9.4±2.2 | 0.49 |
| **Vitamin C (mg/day)** | **Before** | 117±85 | 106±92 | 0.66 |
| | **After** | 113±73 | 109±88 | 0.86 |
| **β carotene (µg/day)** | **Before** | 4521±3570 | 4260±4627 | 0.82 |
| | **After** | 4832±3604 | 4661±3688 | 0.87 |
| **Dietary fiber (g/day)** | **Before** | 12.24±4.16 | 11.83±5.31 | 0.74 |
| | **After** | 12.57±4.29 | 12.14±4.20 | 0.69 |

MUFA: Mono-unsaturated fatty acid; PUFA: Poly-unsaturated fatty acid.

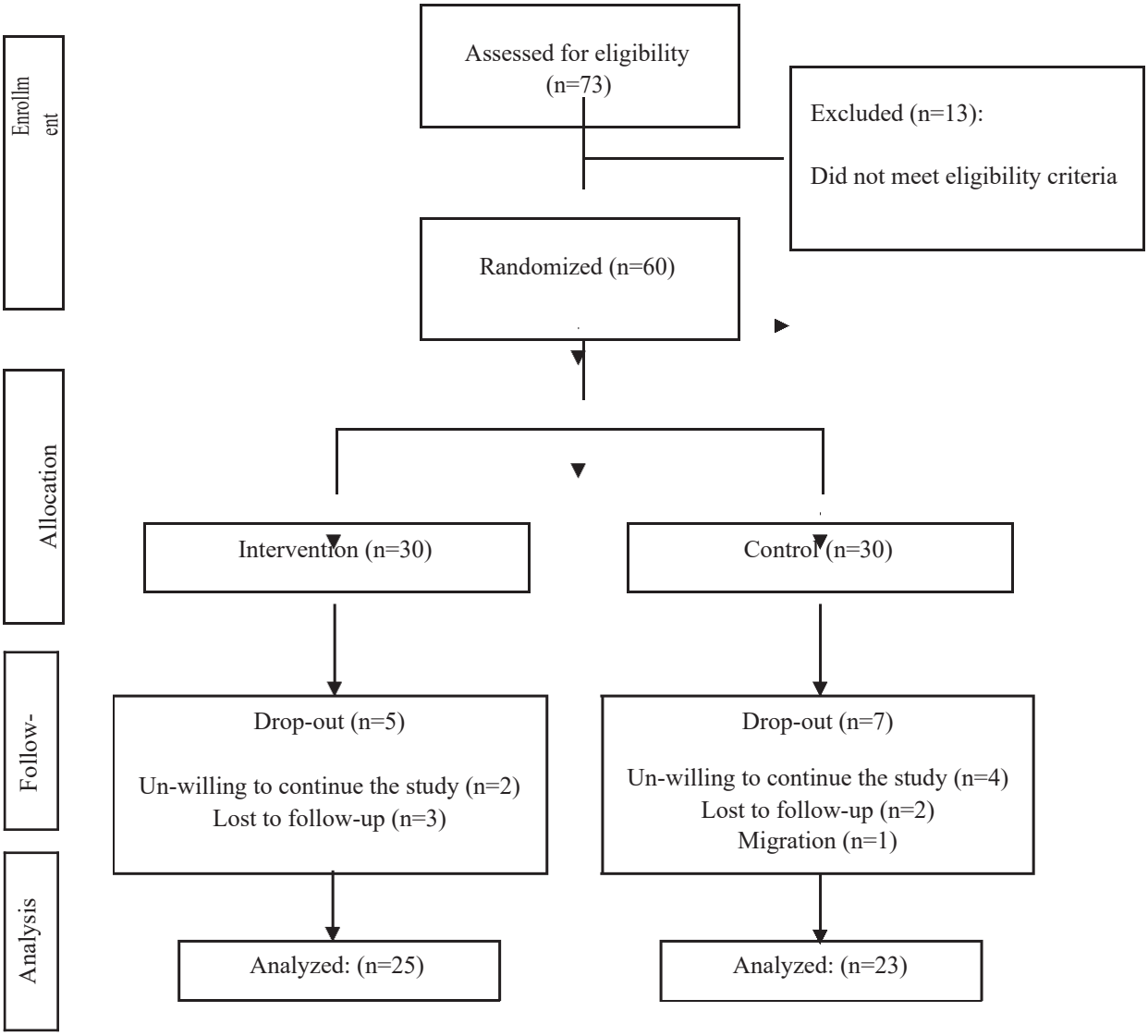
| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| **Glucose (mg/dl)** | | | | | | | | | |
| **HbA1C (%)** | 7.99±0.87 | 7.83±0.92 | -0.16±0.77 | 0.21 | 7.62±1.39 | 7.47±1.28 | -0.14±1.12 | 0.48 | 0.55 |
| **Fasting Insulin (µU/ml)** | 19.68±6.54 | 19.02±6.15 | -0.66±1.98 | 0.08 | 19.31±5.03 | 19.29±4.40 | -0.02±1.65 | 0.92 | 0.23 |
| **HOMA-IR** | 8.91±3.87 | 7.74±3.28 | -1.17±1.61 | <0.001 | 8.66±3.74 | 8.52±3.13 | -0.14±1.64 | 0.63 | 0.01 |
| **QUICKI** | 0.27±0.01 | 0.28±0.01 | 0.01±0.007 | 0.002 | 0.28±0.03 | 0.28±0.02 | 0.00±0.01 | 0.83 | 0.004 |

Data are presented as mean ± SD. * P-value was obtained by Paired t-test. ** P-value was obtained by ANCOVA adjusted for age, sex, baseline energy intake, participant's weight and physical activity.

Abbreviations: BMI: Body mass index; LDL-C: low density lipoprotein cholesterol; HDL-C: low density lipoprotein cholesterol; HbA1C: Glycated hemoglobin; HOMA-IR: Homeostasis Model Assessment for Insulin Resistance; QUICKI: Quantitative insulin sensitivity check index



| Variables | Okra group | | | | Placebo | | | | P-value** |
|---|---|---|---|---|---|---|---|---|---|
| | Baseline | After 8 weeks | Change | P-value * | Baseline | After 8 weeks | Change | P-value * | |
| **ody Weight (kg)** | 71.46±11.33 | 70.96±10.04 | -0.50±2.04 | 0.17 | 72.80±10.63 | 72.76±9.93 | -0.03±1.44 | 0.89 | 0.24 |
| **BMI (kg/m$^2$)** | 24.90±3.94 | 24.72±3.49 | -0.17±0.95 | 0.28 | 25.65±3.46 | 25.64±3.50 | -0.01±0.72 | 0.90 | 0.29 |
| **Triacylglycerol (mg/dl)** | 187.53±61.84 | 165.23±54.13 | -22.30±32.46 | 0.001 | 205.20±61.41 | 201.33±46.84 | -3.86±30.57 | 0.50 | 0.001 |
| **otal cholesterol (mg/dl)** | 191.00±38.47 | 180.76±35.76 | -10.23±10.36 | <0.001 | 203.80±42.69 | 201.76±39.67 | -2.03±13.94 | 0.43 | 0.004 |
| **LDL-C (mg/dl)** | 125.44±34.22 | 117.28±30.66 | -8.15±10.01 | <0.001 | 128.64±44.35 | 126.32±42.95 | -2.31±9.37 | 0.19 | 0.02 |
| **HDL-C (mg/dl)** | 39.16±8.31 | 39.50±7.70 | 0.34±1.09 | 0.11 | 40.70±6.88 | 41.01±6.06 | 0.31±2.32 | 0.49 | 0.76 |
| **L-C/HDL-C ratio** | 3.35±1.15 | 3.07±0.94 | -0.28±0.37 | <0.001 | 3.22±1.14 | 3.14±1.13 | -0.08±0.24 | 0.10 | 0.01 |
| **Systolic blood** | | | | | | | | | |


Enrollment
Allocation
Follow-
Analysis
Assessed for eligibility (n=73)
Excluded (n=13):
Did not meet eligibility criteria
Randomized (n=60)
Intervention (n=30)
Control (n=30)
Drop-out (n=5)
Un-willing to continue the study (n=2)
Lost to follow-up (n=3)
Drop-out (n=7)
Un-willing to continue the study (n=4)
Lost to follow-up (n=2)
Migration (n=1)
Analyzed: (n=25)
Analyzed: (n=23)

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