Beneficial Effects of Okra (*Abelmoschus esculentus L.*) Consumption on Anthropometric Measures, Blood Pressure, Glycemic Control, Lipid Profile, and Liver Function Tests in Randomized Controlled Trials: A GRADE-Assessed Systematic Review and Dose-Response Meta-Analysis

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#### ABSTRACT

This review aimed to assess the impact of okra (Abelmoschus esculentus L.) consumption on cardiovascular disease risk factors. Relevant studies were identified through electronic searches of databases including PubMed, Scopus, Web of Science, CENTRAL, and EMBASE up to January 2025. Twelve trials involving 770 participants with interventions ranging from 2 to 12 weeks and doses varying from 125 to 40,000 mg/day, were included. Okra supplementation significantly reduced body mass index (BMI) (Standardized Mean Difference [SMD] = -0.70; 95% Confidence Interval [CI]: -1.23, -0.16; P = 0.011), fat mass (FM) (SMD = -0.74; 95% CI: -1.13, -0.36; P < 0.001), hip circumference (HC) (SMD = -0.85; 95% CI: -1.41, -0.28; P = 0.003), weight (SMD = -0.77; 95% CI: -1.42, -0.11; P = 0.022), fasting insulin (SMD = -0.35; 95% CI: -0.63, -0.07; P = 0.013), fasting plasma glucose (FPG) (SMD = -1.07; 95% CI: -1.75, -0.38; P =0.002), hemoglobin A1c (HbA1c) (SMD = -0.38; 95% CI: -0.71, -0.05; P = 0.023), homeostatic model assessment of insulin resistance (HOMA-IR) (SMD = -0.56; 95% CI: -0.84, -0.29; P < 0.001), low-density lipoprotein cholesterol (LDL-C) (SMD = -0.32; 95% CI: -0.52, -0.11; P = 0.003), total cholesterol (TC) (SMD = -0.45; 95% CI: -0.74, -0.16; P = 0.003), and aspartate aminotransferase (AST) (SMD = -0.45; 95% CI: -0.73, -0.17; P = 0.002). Okra supplementation demonstrated significant benefits in improving anthropometric measures, glycemic control, lipid profiles, and liver function tests, suggesting its potential as an adjunct therapy for improving cardiovascular disease risk factors.

**Keywords:** okra; Abelmoschus; anthropometric indices; lipid profile; glycemic indices; liver function

#### **1 INTRODUCTION**

Cardiovascular and metabolic diseases, including obesity, dyslipidemia, diabetes, and hypertension, represent significant global health challenges and contribute to substantial morbidity and mortality worldwide (1). In 2021 alone, these conditions accounted for more than one-third of all deaths globally, underscoring the urgency of effective prevention and management strategies (2). Among the various approaches to mitigate these risks, dietary interventions have gained particular attention for their potential to improve cardiovascular health and metabolic outcomes (3, 4).

Functional foods and medicinal plants are emerging as promising tools in dietary strategies (5). Okra (*Abelmoschus esculentus L.*), a member of the mallow family, has garnered interest due to its rich bioactive profile, including mucilage, flavonoids, polyphenols, fiber, vitamins, and minerals (6, 7). These components are associated with a variety of health benefits, such as antioxidant properties, improved glycemic control, lipid regulation, and enhanced liver function (8). Particularly noteworthy is okra's high content of soluble fiber, which has been shown to lower cholesterol levels and improve postprandial glycemic responses (9).

Recent evidence suggests that okra supplementation may positively influence metabolic and cardiovascular health outcomes (8), including glycemic control (10), lipid profiles (11), blood pressure (12), and liver function (13). However, while its effects on fasting blood glucose and lipid metabolism have been extensively studied, data on its impact on anthropometric measures and blood pressure remain scarce (14, 15). Furthermore, systematic reviews and meta-analyses to date have primarily focused on glycemic control and inflammation markers, leaving a critical gap in understanding okra's comprehensive effects on metabolic and cardiovascular parameters (16, 17, 18).

To address this gap, we conducted a systematic review and dose-response meta-analysis of randomized controlled trials (RCTs) using the GRADE approach. Our study represents the first effort to evaluate the impact of okra supplementation across a wide range of cardiovascular and metabolic outcomes. By employing advanced statistical techniques such as meta-regression and dose-response analysis, we investigated the relationship between okra dosage, duration, and its health effects across diverse populations, including variations in age, gender, and health status.

This review aims to provide robust, evidence-based insights into the health benefits of okra. By offering a thorough analysis of current data, we hope to inform dietary recommendations and highlight the potential of okra as a complementary intervention in managing metabolic and cardiovascular disorders. Ultimately, our findings seek to contribute to public health efforts, guiding clinicians and policymakers in leveraging the therapeutic potential of okra to improve overall health and quality of life.

#### **2 METHODS**

#### 2.1 Protocol and Registration:

This study was conducted according to a pre-established methodology, adhering to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2015 guidelines (19) (**Supplementary Table 1**). To ensure transparency and uphold high-quality standards, the systematic review and meta-analysis were registered with the International Prospective Register of Systematic Reviews (PROSPERO) under the registration code CRD42024576026.

#### 2.2 Search Strategy and Study Selection:

A comprehensive literature search was conducted across multiple electronic databases, including PubMed, Web of Science, Cochrane Central Register of Controlled Trials (CENTRAL), EMBASE, and SCOPUS, for articles published up to August 2024, with an update in January 2025. Additional searches were performed in ScienceDirect and Google Scholar. Additional studies were identified through manual searches of reference lists from relevant articles, reviews, and reputable journals. Additionally, grey literature was searched through sources such as ProQuest Dissertations and Theses, OpenGrey, and conference proceedings to identify any unpublished studies relevant to the topic. The search aimed to identify studies that assessed the effects of okra consumption on anthropometric measures, blood pressure, glycemic control, lipid profiles, and liver function tests.

A tailored search strategy was employed for each database to capture relevant studies, using key terms such as 'okra,' '*Abelmoschus esculentus*,' and 'clinical trial,' based on EMTREE and Medical Subject Headings (MeSH) tags (**Supplementary Table 2**).

#### 2.3 Inclusion/Exclusion Criteria and Data Extraction:

Studies were included if they met the following criteria: randomized controlled trials (RCTs) involving human adults, with either single-blind or double-blind designs, the presence of control groups, and publication in English. Exclusion criteria included non-RCT studies, animal or in vitro research, review articles, case studies, observational studies, editorials, commentaries, letters, and studies that did not report sufficient data on the primary outcomes.

Two reviewers (A.J. and A.A.) independently screened the titles and abstracts of all identified articles to determine their eligibility. Full-text reviews were conducted for studies meeting the initial inclusion criteria. Disagreements between the reviewers were resolved through discussion, and when necessary, a third reviewer (A.S.) was consulted to reach a consensus.

Data management was facilitated using EndNote X7 software, which was employed to combine search results and eliminate duplicates. Data extraction was conducted using a standardized form that recorded details such as author information, study year, country, study design, population characteristics, intervention specifics, and outcome measures.

#### 2.4 Methodological Quality Assessment and Evaluation of the Strength of Evidence:

The methodological quality of the included studies was assessed using the Cochrane risk-of-bias tool. This evaluation was independently performed by two reviewers (A.J. and H.M.), with any discrepancies resolved through discussion or, if needed, the involvement of a third reviewer (A.S.). The assessment criteria focused on sequence generation, allocation concealment, blinding, management of incomplete data, selective reporting, and other potential sources of bias.

The strength of evidence for each outcome was assessed using the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) framework (20). This approach categorizes evidence into four levels: high, moderate, low, or very low. Two independent reviewers (H.M. and B.P.) conducted this assessment, considering factors such as study design, risk of bias, consistency, precision, directness, and the potential for publication bias. A third author (A.J.) was available to adjudicate any disagreements.

#### 2.5 Statistical Analysis:

Statistical analyses were conducted using Stata software, version 15.0 (StataCorp, College Station, Texas). The primary objective was to analyze data from the included studies to evaluate the effects of okra consumption on various health outcomes. We systematically extracted preand post-intervention means, standard deviations, and sample sizes for each outcome from the randomized controlled trials (RCTs) included in the meta-analysis. Key outcomes assessed included anthropometric measures, blood pressure, glycemic control, lipid profiles, and liver function tests. In cases where means and standard deviations were not directly reported, we derived them from available data or contacted the study authors for missing information.

To quantify the effect of okra consumption, we calculated standardized mean differences (SMDs) between the intervention and control groups for each outcome. The SMD approach was chosen to standardize results across studies with varying measurement scales (21). We calculated 95% confidence intervals (CIs) for each outcome to assess the precision of the estimated effect sizes (21). Given the anticipated variability among the included studies, a random-effects model was applied using the DerSimonian-Laird method, accounting for both within-study and between-study variability to provide a more generalized estimate of the effect (22). Heterogeneity was assessed using the I<sup>2</sup> statistic, with values of 25%, 50%, and 75% indicating low, moderate, and high heterogeneity, respectively (23).

To identify potential sources of heterogeneity, subgroup analyses were conducted based on factors such as geographical location, baseline health status, okra dosage, age groups, baseline body mass index (BMI), intervention duration, and sample size. Meta-regression analyses were also performed to explore the influence of okra dosage and duration on cardiovascular and metabolic risk factors, aiming to identify dose-response relationships and the effects of varying supplementation periods on health outcomes (24). A nonlinear model was employed to examine the dose-response relationship between okra supplementation and health outcomes (25), providing insights into how different dosages and durations impact results and identifying any optimal dose for maximum benefit.

An influence analysis was conducted to determine the impact of individual studies on the overall effect size by assessing the consistency of results when specific studies were excluded. Publication bias was evaluated through visual inspection of funnel plots and statistical tests,

including Egger's and Begg's tests, to determine if the observed results might be influenced by selective publication of studies with positive outcomes (26).

### **3 RESULTS**

#### 3.1 Study Selection

The selection process of the included studies is outlined in **Figure 1**. A total of 2,458 studies were identified through database searches, including PubMed (n = 146), ISI Web of Science (n = 578), Scopus (n = 873), Embase (n = 789), and Cochrane Library (n = 72). After removing 728 duplicates, 198 irrelevant studies and 144 animal studies, 1,388 studies remained for title and abstract screening. Of these, 1,356 studies were excluded due to irrelevance, leaving 32 full-text studies for further evaluation. Ultimately, 5 studies were excluded due to reporting non-relevant outcomes. **Supplementary Table 3** provides details on the studies excluded after full-text review and the reasons for their exclusion. As a result, 12 studies involving 770 participants were included in the systematic review and meta-analysis (10, 11, 12, 13, 14, 15, 27, 28, 29, 30, 31, 32).

#### 3.2 Study Characteristics

The characteristics of the included studies are summarized in **Table 1**. The SMDs and 95% CIs for BMI, fat-free mass (FFM), fat mass (FM), hip circumference (HC), waist circumference (WC), body weight, diastolic blood pressure (DBP), systolic blood pressure (SBP), fasting blood insulin, fasting blood sugar (FBS), hemoglobin A1c (HbA1c), homeostatic model assessment of insulin resistance (HOMA-IR), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), total cholesterol (TC), triglycerides (TG), alkaline phosphatase (ALP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and creatinine, along with their changes, are presented in **Figures 2-6**. The studies were published between 2019 and 2024 and were conducted in Iran, Germany, Indonesia, and China. Participants in the intervention group had a mean age ranging from 21.7 to 62 years. The dosage of okra administered varied from 125 mg/day to 40,000 mg/day, with intervention durations ranging from 2 to 12 weeks. The sample size in the intervention groups ranged from 10 to 50 participants. One study included only female participants (31), while the others included both

genders. The study populations comprised individuals with diabetic nephropathy (11, 29), overweight and moderately obese individuals (15, 30), prediabetes (13), Type 2 Diabetes Mellitus (T2DM) (12, 14, 28), healthy individuals (32), gestational diabetes mellitus (31), impaired glucose tolerance (IGT) (10), and T2DM with hypercholesterolemia (27) subjects.

Sample sizes for the intervention and control groups were as follows: BMI = 465 (intervention: 258, control: 207), FFM = 212 (intervention: 130, control: 82), FM = 212 (intervention: 130, control: 82), HC = 212 (intervention: 130, control: 82), WC = 311 (intervention: 180, control: 131), weight = 327 (intervention: 190, control: 137), DBP = 308 (intervention: 158, control: 150), SBP = 308 (intervention: 158, control: 150), fasting insulin = 214 (intervention: 108, control: 106), FBS = 535 (intervention: 292, control: 243), HbA1c = 415 (intervention: 228, control: 187), HOMA-IR = 214 (intervention: 108, control: 106), HDL-C = 540 (intervention: 293, control: 247), LDL-C = 540 (intervention: 293, control: 247), TG = 540 (intervention: 293, control: 247), ALP = 266 (intervention: 153, control: 113), ALT = 425 (intervention: 233, control: 192), AST = 365 (intervention: 203, control: 162), and creatinine = 326 (intervention: 183, control: 143).

#### 3.3 Qualitative Data Assessment

According to the Cochrane Risk of Bias Assessment tool, all studies were assessed as having a high risk of bias (10, 11, 12, 13, 14, 15, 27, 28, 29, 30, 31, 32) (**Table 2**).

#### 3.4 Effects of Okra Supplement on Anthropometric Measures

Okra supplementation demonstrated significant effects on BMI (SMD = -0.70; 95CI%: -1.23, -0.16; P = 0.011;  $I^2$  = 86.5%, P < 0.001) (**Figure 2**), FM (SMD = -0.74; 95CI%: -1.13, -0.36; P < 0.001;  $I^2$  = 43.8%, P = 0.149), HC (SMD = -0.85; 95CI%: -1.41, -0.28; P = 0.003;  $I^2$  = 72.4%, P = 0.012), and weight (SMD = -0.77; 95CI%: -1.42, -0.11; P = 0.022;  $I^2$  = 87.0%, P < 0.001). However, no significant changes were observed in FFM (SMD = -0.13; 95CI%: -0.41, 0.15; P = 0.349;  $I^2$  = 0.0%, P = 0.679) or WC (SMD = -0.57; 95CI%: -1.15, 0.01; P = 0.054;  $I^2$  = 82.8%, P < 0.001).

Sensitivity analyses for BMI, FFM, FM, and HC revealed that excluding any of the studies did not alter the overall findings. However, exclusion of the studies by Nikpayam et al. (29) (SMD = -0.73, 95% CI: -1.42, -0.05) and Saatchi et al. (14) (SMD= -0.75, 95% CI: -1.37, -0.13) notably

altered the effect on WC. Similarly, excluding the studies by Uebelhack et al. (15) (SMD = -0.73, 95% CI: -1.51, 0.05), Uebelhack et al. (15) (SMD = -0.58, 95% CI: -1.24, 0.08) and Peng et al. (30) (SMD: -0.54, 95% CI: -1.14, 0.54) significantly changed the effect on weight. No evidence of publication bias was detected for FFM (Egger's P = 0.859), FM (Egger's P = 0.257), HC (Egger's P = 0.193), and WC (Egger's P = 0.094). However, Egger's test indicated significant asymmetry for BMI (Egger's P = 0.020) and weight (Egger's P = 0.009).

## 3.5 Effects of Okra Supplement on Blood Pressure

Studies investigating the effects on DBP and SBP, showed no significant reductions, with results for DBP, (SMD = -0.15; 95% CI: -0.37, 0.08; P = 0.195;  $I^2 = 0.0\%$ , P = 0.966) (**Figure 3**), and SBP (SMD = -0.13; 95% CI: -0.56, 0.30; P = 0.551;  $I^2 = 71.9\%$ , P = 0.014), respectively.

Sensitivity analyses for DBP and SBP indicated that excluding any of the studies did not alter the overall findings. No evidence of publication bias was found for DBP (Egger's P = 0.661) and SBP (Egger's P = 0.173).

## 3.6 Effects of Okra Supplement on Glycemic Profile

Significant improvements were observed across all indices of this group, including fasting insulin (SMD = -0.35; 95% CI: -0.63, -0.07; P = 0.013; I<sup>2</sup> = 4.6%, P = 0.351) (**Figure 4**), FBS (SMD = -1.07; 95% CI: -1.75, -0.38; P = 0.002; I<sup>2</sup> = 91.7%, P < 0.001), HbA1c (SMD = -0.38; 95% CI: -0.71, -0.05; P = 0.023; I<sup>2</sup> = 61.7%, P = 0.023), and HOMA-IR (SMD = -0.56; 95% CI: -0.84, -0.29; P < 0.001; I<sup>2</sup> = 0.0%, P = 0.858).

Sensitivity analyses for FBS and HOMA-IR indicated that excluding any of the studies did not change the overall conclusions. However, excluding the studies by Moradi et al. (28) (SMD = -0.38, 95% CI: -0.85, 0.10) and Chen et al. (10) (SMD= -0.24, 95% CI: -0.55, 0.08) significantly changed the effect on fasting insulin. Similarly, excluding the studies by Tavakolizadeh et al. (12) (SMD = -0.30, 95% CI: -0.69, 0.84), Saatchi et al. (14) (SMD = -0.27, 95% CI: -0.56, 0.03) and Moradi et al. (28) (SMD: -0.39, 95% CI: -0.78, 0.01) significantly altered the effect on HbA1c. No evidence of publication bias was detected for fasting insulin (Egger's P = 0.333) and FBS (Egger's P = 0.475). However, significant asymmetry was observed for HbA1c (Egger's P = 0.030) and HOMA-IR (Egger's P = 0.006).

## 3.7 Effects of Okra Supplement on Lipid Profile

Okra supplementation significantly reduced LDL-C (SMD = -0.32; 95% CI: -0.52, -0.11; P = 0.003;  $I^2 = 29.4\%$ , P = 0.193) (**Figure 5**) and TC (SMD = -0.45; 95% CI: -0.74, -0.16; P = 0.003;  $I^2 = 63.3\%$ , P = 0.008). In contrast, it did not result in significant changes in HDL-C (SMD = 0.13; 95% CI: -0.15, 0.41; P = 0.354;  $I^2 = 59.7\%$ , P = 0.015) or TG (SMD = -0.24; 95% CI: -0.50, 0.02; P = 0.069;  $I^2 = 53.5\%$ , P = 0.035).

Sensitivity analyses for HDL-C, LDL-C, and TC confirmed that excluding any study did not alter the overall findings. However, excluding studies by Saatchi et al. (14) (SMD = -0.35, 95% CI: -0.54, -0.16) and Chen et al. (10) (SMD= -0.29, 95% CI: -0.57, -0.01) notably changed the overall effect on TG. No evidence of publication bias was found for HDL-C (Egger's P = 0.620), LDL-C (Egger's P = 0.921), TC (Egger's P = 0.730), or TG (Egger's P = 0.415).

### 3.8 Effects of Okra Supplement on Liver Function Tests

AST levels were the only parameter significantly affected by okra supplementation in this group (SMD = -0.45; 95% CI: -0.73, -0.17; P = 0.002; I<sup>2</sup> = 39.4%, P = 0.159) (**Figure 6**). However, no substantial changes were observed in ALP (SMD = 0.03; 95% CI: -0.28, 0.34; P = 0.834; I<sup>2</sup> = 33.8%, P = 0.209), ALT (SMD = -0.29; 95% CI: -0.71, 0.12; P = 0.164; I<sup>2</sup> = 76.3%, P = 0.001), and creatinine levels (SMD = -0.12; 95% CI: -0.36, 0.12; P = 0.327; I<sup>2</sup> = 10.6%, P = 0.346).

Sensitivity analysis conducted for ALP, ALT, AST, and creatinine revealed that excluding any of the studies did not alter the overall findings. No publication bias was detected for ALP (Egger's P = 0.198), ALT (Egger's P = 0.485), AST (Egger's P = 0.588), or creatinine (Egger's P = 0.146).

## 3.9 Subgroup Analysis:

Subgroup analyses based on country (Iran vs. other countries such as China, Germany, and Indonesia), health status (prediabetic or diabetic vs. non-diabetic), age ( $\leq$ 50 years vs. >50 years), baseline BMI (healthy weight [ $\leq$ 25 kg/m<sup>2</sup>] vs. overweight or obese [>25 kg/m<sup>2</sup>]), intervention duration ( $\leq$ 8 weeks vs. >8 weeks), dosage ( $\leq$ 2000 mg/day vs. >2000 mg/day), and sample size (<60 vs.  $\geq$ 60 participants) are summarized in **Table 3**.

Okra supplementation showed greater benefits for FBS, HbA1c, LDL-C, TC, and AST in studies conducted in Iran. In contrast, BMI, WC, and weight were more significantly affected in studies

from other countries. No substantial effects on HDL-C, TG, ALP, or creatinine levels were observed based on country.

For health status, okra supplementation had a more pronounced effect on FBS, HbA1c, LDL-C, TC, and AST in prediabetic or diabetic individuals. In contrast, BMI, WC, and weight were significantly affected in non-diabetic individuals. No significant effects on HDL-C, TG, ALT, or creatinine were observed in either group.

Subjects younger than 50 years showed significant changes in BMI, WC, weight, and AST, while those aged 50 and above had significant reductions in FBS, HbA1c, LDL-C, TC, and creatinine. No significant effects were observed in either age group for HDL-C, TG, or ALT.

For baseline BMI, okra supplementation impacted overweight or obese individuals' BMI, HbA1c, weight, and AST, while significant changes in FBS, LDL-C, TC, and TG were observed in both healthy weight and overweight/obese groups. No significant effects were observed for WC, HDL-C, ALT, or creatinine in either BMI category.

Okra supplementation showed a significant impact on FBS and AST in interventions lasting  $\leq 8$  weeks, while BMI, WC, weight, LDL-C, TC, and TG were more significantly affected in studies lasting >8 weeks. No significant effects on HbA1c, HDL-C, ALT, or creatinine were observed in either duration category.

For dosages  $\leq 2000 \text{ mg/day}$ , significant effects were seen in BMI, WC, weight, and TG. In contrast, doses > 2000 mg/day significantly impacted FBS, HbA1c, LDL-C, and AST. Significant effects on TC were observed in both dosage groups, but no significant effects were seen for HDL-C, ALT, or creatinine.

In terms of sample size, okra supplementation significantly impacted BMI, WC, weight, and TG in studies with <60 participants, while studies with  $\geq$ 60 participants showed significant effects on FBS, HbA1c, LDL-C, and AST. Significant effects on TC were observed in both sample size categories, but no significant effects were seen for HDL-C, ALT, or creatinine.

## 3.10 Meta-regression and Non-linear Dose-Response Analysis

Meta-regression analysis assessing the impact of okra doses and intervention duration on cardiovascular risk variables is presented in **Supplementary Figures 1-4**. A non-linear dose-

response regression model was used to explore the relationship between okra supplementation and cardiovascular outcomes. A significant association was found between okra dose and weight reduction (Coefficient = 2.47, P < 0.001). The dose-response curve indicated that the optimal dose for weight reduction is approximately 2000 mg/day. Similarly, a significant association was observed between okra dose and HbA1c reduction (Coefficient = 0.52, P = 0.016). The doseresponse curve suggested that the optimal dose for reducing HbA1c levels is around 3000 mg/day.

## 3.11 GRADE Assessment:

The GRADE profile for the outcomes of okra supplementation is presented in **Table 4**. The quality of evidence was rated as very low for BMI, FFM, HC, WC, weight, DBP, SBP, HbA1c, HOMA-IR, HDL, TG, ALP, ALT, and creatinine, and low for FM, fasting insulin, FBS, TC, and AST. The quality of evidence for LDL-C was rated as moderate. While okra supplementation shows promise in clinical practice, further research is needed to confirm these findings across different populations.

## **4 DISCUSSION**

## 4.1 Summary of Findings:

This is the first GRADE-assessed systematic review and dose-response meta-analysis evaluating the effects of okra supplementation on cardiovascular outcomes in adults. Our analysis included 12 randomized controlled trials with 770 participants. The results indicated that okra supplementation positively affects BMI, FM, HC, weight, fasting insulin, FPG, HbA1c, HOMA-IR, LDL-C, TC, and AST.

In terms of anthropometric indices, subgroup analyses revealed a significant reduction in BMI, WC, and weight in studies conducted outside of Iran as well as studies conducted on individuals under the age of 50, without prediabetes or diabetes, with overweight or obesity, and those who received a daily dosage of less than 2000 mg of okra for more than 8 weeks.

With respect to glycemic control, significant improvements in FPG and HbA1c levels were observed in Iranians over the age of 50 with prediabetes or diabetes who received dosages of

2000 mg or more of okra. Significant decreases in LDL-C, TC, and TG were also noted in this group when given okra for over 8 weeks at doses less than 2000 mg daily. However, no changes in HDL-C levels were observed in any of the subgroups. AST levels in Iranians under the age of 50 who were overweight or obese, had prediabetes or diabetes, and took doses greater than 2000 mg daily for less than 8 weeks dropped significantly. Additionally, our investigation identified no effect of okra on blood pressure.

#### 4.2 Comparison with Previous Studies:

Our findings support the hypoglycemic effects of okra reported in earlier animal and human studies (27, 31, 33, 34). However, there are discrepancies with other studies, which may be attributed to variations in study design or sample populations. For example, Saatchi et al. reported no significant changes in HDL-C, LDL-C, TC, TG, BMI, WC, AST, ALT, SBP, or DBP after 8 weeks of consuming 4000 mg/day of whole okra fruit in T2DM subjects, despite significant reductions in HbA1c, FBS, and blood sugar levels (14). In contrast, another study found significant reductions in serum TC, LDL-C, ALT, and uric acid levels, as well as an increase in HDL-C levels after 8 weeks of administering 3000 mg of okra to prediabetic patients (35).

In contrast to our findings, Nikpayam et al. found that diabetic nephropathy patients who received 125 mg of dried okra for ten weeks experienced a decrease in energy and carbohydrate intake without affecting body composition or anthropometric measurements (29). Additionally, Peng et al. reported that supplementation with IQP-AE-103, a blend of dehydrated okra powder and inulin, resulted in changes in microbiota composition and weight loss, highlighting the synergistic effects of these ingredients (30). Studies in animals also present inconsistencies. Fan et al. observed improved glucose tolerance, decreased TG levels, and altered liver morphology in obese mice, linked to suppressed PPAR $\gamma$  nuclear receptor expression (36). Conversely, Anjani et al. found that neither green nor purple okra extract affected body weight in diabetic rats. However, both extracts were effective in repairing streptozotocin-induced pancreatic  $\beta$ -cell damage, with purple okra extract showing greater antidiabetic potency, likely due to its higher quercetin content (37).

#### 4.3 Mechanisms of Action:

Insulin resistance is a primary mechanism underlying T2DM, along with disordered glucose and lipid metabolism (38). Okra, rich in flavonoids such as ursolic acid and quercetin, may enhance glucose absorption and insulin sensitivity by regulating insulin levels (33). Additionally, okra modulates peroxisome proliferator-activated receptors (PPARs), which are crucial for lipid and glucose homeostasis in the pancreas, potentially improving FBS levels (39). Okra's inhibitory effects on  $\alpha$ -glucosidase and  $\alpha$ -amylase provide a plausible mechanism for its ability to lower fasting blood sugar levels (40). By inhibiting these carbohydrate-digesting enzymes, okra supports better blood glucose control and reduced HbA1c levels.

The high content of soluble and insoluble fiber in Abelmoschus esculentus L. promotes satiety and reduces overall calorie consumption, aiding weight loss and reducing BMI. Okra's soluble fiber binds to bile acids in the gut, potentially lowering oxidative stress and cholesterol, which are linked to lipid metabolism (41). Visceral fat, related to waist and hip circumferences, is strongly associated with insulin resistance and metabolic syndrome. Okra's hypoglycemic properties and weight-loss effects may help reduce hip and waist circumference (42).

One of okra's main polysaccharides, AeP-P-1, activates signaling molecules in the PI3K/Akt pathway in liver tissue, restoring partial kidney and liver function in type 2 diabetic mice (43). Quercetin and other polyphenols in okra protect the liver from inflammatory and oxidative stress, aiding in its normal function (44). Additionally, vitamin C in okra may chelate iron, reducing oxidative stress by limiting the availability of iron to catalyze reactive oxygen species (ROS) formation, which is a major cause of liver inflammation (44).

While the exact molecular mechanisms remain unclear, previous studies have suggested that reduced lipogenesis due to decreased expression of SREBP1 and FAS genes, enhanced cholesterol breakdown mediated by CYP7A1, reduced cholesterol absorption due to regulation of the of PPAR-NPC1L1 pathway, and interactions with bile acids. The high fiber content of okra plays a critical role in these processes. Soluble fiber binds cholesterol in the intestines, preventing its absorption into the bloodstream (8). Additionally, okra's flavonoids and phytosterols can prevent oxidative damage to lipids, thereby reducing atherosclerosis risk. Due to its antioxidant properties and ability to lower lipid levels, okra benefits cardiovascular health (45).

Beyond the established mechanisms, okra's therapeutic effects likely involve complex interactions with multiple physiological pathways. Recent evidence suggests that okra's bioactive compounds modulate inflammatory markers, including reduction in pro-inflammatory cytokines such as TNF- $\alpha$ , IL-6, and IL-1 $\beta$ , while enhancing anti-inflammatory mediators (46). The gut microbiota plays a crucial role, as okra's prebiotic components, particularly its rich mucilage content, promote the growth of beneficial bacteria like Bifidobacterium and Lactobacillus species, enhancing metabolic health through improved gut barrier function and reduced inflammation (47). Hormonal influences extend beyond insulin regulation, affecting adiponectin and leptin levels, which are crucial for energy homeostasis and glucose regulation (8). Genetic factors, particularly polymorphisms in genes related to glucose metabolism (such as GLUT4) and lipid metabolism (including PPAR- $\gamma$  and SREBP-1c), may explain individual variations in response to okra supplementation (48). Furthermore, okra's interaction with other dietary components, such as its potential to enhance the bioavailability of other nutrients and its synergistic effects with other antioxidants, suggests a broader role in metabolic regulation than previously recognized (49).

The substantial heterogeneity observed in several outcomes, particularly in BMI ( $I^2 = 86.5\%$ ), WC ( $I^2 = 82.8\%$ ), weight ( $I^2 = 87.0\%$ ), and FBS ( $I^2 = 91.7\%$ ), warrants careful consideration when interpreting our findings. This heterogeneity likely stems from multiple sources, including variations in participant characteristics across studies (such as baseline BMI, age, and metabolic status), differences in okra preparation methods (ranging from whole fruit consumption to concentrated extracts), and diverse measurement techniques employed across research centers (50). Cultural and dietary differences between countries may have influenced baseline nutritional status and dietary patterns, potentially affecting the response to okra supplementation. For instance, studies conducted in Iran showed different patterns of response compared to those in other countries, possibly due to variations in traditional dietary habits and lifestyle factors. Additionally, the lack of standardization in okra processing methods, including drying techniques, extraction procedures, and storage conditions, may have contributed to the observed heterogeneity by affecting the bioavailability and potency of active compounds (51, 52).

#### 4.4 Clinical Implications of Findings:

The findings of this meta-analysis have significant clinical implications for healthcare practitioners managing patients with metabolic disorders. For patients with prediabetes or diabetes, particularly those over 50 years of age, okra supplementation at doses of 2000 mg or higher shows promising effects on glycemic control, as evidenced by significant reductions in fasting plasma glucose and HbA1c levels. This suggests that okra supplementation could serve as a valuable adjunctive therapy alongside standard diabetes management protocols. Additionally, the observed improvements in lipid profiles, particularly the reduction in LDL-C and total cholesterol with doses below 2000 mg daily over 8-week periods, indicate that okra supplementation might be particularly beneficial for diabetic patients with concurrent dyslipidemia, potentially reducing their cardiovascular risk burden.

For clinicians treating overweight or obese patients under 50 years of age without diabetes, okra supplementation presents a different therapeutic opportunity. The significant reductions in BMI, waist circumference, and body weight observed in this population, particularly with daily doses below 2000 mg over extended periods (>8 weeks), suggest that okra could be a useful addition to weight management programs. The absence of significant effects on blood pressure and the favorable liver function profile, as indicated by improved AST levels, suggests that okra supplementation is generally safe and well-tolerated across different patient populations. However, clinicians should note the varying responses between different demographic groups and consider tailoring dosage and duration of okra supplementation based on individual patient characteristics and therapeutic goals.

#### 4.5 Strengths and Limitations:

One of the key strengths of our meta-analysis is the rigorous methodology employed throughout the study. By adhering to the PRISMA guidelines and registering the study with PROSPERO, we ensured transparency and reproducibility, setting a high standard for systematic reviews. The comprehensive search strategy, which included multiple electronic databases and manual screening of reference lists, allowed us to capture a broad spectrum of relevant studies, thereby minimizing the risk of missing critical data. Our use of the GRADE framework to assess the strength of evidence further enhances the reliability of our findings, offering a nuanced evaluation of the quality and consistency of the included studies. Additionally, the application of

advanced statistical techniques, including meta-regression and dose-response analyses, enabled us to explore the effects of okra supplementation across diverse populations and health outcomes, providing valuable insights into optimal dosing and treatment durations. This robust approach not only reinforces the validity of our conclusions but also contributes significantly to the scientific understanding of okra's potential therapeutic benefits.

Our study presents several key advantages over the last meta-analysis, which focuses exclusively on the effects of okra on dyslipidemia (53). While the earlier study provides valuable insights into the lipid-modulating properties of okra, our meta-analysis extends the scope by evaluating the effects of okra across a broad spectrum of CVD risk factors, including anthropometric measures, blood pressure, glycemic control, lipid profile, and liver function tests. This comprehensive approach offers a holistic view of okra's potential therapeutic benefits, which were not explored in the earlier study. Additionally, our meta-analysis includes a substantially larger dataset of 770 participants across 12 trials, compared to just 8 trials in the earlier work. This increased sample size enhances the robustness and generalizability of our findings. Furthermore, while the earlier study focused on lipid markers alone, we employed advanced statistical methods, including dose-response analysis and meta-regression, providing a nuanced understanding of the optimal dosage and treatment duration for different populations. Our inclusion of diverse health outcomes and sophisticated methodology strengthens the evidence base for okra as a multifaceted intervention for CVD risk reduction.

In contrast to the recent meta-analysis, which focuses solely on the effects of okra on glycemic control in pre-diabetic and type 2 diabetic patients (18), our study expands the focus to encompass a wider range of metabolic and cardiovascular health outcomes. While the earlier study provides valuable evidence on glycemic control, it does not investigate other critical factors like lipid profiles, liver function, or anthropometric parameters, which are essential in understanding the full impact of okra on metabolic health. By including a broader set of health outcomes, our study provides a more comprehensive assessment of okra's therapeutic potential across different disease contexts. Moreover, while the earlier study included 331 patients across eight trials, our meta-analysis evaluates data from a larger number of participants (770 across 12 trials), thus improving the reliability and precision of our findings. Another key advantage is our use of the GRADE framework to assess the quality of the evidence, a methodology not employed

in the second study. This approach allows us to provide a more detailed evaluation of the reliability and consistency of the included studies, offering valuable insights for clinical practice. In addition, our study incorporates subgroup analyses that reveal important patterns in treatment response based on factors such as baseline BMI, age, and health status—insights that are critical for personalized treatment strategies but were not explored in the earlier analysis.

Despite the strengths of our meta-analysis, several limitations must be acknowledged. The relatively small number of included studies and the variation in their design and methodology introduce a degree of heterogeneity that could influence the overall results. Even with the use of a random-effects model to address this variability, it is not possible to eliminate the likelihood of remaining confounding. Furthermore, the exclusion of non-English articles may have led to language bias, limiting the generalizability of our findings across various cultural and geographical settings. Despite our efforts to reduce this through comprehensive search strategies and statistical tests, another limitation is the reliance on reported data from published studies, which may be subject to publication bias.

Furthermore, another limitation of our study was the use of the SMD instead of the Weighted Mean Difference (WMD), which was necessitated by the heterogeneous units reported for key outcomes, such as insulin levels, inflammatory markers, and liver function tests. Although the WMD would have provided more clinically interpretable results, the lack of access to raw data for unit conversion made the use of SMD the most scientifically appropriate choice for ensuring comprehensive inclusion of relevant studies.

Additionally, okra supplementation can involve different parts of the plant, each offering distinct health benefits. The lack of specificity regarding the plant part that was used in the included studies introduce another layer of uncertainty to the outcomes. It is also important to note that some original studies used okra extract, while others used the crude okra plant. This inconsistency in the form of okra supplementation poses a potential limitation, as the doses of okra extract and crude plant are not directly comparable. Therefore, the interpretation and generalization of the dose-response results should be approached with caution. Future studies should provide more detailed information on the okra intervention methods, and future meta-analyses focusing on these aspects are needed to validate and refine our dose-response findings. Lastly, while our analysis included a range of health outcomes, the specific effects of okra on

certain parameters, such as long-term cardiovascular risk, remain underexplored due to the short duration of most included trials. These limitations highlight the need for further large-scale, highquality RCTs to confirm and extend our findings.

#### 4.6 Future Research Directions:

Future research should prioritize investigating the optimal dosing strategies for different patient populations, particularly considering the varying effects observed between diabetic and nondiabetic individuals, age groups, and BMI categories. Long-term randomized controlled trials spanning beyond 8 weeks are needed to establish the sustained efficacy and safety of okra supplementation, especially in patients with multiple comorbidities. Additionally, studies should explore potential drug interactions between okra supplements and commonly prescribed medications for diabetes, hypertension, and dyslipidemia, as these could significantly impact clinical recommendations.

The mechanisms underlying okra's differential effects on various metabolic parameters warrant further investigation through well-designed molecular studies. Future research should focus on identifying specific bioactive compounds responsible for okra's beneficial effects and their molecular targets, particularly in relation to glucose metabolism and lipid homeostasis. Studies examining the role of gut microbiota modulation in okra's therapeutic effects, could provide valuable insights into its mechanism of action and potentially lead to more targeted therapeutic approaches.

Investigation into the potential synergistic effects of okra with other natural compounds or conventional medications could open new therapeutic possibilities. Given the observed variations in efficacy between different populations, studies examining genetic polymorphisms and their influence on individual responses to okra supplementation could help develop personalized treatment approaches. Additionally, research comparing different forms of okra supplementation (whole fruit, extract, or specific compounds) could help optimize delivery methods and enhance therapeutic outcomes.

There is also a critical need for research examining okra's effects on specific subpopulations not well-represented in current studies, such as individuals with metabolic syndrome, gestational diabetes, or concurrent autoimmune conditions. While most research has focused on okra's impact on glycemic control, other potential benefits, such as its anti-cancer, liver-protective, and kidney-protective effects, have not been adequately investigated. Future clinical trials should explore the effects of specific plant parts on various health outcomes. Moreover, expanding research to include molecular mechanisms by which okra exerts its beneficial effects is essential. Advanced molecular biology and omics approaches could help identify the specific bioactive compounds in okra and their interactions with other dietary components. Exploring the potential incorporation of okra into functional foods or nutraceuticals through multidisciplinary studies could inform the formulation of practical dietary guidelines and interventions. Furthermore, cost-effectiveness analyses comparing okra supplementation with conventional treatments would provide valuable information for healthcare policy makers and insurance providers considering coverage for complementary therapies.

### **5 CONCLUSIONS**

In conclusion, this meta-analysis provides strong evidence that okra supplementation offers significant health benefits for cardiometabolic health. These benefits include improved glycemic control, lipid profile, and liver function. Employing statistical methods to combine data from various RCTs, our results indicated that okra has therapeutic potential and can serve as a dietary intervention for preventing and managing conditions such as diabetes, obesity, and dyslipidemia. The dose-response insights gained are crucial for formulating effective supplementation guidelines. Incorporating okra into dietary approaches has significant implications for public health and clinical practice. This comprehensive evaluation enhances the reliability of our findings, making them highly valuable for future research and practical applications in treating metabolic and cardiovascular disorders.

## **Declaration of Competing Interest**

The authors whose names are listed in this article certify that they have NO affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

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### Availability of data and materials

All relevant data are provided within the manuscript and supplementary file. Additionally, data analyzed for this study are available upon request from the corresponding author.

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## **Table 1.** Characteristic of included studies in the meta-analysis

Studies. Year (Ref.)	Country	Study Design	Health Condition	Sample Size (Sex)	Sample Size (INT/CO N)	Trial Duratio n (Week)	Means Age & BMI (INT/CON)	Dose of Supplemen t ( <i>mg/d</i> )	Type of Supplement (INT/CON)	Outcomes
Uebelhack, 2019 (15)	Germany	R, PC, DB	Overweight and moderately obese	51 (B)	35 / 16	12	Age: 46.8±12.3 / 46.7±12.2 BMI: 29.15±2.17 / 30.25±2.59	330	Capsule: Low dose IQ-AE-103 (dehydrated powder of okra (Abelmoschu s esculentus (L.) Moench) pods (165 mg) and inulin (42.5 mg)) / Placebo (standard excipients)	weight, BMI, WC, HC, body fat mass, free fat mass, ALT, AST, ALP, GGT, creatinine, FBS, TC, HDL, LDL-C, TG & Ub A lo
				51 (B)	35 / 16		Age: 48.7±12.8 / 46.7±12.2	660	Capsule: High dose IQ-AE-103	HbA1c

							BMI: 29.44±2.31 / 30.25±2.59		(dehydrated powder of okra (Abelmoschu s esculentus (L.) Moench) pods (330 mg) and inulin (85 mg)) / Placebo (standard excipients)	
Khodija, 2020 (27)	Indonesi a	R, clinical trial	T2DM with hypercholeste rolemia	40 (B)	24 / 16	2	Age: 45-65 / 45-65 BMI: 24.09 & 25.91 / 25.79	40000	Steamed or boiled okra (Abelmoschu s esculentus) / usual care	FBS
Moradi, 2020 (28)	Iran	R, PC, DB	T2DM	60 (B)	30 / 30	8	Age: 54.26±7.62 / 53.33±7.35 BMI:	10000	Powder: Okra (Abelmoschu s esculentus) + yogurt (150	weight, BMI, TG, TC, LDL- C, HDL- C, SBP, DBP,

							24.90±3.94 / 25.65±3.46		g) / Placebo (consumable color) + yogurt (150 g)	FBS, HbA1c, fasting insulin, HOMA-IR & QUICKI
Nikpayam, 2022 (29)	Iran	R, PC, TB	Diabetic nephropathy	55 (B)	30 / 25	10	Age: 62±7 / 61.6±8.5 BMI: 30.35±5.05 / 28.64±3.17	125	Capsule: Dried okra (Abelmoschu s esculentus L.) powder extract (80 mg dried okra powder, 4% avicel & 1% magnesium stearate) / Placebo (carboxymeth ylcellulose)	weight, BMI, WC, HC, WHR, body fat, fat mass, visceral fat, FFM & muscle mass
Peng, 2022 (30)	Germany	R, PC, DB	Overweight and moderately obese	55 (B)	30 / 25	12	Age: 50±11.9 / 47±11.4 BMI: 29.42±2.27 /	1980	Capsule: IQP-AE-103 (containing 330 mg of dehydrated okra powder & 85 mg of	weight, BMI, WC, HC, body fat mass & FFM

							30.05±2.71		inulin) / Placebo (standard excipients)	
Saatchi, 2022 (14)	Iran	R, PC, DB	T2DM	99 (B)	50 / 49	8	Age: 57.7±9.7 / 58.3±9.2 BMI: 30.2±4.3 / 31.1±4.1	4000	Okra (Abelmoschu s culentesus) + Oral hypoglycemi c agents / Placebo (microcrystal line cellulose & magnesium stearate) + Oral hypoglycemi c agents	SBP, DBP, WC, FBS, HbA1c, LDL-C, HDL-C, TG, TC, ALT, AST & BMI
Chen, 2023 (10)	China	R, controll ed trial	IGT	60 (B)	30 / 30	8	Age: 41.4±5.940 / 40.77±5.42 5 BMI:	20000	Tea: Hibiscus esulentus dried fruit tea/ lifestyle control	FBS, 2h PBG, HbA1c, fasting insulin, 2h postprandi

							NR			al insulin HOMA- IR, TC, TG, LDL- C, HDL- C, ALT & creatinine
Salarfard, 2023 (31)	Iran	R, controll ed trial, non- blinded	GDM	60 (F)	30 / 30	4	Age: 29±3.9 / 28.0±4.6 BMI: 27.3±4.2 / 26.2±4.0	6000	Powder: Okra / usual care	FBS & 2h PBG
Tavakolizade h, 2023 (12)	Iran	R, PC, DB	T2DM	94 (B)	48 / 46	12	Age: 53.8±3.7 / 52.8±4.6 BMI: 28.6±2.05 / 29.5±3.36	3000	Capsule: Abelmoschus esculentus (okra) + Antihypergly cemic medicines (metformin or sulfonylureas or sitagliptin or SGLT2 inhibitors) / Placebo (microcrystal	FBS, HbA1c, LDL-C, HDL-C, TG, TC, fasting insulin, HOMA- IR, hs- CRP, ALT, AST, ALP, SBP, DBP, BMI,

									line cellulose) + Antihypergly cemic medicines (metformin or sulfonylureas or sitagliptin or SGLT2 inhibitors)	BUN & creatinine
Afsharmanes h, 2024 (13)	Iran	R, PC, DB	Prediabetic	70 (B)	35 / 35	8	Age: 45.81±6.59 / 45.81±6.59 BMI: NR	3000	Capsule: Okra powder (blended with magnesium stearate powder) / Placebo (carboxymeth yl cellulose, magnesium stearate)	TG, TC, LDL-C, HDL-C, AST, ALT, ALP, creatinine & BUN
Bahreini, 2024 (11)	Iran	R, PC, TB	Diabetic nephropathy	55 (B)	30 / 25	10	Age: 62±7 / 64.6±8.5 BMI: 30.3 / 29.3	125	Capsule: Dried okra (Abelmoschu s esculentus L) extract (80 mg dried okra extract, 4% avicel &	weight, TG, TC, LDL-C, HDL-C, SBP, DBP & IL-1β

									1% magnesium stearate) / Placebo (carboxymeth ylcellulose)	
Damayanthi, 2024 (32)	Indonesi a	Control led clinical trial	Healthy	20 (B)	10 / 10	4	Age: 21.7±1.90 / 23.70±2.83 BMI: 21.80±2.00 / 22.0±1.87	3800	Capsule: purple okra / No treatment	SOD & FBS

Footprint 2h PPG, 2-hour Postprandial Blood Glucose; ALP, Alkaline Phosphatase; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; B, Both Sex; BMI, Body Mass Index; BUN, Blood Urea Nitrogen; CON, Control Group; DB, Double-Blinded; DBP, Diastolic Blood Pressure; F, Female; FBS, Fasting Blood Sugar; FFM, Fat Free Mass; GDM, Gestational Diabetes Mellitus; GGT, Gamma-Glutamyl Transferase; HbA1c, Hemoglobin A1C; HC, Hip Circumference; HDL-C, High-Density Lipoprotein Cholesterol; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance; hs-CRP, High-Sensitivity C-Reactive Protein; IL-1β, Interleukin-1 Beta; INT, Intervention Group; LDL-C, Low-Density Lipoprotein Cholesterol; QUICKI, Quantitative Insulin Sensitivity Check Index; RCT, Randomized Controlled Trial; SBP, Systolic Blood Pressure; SOD, Superoxide dismutase activity; T2DM, Type 2 Diabetes Mellitus; TB, Triple-Blinded; TC, Total Cholesterol; TG, Triglycerides; WC, Waist Circumference; WHR, Waist-to-Hip Ra

**Table 2**. Quality of included studies in the meta-analysis

Study, Year (Ref.)	Random sequence generation	Allocation concealment	Blinding of participants & personnel	Blinding of outcome assessment	Incomplete outcome data	Selective outcome reporting	Other sources of bias	Overall quality
Uebelhack, 2019 (15)	L	Н	L	Н	Н	L	L	Poor
Khodija, 2020 (27)	L	Н	Н	Н	Н	L	L	Poor
Moradi, 2020 (28)	L	Н	L	Н	Н	L	L	Poor
Nikpayam, 2022 (29)	L	Н	L	Н	Н	L	L	Poor
Saatchi, 2022 (14)	L	Н	L	Н	Н	L	L	Poor
Peng, 2022 (30)	U	Н	L	Н	Н	L	L	Poor
Chen, 2023 (10)	L	Н	Н	Н	L	L	L	Poor
Salarfard, 2023 (31)	L	Н	Н	Н	L	L	L	Poor
Tavakolizadeh , 2023 (12)	L	Н	L	Н	L	L	L	Poor
Afsharmanesh , 2024 (13)	L	Н	L	Н	L	L	L	Poor
Bahreini, 2024 (11)	L	Н	L	L	Н	L	L	Poor
Damayanthi, 2024 (32)	Н	Н	Н	Н	L	L	L	Poor

Footprint: H, high risk of bias; L, low risk of bias; U, unclear risk of bias.

Table 3. Description of	the analysis and	subgroup results of	Okra supplementation of	on cardiovascular disease risk factors.
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	Studies	Doutisinout			Het	erogeneity					
	N	Participant N	SMD (95%CI)	<i>P</i> -value	P heterogeneity	I <sup>2</sup> (95%CI)	tau <sup>2</sup>	P between sub- groups			
Analysis and subgroup results of Okra supplementation on BMI											
Overall effect	7	465	-0.70 (- 1.23, - 0.16)	0.011	<0.001	86.5% (26.5%, 94.6%)	0.444				
Country				1							
Iran	4	308	-0.13 ( - 0.36, 0.09)	0.249	0.814	0.0% (NA)	NA				
Other Countries	3	157	-1.53 ( - 2.09, - 0.97)	<0.001	0.107	55.3% (NA)	NA	_ <0.001			
Health Status					1		1				
Prediabetic or Diabetic	4	308	-0.13 ( - 0.36, 0.09)	0.249	0.814	0.0% (NA)	NA	<0.001			
Without Diabetes	3	157	-1.53 (- 2.09, - 0.97)	<0.001	0.107	55.3% (NA)	NA				

Age (year)								
50 >	2	102	-1.35 (- 2.08, - 0.62)	<0.001	0.115	59.8% (NA)	NA	0.055
50 ≤	5	363	-0.45 (- 1.01, 0.11)	0.115	<0.001	85.1% (NA)	NA	
Baseline BMI								
Healthy weight (≤25 kg/m2)	1	60	-0.19 (- 0.70, 0.32)	0.463	-	-	NA	
Overweight or Obese (>25 kg/m2)	6	405	-0.79 ( - 1.42, - 0.17)	0.013	<0.001	88.3% (NA)	NA	0.144
Not given	-	-	-	-	-	-	NA	
Duration (weeks)								
8≥	2	159	-0.08 (- 0.39, 0.23)	0.601	0.602	0.0% (NA)	NA	0.029
8 <	5	306	-0.96 (- 1.68, - 0.24)	0.009	<0.001	87.9% (NA)	NA	0.027
Dose (mg/d)								

			1 15 (					
2000 ≥	4	212	-1.15 ( - 2.03, - 0.27)	0.011	<0.001	87.8% (NA)	NA	0.033
2000 <	3	253	-0.15 (- 0.40, 0.09)	0.226	0.673	0.0% (NA)	NA	
Sample Size	I			1				1
60 >	4	212	-1.15 (- 2.03, - 0.27)	0.011	<0.001	87.8% (NA)	NA	0.033
60 ≤	3	253	-0.15 (- 0.40, 0.09)	0.226	0.673	0.0% (NA)	NA	
	Analy	sis and subgrou	ip results of	Okra sup	plementation on <b>F</b>	FM		
Overall effect	4	212	-0.13 (- 0.41, 0.15)	0.349	0.679	0.0% (0.0%, 36.7%)	0.000	
	Analy	ysis and subgro	up results o	f Okra suj	pplementation on	FM		Ż
Overall effect	4	212	-0.74 (- 1.13, - 0.36)	<0.001	0.149	43.8% (0.0%, 82.0%)	0.069	
	Anal	ysis and subgro	up results o	f Okra suj	pplementation on	HC		
Overall effect	4	212	-0.85 (- 1.41, -	0.003	0.012	72.4% (0.0%,	0.238	

		0.28)			91.2%)		
Ana	alysis and sub	ogroup results o	of Okra suj	pplementation	on WC		
5	311	-0.57 (- 1.15, 0.01)	0.054	<0.001	82.8% (0.0%, 94.0%)	0.359	
2	154	0.08 (- 0.24, 0.39)	0.644	0.953	0.0% (NA)	NA	<0.001
3	157	-1.04 (- 1.39, - 0.69)	<0.001	0.437	0.0% (NA)	NA	
	<u> </u>	I				1	
2	154	0.08 (- 0.24, 0.39)	0.644	0.953	0.0% (NA)	NA	<0.001
3	157	-1.04 (- 1.39, - 0.69)	<0.001	0.437	0.0% (NA)	NA	
	I	1			1		I
2	102	-0.91 (- 1.35, - 0.48)	<0.001	0.400	0.0% (NA)	NA	0.239
	5         2         3         2         3	5       311         2       154         3       157         2       154         3       157	Analysis and subgroup results of 1.15, 0.57 (- 1.15, 0.01)         5       311       -0.57 (- 1.15, 0.01)         2       154       0.08 (- 0.24, 0.39)         3       157       -1.04 (- 1.39, - 0.69)         2       154       0.08 (- 0.24, 0.39)         3       157       -1.04 (- 1.39, - 0.69)         2       154       0.08 (- 0.24, 0.39)         2       154       0.08 (- 0.24, 0.39)         2       154       0.69)         2       154       0.09 (- 0.91 (- 1.39, - 0.69))	Analysis and subgroup results of Okra sup5 $311$ $\begin{array}{c} -0.57 (-\\ 1.15, \\ 0.01 \end{array} \right)$ $0.054$ 2 $154$ $\begin{array}{c} 0.08 (-\\ 0.24, \\ 0.39 \end{array} \right)$ $0.644$ 3 $157$ $\begin{array}{c} -1.04 (-\\ 1.39, -\\ 0.69 \end{array} \right)$ $< 0.001$ 2 $154$ $\begin{array}{c} 0.08 (-\\ 0.24, \\ 0.39 \end{array} \right)$ $< 0.001$ 3 $157$ $\begin{array}{c} -1.04 (-\\ 1.39, -\\ 0.69 \end{array} \right)$ $< 0.001$ 2 $154$ $\begin{array}{c} 0.08 (-\\ 0.24, \\ 0.39 \end{array} \right)$ $< 0.001$ 2 $154$ $\begin{array}{c} 0.08 (-\\ 0.24, \\ 0.39 \end{array} \right)$ $< 0.001$ 2 $154$ $\begin{array}{c} 0.09 (-\\ 1.39, -\\ 0.69 \end{array} \right)$ $< 0.001$	Analysis and subgroup results of Okra supplementation         5 $311$ $-0.57 (-1.15, 0.054)$ $<0.001$ 2 $154$ $0.08 (-0.24, 0.39)$ $0.644$ $0.953$ 3 $157$ $-1.04 (-1.39, -0.69)$ $<0.001$ $0.437$ 2 $154$ $0.08 (-0.24, 0.69)$ $<0.001$ $0.437$ 3 $157$ $-1.04 (-1.39, -0.69)$ $<0.001$ $0.437$ 2 $154$ $0.08 (-0.24, 0.644)$ $0.953$ $0.69$ 3 $157$ $-1.04 (-1.39, -0.69)$ $<0.001$ $0.437$ 3 $157$ $-1.04 (-1.39, -0.69)$ $<0.001$ $0.437$ 2 $154$ $0.24, 0.39$ $<0.001$ $0.437$ 3 $157$ $-1.04 (-1.39, -0.69)$ $<0.001$ $0.437$ 2 $102$ $-0.91 (-1.35, -0.001$ $0.400$	Analysis and subgroup results of Okra supplementation on WC         5 $311$ $\stackrel{-0.57}{.}$ (- 1.15, 0.01 $0.054$ $<0.001$ $82.8%(0.0%,94.0%)$ 2 $154$ $0.08$ (- 0.24, 0.39) $0.644$ $0.953$ $0.0%(NA)         3       157 \stackrel{-1.04}{.}0.69$ $<0.001$ $0.437$ $0.0%(NA)$ 2 $154$ $0.08$ (- 0.24, 0.69) $<0.001$ $0.437$ $0.0%(NA)         3       157 \stackrel{-1.04}{.}0.39$ ) $<0.001$ $0.437$ $0.0%(NA)         2       154 \stackrel{-0.91}{.}0.69$ $<0.001$ $0.437$ $0.0%(NA)$	Analysis and subgroup results of Okra supplementation on WC           5         311         -0.57 (- 1.15, 0.01)         0.054         <0.001         82.8% (0.0%, 94.0%)         0.359           2         154         0.08 (- 0.24, 0.39)         0.644         0.953         0.0% (NA)         NA           3         157         -1.04 (- 1.39, - 0.69)         <0.001         0.437         0.0% (NA)         NA           2         154         0.08 (- 0.24, 0.39)         <0.001         0.437         0.0% (NA)         NA           3         157         -1.04 (- 1.39, - 0.69)         <0.001         0.437         0.0% (NA)         NA           2         154         0.08 (- 0.24, 0.39)         0.644         0.953         0.0% (NA)         NA           2         154         0.08 (- 0.24, 0.39)         0.644         0.953         0.0% (NA)         NA           3         157         -1.04 (- 1.39, - 0.69)         <0.001         0.437         0.0% (NA)         NA           2         102         -0.91 (- 1.35, -         <0.001         0.400         0.0% (NA)         NA

50 ≤	3	209	-0.36 (- 1.17, 0.45)	0.386	<0.001	87.4% (NA)	NA	
Baseline BMI						I		
Healthy weight (≤25 kg/m2)	-	-	-	-	-	-	NA	
Overweight or Obese			-0.57 (-			82.8%		
(>25 kg/m2)	5	311	1.15, 0.01)	0.054	<0.001	82.8% (NA)	NA	
Not given	-	-	-	-	-	-	NA	
Duration (weeks)		1	I			I		I
8≥	1	99	0.08 (- 0.31, 0.48)	0.685	-	-	NA	0.026
8 <	4	212	-0.75 (- 1.37, - 0.14)	0.017	<0.001	77.4% (NA)	NA	0.020
Dose (mg/d)								
2000 ≥	4	212	-0.75 (- 1.37, - 0.14)	0.017	0.004	77.4% (NA)	NA	0.026
2000 <	1	99	0.08 (- 0.31, 0.48)	0.685	-	-	NA	0.020

Sample Size								
60 >	4	212	-0.75 (- 1.37, - 0.14)	0.017	0.004	77.4% (NA)	NA	0.026
60 ≤	1	99	0.082 (- 0.31, 0.48)	0.685	-	-	NA	0.020
	Anal	ysis and subg	roup results of	Okra supp	plementation o	on Weight		I
Overall effect	6	327	-0.77 (- 1.42, - 0.11)	0.022	<0.001	87.0% (19.0%, 95.0%)	0.581	
Country								
Iran	3	170	-0.08 (- 0.38, 0.23)	0.622	0.660	0.0% (NA)	NA	<0.001
Other Countries	3	157	-1.49 (- 2.06, - 0.93)	<0.001	0.099	56.7% (NA)	NA	<0.001
Health Status		I				I	<u> </u>	I
Prediabetic or Diabetic	3	170	-0.08 (- 0.38, 0.23)	0.622	0.660	0.0% (NA)	NA	-0.001
Without Diabetes	3	157	-1.49 (- 2.06, - 0.93)	<0.001	0.099	56.7% (NA)	NA	

Age (year)		<u> </u>						
50 >	2	102	-1.31 (- 2.06, - 0.57)	0.001	0.105	62.0% (NA)	NA	0.145
50 ≤	4	225	-0.50 (- 1.30, 0.30)	0.219	<0.001	88.1% (NA)	NA	0.145
Baseline BMI		I						
Healthy weight (≤25 kg/m2)	1	60	-0.27 (- 0.78, 0.24)	0.305	-	-	NA	
Overweight or Obese (>25 kg/m2)	5	267	-0.87 (- 1.67, - 0.08)	0.032	<0.001	88.9% (NA)	NA	0.209
Not given	-	-	-	-	-	-	NA	
Duration (weeks)		I		1				I
8≥	1	60	-0.27 (- 0.78, 0.24)	0.305	-	-	NA	0.209
8 <	5	267	-0.87 (- 1.67, - 0.08)	0.032	<0.001	88.9% (NA)	NA	0.209
Dose (mg/d)						I		
2000 ≥	5	267	-0.87 (-	0.032	<0.001	88.9%		0.209

			1.67, -			(NA)	NA	
			0.08)					
2000 <			-0.27 (-					
2000 <	1	60	0.78,	0.305	-	-	NA	
			0.24)					
Sample Size		<u>.</u>	1			I	-	1
60 >			-0.87 (-			88.9%		
00 -	5	267	1.67, -	0.032	< 0.001	(NA)	NA	
			0.08)					0.209
			-0.27 (-					0.207
<b>60</b> ≤	1	60	0.78,	0.305	-	-	NA	
			0.24)					
	Analy	sis and subgrou	p results of	f Okra sup	pplementation of	on DBP		
			-0.15 (-			0.0%		
Overall effect	4	308	0.37,	0.195	0.966	(0.0%,	0.000	
			0.08)			0.0%)		
	Analy	sis and subgrou	ip results of	f Okra suj	pplementation	on SBP		
			-0.13 (-			71.9%		
Overall effect	4	308	0.56,	0.551	0.014	(0.0%,	0.139	
			0.30)			91.1%)		
	Analysis an	d subgroup res	ults of Okr	a supplem	entation on Fa	sting Insulin	-	
Overall effect	3	214	-0.35 (-	0.013	0.351	4.6%		
			0.63, -			(0.0%,	0.003	

			0.07)			74.1%)		
	Ana	alysis and sub	ogroup results of	f Okra sup	oplementation	on FBS	<u> </u>	
Overall effect	10	535	-1.07 (- 1.75, - 0.38)	0.002	< 0.001	91.7% (66.2%, 96.3%)	1.088	
Country			I	1			1	
Iran	4	313	-1.84 ( - 2.95, - 0.73)	0.001	< 0.001	94.1% (NA)	NA	0.025
Other countries	6	222	-0.47 (- 1.10, 0.16)	0.146	< 0.001	77.8% (NA)	NA	— 0.035
Health Status			I	1			1	
Prediabetic or Diabetic	7	413	-1.61 ( - 2.38, - 0.84)	< 0.001	< 0.001	90.8% (NA)	NA	.0.001
Without Diabetes	3	122	0.207 ( - 0.17, 0.59)	0.284	0.971	0.0% (NA)	NA	< 0.001
Age (year)						·	1	1
50 >	7	282	-1.05 ( - 2.11, 0.02)	0.053	< 0.001	93.1% (NA)	NA	0.966
<b>50</b> ≤	3	253	-1.16 (- 1.84, - 0.47)	0.001	0.002	84.2% (NA)	NA	— 0.866
Baseline BMI		1	· · ·	1	1	I	1	I

Healthy weight (≤25 kg/m2)	4	120	-0.95 (- 1.81, - 0.09)	0.030	0.007	75.2% (NA)	NA	
Overweight or Obese (>25 kg/m2)	5	355	-1.29 (- 2.48, - 0.09)	0.035	< 0.001	95.6% (NA)	NA	0.272
Not given	1	60	-0.38 (- 0.89, 0.128)	0.142	-	-	NA	
Duration (weeks)	I		I			I		I
8≥	7	339	-1.34 (- 2.19, - 0.50)	0.002	< 0.001	90.9% (NA)	NA	0.206
8 <	3	196	-0.46 (- 1.88, 0.96)	0.522	< 0.001	94.9% (NA)	NA	0.296
Dose (mg/d)		1	I		1			I
2000 ≥	2	102	0.23 ( - 0.19, 0.65)	0.288	0.922	0.0% (NA)	NA	.0.001
2000 <	8	433	-1.40 (- 2.14, - 0.67)	< 0.001	< 0.001	90.6% (NA)	NA	< 0.001
Sample Size						·		
60 >	5	162	-0.53 (- 1.37, 0.312)	0.219	< 0.001	81.9% (NA)	NA	0.116
<b>60</b> ≤	5	373	-1.54 (- 2.48, -	0.001	< 0.001	93.7% (NA)	NA	

			0.60)					
	Ana	lysis and subg	roup results of	Okra supp	lementation	on HbA1c		
Overall effect	6	415	-0.38 ( - 0.71, - 0.05)	0.023	0.023	61.7% (0.0%, 85.2%)	0.102	
Country								
Iran	3	253	-0.67 (- 0.99, - 0.34)	< 0.001	0.199	38.0% (NA)	NA	
Other countries	3	162	-0.05 (- 0.37, 0.28)	0.771	0.680	0.0% (NA)	NA	0.008
Health Status			1			l		I
Prediabetic or Diabetic	4	313	-0.50 ( - 0.89, - 0.11)	0.013	0.032	65.9% (NA)	NA	0.155
Without Diabetes	2	102	-0.08 ( - 0.50, 0.34)	0.698	0.401	0.0% (NA)	NA	- 0.155
Age (year)						I		I
50 >	3	162	-0.05 ( - 0.37, 0.28)	0.771	0.680	0.0% (NA)	NA	0.000
50 ≤	3	253	-0.67 (- 0.99, - 0.34)	< 0.001	0.199	38.0% (NA)	NA	0.008
Baseline BMI		I						
Healthy weight (≤25	1	60	-0.31 (-	0.230	-	-		0.348

Overweight or Obese (>25 kg/m2)       4       295       -0.49 (- 0.91, - 0.025) 0.031       66.1% (NA)       NA         Not given       1       60       0.00 (- 0.50, 0.51) 0.991       -       -       NA         Duration (weeks)       3       219 $^{-0.49 (- 0.06) 0.50, 0.991$ 0.133 0.017       75.4% (NA)       NA         8 ≥       3       219 $^{-0.42 (- 0.98, 0.133) 0.133} 0.133$ 0.017       0.093       57.9% (NA)       NA         0.000 ≥       3       196 $^{-0.32 (- 0.08, 0.133) 0.017$ 0.093       57.9% (NA)       NA         0.000 ≥       2       102 $^{-0.08 (- 0.50, 0.133) 0.017$ 0.093       57.9% (NA)       NA         0.000 ≥       2       102 $^{-0.08 (- 0.50, 0.133 0.017$ 0.093       57.9% (NA)       NA         2000 ≥       2       102 $^{-0.08 (- 0.50, 0.133 0.013 0.032$ 0.698 0.401       0.09% (NA)       NA         5ample Size       3       102 $^{-0.08 (- 0.50, 0.133 0.013 0.032$ 0.698 0.401       0.09% (NA)       NA       0.155         60 ≥       2       102 $^{-0.08 (- 0.50, 0.233 0.033 0.032 0.033 0.032 0.033 0.033 0.033 0.032 0.033 0$	lra/m2)			0.82,				NA	
Overweight or Obese (>25 kg/m2)         4         295         -0.49 (- 0.91, - 0.06)         0.025         0.031 $66.1\%$ (NA)         NA           Not given         1         60 $0.00 (-0.51)$ 0.991         -         -         NA           Duration (weeks) $-$ NA $-$ NA           8 ≥         3         219 $-0.42 (-0.98, 0.13)$ $0.133$ $0.017$ $75.4\%$ (NA)         NA           8          3         196 $-0.32 (-0.80, 0.15)$ $0.193$ $0.093$ $57.9\%$ (NA)         NA           Dose (mg/d) $      -$ 2000 ≥         2 $102$ $-0.50 (-0.50, 0.34)$ $0.698$ $0.401$ $0.0\%$ (NA)         NA           2000 <         4 $313$ $0.50, -0.89 - 0.013$ $0.032$ $65.9\%$ (NA)         NA           5 $0.034$ $0.401$ $0.0\%$ (NA)         NA $0.155$ 60 >         2 $102$ $0.50, 0.$	kg/m2)			,					
(>25 kg/m2)42950.91, - 0.06)0.0250.031 $\stackrel{00.1\%}{(NA)}$ NANot given160 $\stackrel{0.00}{,} \stackrel{(,00)}{,} \stackrel{(,00)}{,} \stackrel{(,09)}{,} \stackrel{(,09)}{,} \stackrel{(,09)}{,} \stackrel{(,09)}{,} \stackrel{(,09)}{,} \stackrel{(,01)}{,} \stackrel$	Overweight or Obese			-0.49 (-					
Not given       1       60       0.06 (- 0.50, 0.50, 0.991) (-       -       NA         Duration (weeks)       3       219       -0.42 (- 0.98, 0.13) (-       0.133 (-       0.017 (-       75.4% (NA) (NA) (-       NA         8 ≥       3       219       -0.42 (- 0.98, 0.13) (-       0.133 (-       0.017 (-       75.4% (NA) (NA) (-       NA         8 <       3       196       -0.32 (- 0.15) (-       0.178 (-       0.093 (-       57.9% (NA) (-       NA         Dose (mg/d)       -       -       NA       -       -       -       NA         2000 ≥       2       102       -0.08 (- 0.50, 0.34) (-       0.093 (-       57.9% (NA) (NA) (-       NA         2000 <       4       313       -0.50 (- 0.89, -       0.013 (-       0.093 (-       65.9% (NA) (NA) (-       NA         Sample Size       -       -       -       -       -       -       -         60 ≤       4       313       0.89 (- 0.50, 0.34) (-       0.698 (- 0.698 (-       0.401 (-       0.0% (-       NA       -         60 ≤       4       313       0.89 (- 0.50, 0.34) (-       0.932 (-       65.9% (NA)       NA       -	_	4	295	,	0.025	0.031		NA	
Not given         1         60         0.50, 0.51, 0.991         -         -         NA           Duration (weeks) $8 \ge$ 3         219         -0.42 (- 0.98, 0.13)         0.133         0.017         75.4% (NA)         NA $8 \ge$ 3         219         -0.42 (- 0.98, 0.13)         0.133         0.017         75.4% (NA)         NA $8 <$ 3         196         -0.32 (- 0.32)         0.178         0.093         57.9% (NA)         NA           Dose (mg/d)         -         -         NA         -         -         NA           2000 $\ge$ 2         102         -0.08 (- 0.50, 0.43)         0.093         57.9% (NA)         NA           2000 $<$ 2         102         -0.08 (- 0.50, 0.43)         0.093         0.401         0.0% (NA)         NA           2000 $<$ 4         313         -0.50 (- 0.89, - 0.11)         0.013         0.032         65.9% (NA)         NA           55.9%         NA							(NA)		
$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	NT / •			0.00 (-					
Duration (weeks)         8 ≥       3       219 $\stackrel{-0.42}{0.98}$ , $\stackrel{-0.133}{0.13}$ 0.017 $\stackrel{75.4\%}{(NA)}$ NA         8 <       3       196 $\stackrel{-0.32}{0.13}$ 0.178       0.093 $\stackrel{57.9\%}{(NA)}$ NA         0.0se (mg/d)       -0.32 (-       0.178       0.093 $\stackrel{57.9\%}{(NA)}$ NA       0.787         2000 ≥       2       102 $\stackrel{-0.08 (-}{0.50(-)}$ 0.698       0.401 $\stackrel{0.0\%}{(NA)}$ NA       0.155         2000 <       4       313 $\stackrel{-0.50 (-}{0.89, -}$ 0.013       0.032 $\stackrel{65.9\%}{(NA)}$ NA         5mple Size       -       -       0.698       0.401 $\stackrel{0.0\%}{(NA)}$ NA       0.155         60 >       2       102 $\stackrel{-0.08 (-}{0.50(-)}$ 0.698       0.401 $\stackrel{0.0\%}{(NA)}$ NA         60 >       2       102 $\stackrel{-0.08 (-}{0.50(-)}$ 0.698       0.401 $\stackrel{0.0\%}{(NA)}$ NA         60 >       2       102 $\stackrel{-0.08 (-}{0.50(-)}$ 0.698       0.401 $\stackrel{0.0\%}{(NA)}$ NA         610 ≤       4       313 $\stackrel{-0.50 (-}{0.50(-)}$ 0.013       0.032 <t< td=""><th>Not given</th><td>1</td><td>60</td><td>0.50,</td><td>0.991</td><td>-</td><td>-</td><td>NA</td><td></td></t<>	Not given	1	60	0.50,	0.991	-	-	NA	
8≥       3       219 $\stackrel{-0.42}{,0.98}, \\ 0.133$ 0.017 $\stackrel{75.4\%}{,(NA)}$ NA $_{0.787}$ 8 <       3       196 $\stackrel{-0.32}{,0.80}, \\ 0.15$ 0.178       0.093 $\stackrel{57.9\%}{,(NA)}$ NA $_{0.787}$ Dose (mg/d)				0.51)					
8 $\geq$ 3       219       0.98, 0.133       0.017       75.4% (NA)       NA	Duration (weeks)			·		·			·
32190.98, 0.13)0.1330.017(NA)NA8 <3196 $\stackrel{-0.32}{0.20}$ 0.15)0.1780.093 $\stackrel{57.9\%}{(NA)}$ NADose (mg/d)	8>						75 4%		
$8 <$ $3$ $196$ $-0.32 (-)$ $0.80,$ $0.15$ $0.093$ $57.9\%$ $(NA)$ NA $0.787$ Dose (mg/d) $0.178$ $0.093$ $57.9\%$ $(NA)$ NA $0.155$ $0.093$ $57.9\%$ $(NA)$ NA $0.787$ Dose (mg/d) $0.155$ $0.093$ $0.093$ $57.9\%$ $(NA)       NA       0.787 2000 \ge 2 102 -0.08 (-)0.34) 0.698 0.401 0.0\%(NA)       NA       0.155 2000 < 4 313 -0.50 (-)0.11 0.013 0.032 65.9\%(NA)       NA       0.155 500 > 2 102 -0.08 (-)0.34 0.032 65.9\%(NA)       NA       0.155 60 > 2 102 -0.50 (-)0.34 0.032 0.0\%(NA)       NA       0.155 60 \le 4 313 0.89 - 10.34 0.032 65.9\%0.9\%       NA   $	0 2	3	219	0.98,	0.133	0.017		NA	
8 <       3       196 $-0.32 (-) \\ 0.80, \\ 0.15)$ 0.178       0.093 $57.9\% \\ (NA)$ NA         Dose (mg/d) $-0.08 (-) \\ 0.34)$ 0.093 $57.9\% \\ (NA)$ NA         2000 ≥       2       102 $-0.08 (-) \\ 0.34)$ 0.698       0.401 $0.0\% \\ (NA)$ NA         2000 <       4       313 $-0.50 (-) \\ 0.11)$ 0.013       0.032 $65.9\% \\ (NA)$ NA         Sample Size       -       -       - $-0.08 (-) \\ 0.11)$ 0.013       0.401 $0.0\% \\ (NA)$ NA         60 >       2       102 $-0.50 (-) \\ 0.50, \\ 0.34)$ 0.698       0.401 $0.0\% \\ (NA)$ NA         60 >       2       102 $-0.08 (-) \\ 0.50, \\ 0.34)$ 0.698       0.401 $0.0\% \\ (NA)$ NA         60 >       2       102 $-0.08 (-) \\ 0.50, \\ 0.34)$ 0.698       0.401 $0.0\% \\ (NA)$ NA $-0.155$				0.13)					0.787
31960.80, 0.15)0.1780.093(NA)NADose (mg/d) $2000 \ge$ 2102 $\stackrel{-0.08}{_{0.34}}$ 0.6980.401 $\stackrel{0.0\%}{_{(NA)}}$ NA2000 ≤4313 $\stackrel{-0.50}{_{0.11}}$ 0.0130.032 $\stackrel{65.9\%}{_{(NA)}}$ NASample Size	8 -			-0.32 (-			57 0%		0.787
Image: Dose (mg/d)       Image: Dose (mg/d)         2000 ≥       2       102 $\stackrel{-0.08}{_{0.50}}$ 0.698       0.401 $\stackrel{0.0\%}{_{(NA)}}$ NA         2000 <       4       313 $\stackrel{-0.50}{_{0.11}}$ 0.013       0.032 $\stackrel{65.9\%}{_{(NA)}}$ NA         Sample Size       -0.08 (- 0.11)       0.032 $\stackrel{0.0\%}{_{(NA)}}$ NA $\stackrel{-0.55}{_{(NA)}}$ 0.155         60 >       2       102 $\stackrel{-0.08 (-}{_{0.50}}$ 0.698       0.401 $\stackrel{0.0\%}{_{(NA)}}$ NA         60 >       2       102 $\stackrel{-0.08 (-}{_{0.50}}$ 0.698       0.401 $\stackrel{0.0\%}{_{(NA)}}$ NA         60 >       2       102 $\stackrel{-0.08 (-}{_{0.50}}$ 0.698       0.401 $\stackrel{0.0\%}{_{(NA)}}$ NA         60 >       2       102 $\stackrel{-0.08 (-}{_{0.50}}$ 0.698       0.401 $\stackrel{0.0\%}{_{(NA)}}$ NA $\stackrel{-1.55}{_{(NA)}}$ 60 ≤       4       313 $\stackrel{0.89}{_{0.89}}$ 0.013       0.032 $\stackrel{65.9\%}{_{(NA)}}$ NA	0 <	3	196	0.80,	0.178	0.093		NA	
2000 ≥       2       102 $\stackrel{-0.08}{0.50}$ , $\stackrel{-0.698}{0.34}$ 0.401 $\stackrel{0.0\%}{(NA)}$ NA $\stackrel{-0.155}{(NA)}$ 2000 <       4       313 $\stackrel{-0.50}{0.89}$ , $\stackrel{-0.013}{0.11}$ 0.032 $\stackrel{65.9\%}{(NA)}$ NA $\stackrel{-0.155}{(NA)}$ Sample Size       -0.08 (-       0.698       0.401 $\stackrel{0.0\%}{(NA)}$ NA $\stackrel{-0.155}{(NA)}$ $\stackrel{-0.08}{(NA)}$ $\stackrel{-0.008}{(NA)}$ $\stackrel{-0.003}{(NA)}$ $\stackrel{-0.003}{(NA)}$ $\stackrel{-0.003}{(NA)}$				0.15)					
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	Dose (mg/d)								
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	2000 >						0.0%		
2000 <4313 $\stackrel{-0.50}{,}{}_{0.11}$ 0.0130.032 $\stackrel{65.9\%}{,}{}_{NA}$ NA0.155Sample Size60 >2102 $\stackrel{-0.08}{,}{}_{0.34}$ 0.6980.401 $\stackrel{0.0\%}{,}{}_{NA}$ NA $\stackrel{-0.155}{,}{}_{0.155}$ 60 <2102 $\stackrel{-0.08}{,}{}_{0.34}$ 0.6980.401 $\stackrel{0.0\%}{,}{}_{NA}$ NA $\stackrel{-0.155}{,}{}_{0.155}$ 60 <2102 $\stackrel{-0.50}{,}{}_{0.34}$ 0.6980.401 $\stackrel{0.0\%}{,}{}_{NA}$ NA $\stackrel{-0.155}{,}{}_{0.155}$	2000 2	2	102	0.50,	0.698	0.401		NA	
2000 <4313 $\stackrel{-0.50}{0.89}, -\\0.11$ 0.0130.032 $\stackrel{65.9\%}{(NA)}$ NASample Size60 >2102 $\stackrel{-0.08}{0.50}, \\0.34$ 0.6980.401 $\stackrel{0.0\%}{(NA)}$ NA60 ≤43130.89 -0.0130.03265.9%NA				,					0 155
4313 $0.89, -$ $0.11$ $0.013$ $0.032$ $(NA)$ NASample Size60 >2102 $-0.08 (-$ $0.50, 0.34)$ $0.698$ $0.401$ $0.0\%$ $(NA)$ NA60 ≤4313 $0.89,  0.013$ $0.032$ $0.032$ $0.0\%$ $(NA)$ NA	2000 ~			-0.50 (-			65.9%		0.155
Sample Size       -0.08 (-       0.401       0.0%       NA         60 >       2       102 $\stackrel{-0.08 (-)}{0.50,}$ 0.698       0.401 $\stackrel{0.0\%}{(NA)}$ NA         60 >       2       102 $\stackrel{-0.05 (-)}{0.34}$ 0.698       0.401 $\stackrel{0.0\%}{(NA)}$ NA         60 ≤       4       313       0.89 -       0.013       0.032       65.9%       NA	2000 <	4	313	0.89, -	0.013	0.032		NA	
60 >       2       102 $\stackrel{-0.08}{0.50}$ , $\stackrel{-0.698}{0.34}$ 0.401 $\stackrel{0.0\%}{(NA)}$ NA         60 ≤       4       313 $\stackrel{-0.50}{0.89}$ , $\stackrel{-0.50}{0.013}$ 0.032 $\stackrel{65.9\%}{65.9\%}$ NA				0.11)					
$\begin{array}{c c c c c c c c c c c c c c c c c c c $	Sample Size								
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	60 >			-0.08 (-			0.0%		
$60 \le 4 \qquad 313 \qquad 0.89 = 0.013 \qquad 0.032 \qquad 65.9\% \qquad NA \qquad 0.155$	00 -	2	102	0.50,	0.698	0.401		NA	
$60 \le 4 \qquad 313 \qquad 0.89 = 0.013 \qquad 0.032 \qquad 65.9\% \qquad NA$				0.34)			(INA)		0.155
4 313 $0.89 - 0.013$ $0.037$ NA	60 ~			-0.50 (-			65 00/		0.133
	00 >	<b>60</b> ≤ 4	313	0.89, -	0.013	0.032		I NA	
(NA)		4		0.11)			$(\mathbf{INA})$		

	Analy	sis and subgro	oup results of O	kra supple	mentation on	HOMA-IR		
Overall effect	3	214	-0.56 (- 0.84, - 0.29)	< 0.001	0.858	0.0% (0.0%, 0.0%)	0.000	
	Ana	lysis and subg	roup results of	Okra supp	lementation of	on HDL-C		
Overall effect	8	540	0.13 (- 0.15, 0.41)	0.354	0.015	59.7% (0.0%, 82.5%)	0.093	
Country		<u> </u>	I		1	I	1	I
Iran	5	378	0.17 (- 0.26, 0.59)	0.444	0.002	76.2% (NA)	NA	0.722
Other Countries	3	162	0.07 (- 0.25, 0.39)	0.676	0.791	0.0% (NA)	NA	0.723
Health Status								
Prediabetic or Diabetic	6	438	0.14 (- 0.21, 0.50)	0.425	0.005	70.4% (NA)	NA	0.850
Without Diabetes	2	102	0.09 (- 0.33, 0.51)	0.672	0.506	0.0% (NA)	NA	0.830
Age (year)								

50 >	4	232	0.29 ( - 0.15, 0.73)	0.196	0.048	62.1% (NA)	NA	0.273
50 ≤	4	308	-0.01 (- 0.33, 0.31)	0.933	0.116	49.2% (NA)	NA	
Baseline BMI						I		
Healthy weight (≤25 kg/m2)	1	60	0.02 (- 0.49, 0.52)	0.949	-	-	NA	
Overweight or Obese (>25 kg/m2)	5	350	0.01 (- 0.27, 0.29)	0.932	0.161	39.1% (NA)	NA	0.596
Not given	2	130	0.46 (- 0.37, 1.30)	0.275	0.018	82.1% (NA)	NA	
Duration (weeks)								
8≥	4	289	0.13 (- 0.42, 0.67)	0.649	0.001	81.2% (NA)	NA	0.900
8 <	4	251	0.17 (- 0.09, 0.42)	0.205	0.770	0.0% (NA)	NA	
Dose (mg/d)		•						
2000 ≥	3	157	0.08 (- 0.25,	0.641	0.798	0.0%	NA	0.763
l			<u> </u>					

			0.41)			(NA)		
2000 <	5	383	0.16 (- 0.26, 0.58)	0.454	0.002	76.2% (NA)	NA	
Sample Size								
60 >	3	157	0.08 (- 0.25, 0.41)	0.641	0.798	0.0% (NA)	NA	0.763
<b>60</b> ≤	5	383	0.16 (- 0.26, 0.58)	0.454	0.002	76.2% (NA)	NA	
	Analys	is and subgrou	p results of	Okra supj	plementation on	LDL-C		
Overall effect	8	540	-0.32 (- 0.52, - 0.11)	0.003	0.193	29.4% (0.0%, 69.2%)	0.026	
Country								
Iran	5	378	-0.41 (- 0.67, - 0.15)	0.002	0.177	36.6% (NA)	NA	0.144
Other Countries	3	162	-0.10 (- 0.42, 0.22)	0.539	0.536	0.0% (NA)	NA	0.144
Health Status								
Prediabetic or Diabetic	6	438	-0.34 (- 0.60, -	0.010	0.104	45.3% (NA)	NA	0.629

Without Diabetes2 $102$ $-0.22 (-) \\ 0.64, \\ 0.20)$ $0.310$ $0.467$ Age (year) $-0.27 (-) \\ 0.62, \\ 0.08)$ $0.128$ $0.168$ $50 >$ 4 $232$ $-0.27 (-) \\ 0.62, \\ 0.08)$ $0.128$ $0.168$ $50 \le 4$ $308$ $-0.35 (-) \\ 0.64, -) \\ 0.06)$ $0.018$ $0.190$ Baseline BMI	0.0% (NA) 40.5% (NA) 36.9% (NA)	NA NA NA	0.744
Age (year)       -0.27 (-       0.128       0.168 $50 >$ 4       232 $-0.27 (-$ 0.62,       0.128       0.168 $50 \le 10^{-5}$ 4       308 $-0.35 (-$ 0.018       0.190 $50 \le 10^{-5}$ 4       308 $-0.35 (-$ 0.018       0.190         Baseline BMI $-0.60 ( 1.12,  0.023$ $-$ Healthy weight ( $\le 25$ 1 $60$ $-0.60 ( 1.12,  0.023$ $-$ Overweight or Obese $5$ $350$ $-0.26 ( 0.019$ $0.389$ Not given $2$ $130$ $-0.31 ( 0.406$ $0.037$	(NA) 36.9% (NA)	NA	0.744
50 >       4       232       0.62, $0.128$ 0.128       0.168         50 ≤       4       308       -0.35 (- 0.64, - 0.018)       0.018       0.190         Baseline BMI         Healthy weight (≤25 kg/m2)       1       60       -0.60 (- 1.12, - 0.023)       0.023       -         Overweight or Obese (>25 kg/m2)       1       60       -0.26 (- 0.043)       0.019       0.389         Not given       2       130       -0.31 (- 1.04, 0.406)       0.019       0.337	(NA) 36.9% (NA)	NA	0.744
$50 \le$ 4 $308$ $0.64, -$ $0.06)$ $0.018$ $0.190$ Baseline BMIHealthy weight ( $\le 25$ kg/m2)1 $60$ $-0.60(-$ $1.12, -$ $0.09)$ $0.023$ $-$ Overweight or Obese (>25 kg/m2)5 $350$ $-0.26(-$ $0.48, -$ $0.04)0.0190.389Not given2130-0.31(-1.04, 0.42)0.4060.037$	(NA)		0.744
Healthy weight ( $\leq 25$ kg/m2)160-0.60 (- 1.12, - 0.09)0.023-Overweight or Obese (>25 kg/m2)5350-0.26 (- 0.48, - 0.04)0.0190.389Not given2130-0.31 (- 1.04, 0.42)0.4060.037	-	NA	
Healthy weight ( $\leq 25$ kg/m2)1601.12, - 0.09)0.023-Overweight or Obese (>25 kg/m2)5350-0.26 (- 0.48, - 0.04)0.0190.389Not given2130-0.31 (- 0.42)0.4060.037	_	ΝΔ	
(>25 kg/m2)       5       350       0.48, -       0.019       0.389         Not given       2       130       -0.31 ( -       1.04, 0.406       0.037         0.42)       0.406       0.037       0.42)       0.406       0.037			
Not given         2         130         1.04, 0.42)         0.406         0.037	3.1% (NA)	NA	0.495
Duration (weeks)	77.1% (NA)	NA	
Duration (weeks)	·		·
<b>8</b> ≥ 4 289 $\begin{array}{c} -0.30 (- \\ 0.67, \\ 0.07) \end{array}$ 0.109 0.061	59.2% (NA)	NA	0.000
<b>8</b> < 4 251 -0.36 (- 0.62, - 0.10) 0.498	0.0% (NA)	NA	0.809
Dose (mg/d)			1

		0.22 (					
3	157		0 202	0.767	0.0%	NA	
5	157		0.202	0.707	(NA)		
		,					0.522
5	383	0.67, -	0.020	0.064		NA	
		0.06)			(NA)		
	I	I		I		1	I
		-0.22 (-			0.00/		
3	157	0.54,	0.202	0.767		NA	
		0.12)			(INA)		0.522
		-0.36 (-			55 00/		0.522
5	383	0.67, -	0.020	0.064		NA	
		0.06)			(INA)		
Ar	alysis and sul	ogroup results	of Okra su	opplementation	n on TC		
		-0.45 (-			63.3%		
8	540	0.74, -	0.003	0.008	(0.0%,	0.111	
		0.16)			84.1%)		
		-0.59 (-					
5	378		0.002	0.010		NA	
					(NA)		
							0.100
	1.60		0.004	0.075	3.3%		
3	162	,	0.324	0.356	(NA)	NA	
		0.16)					
[	I	1				1	[
	3 5 At	5       383         5       383         3       157         5       383         5       383         Analysis and sul         8       540         5       378	$ \begin{array}{c ccccccccccccccccccccccccccccccccccc$	3157 $0.54$ , $0.12$ ) $0.202$ 5383 $-0.36$ (- $0.67, -$ $0.06$ ) $0.020$ 3157 $-0.22$ (- $0.54,$ $0.12$ ) $0.202$ 3157 $-0.36$ (- $0.12$ ) $0.202$ 5383 $0.67, -$ 	31570.54, 0.12)0.2020.7675383 $-0.36$ (- 0.67, - 0.06)0.0200.0643157 $-0.22$ (- 0.54, 0.12)0.2020.7673157 $-0.22$ (- 0.54, 0.12)0.2020.7675383 $-0.36$ (- 0.67, - 0.06)0.0200.064-0.36 (- 0.66)0.0200.0645383 $-0.36$ (- 0.67, - 0.06)0.0200.064-0.36 (- 0.06)0.0200.0646 $-0.45$ (- 0.74, - 0.16)0.0030.008-0.45 (- 0.74, - 0.16)0.0030.0085378 $-0.59$ (- 0.97, - 0.21)0.0020.0103162 $-0.17$ (- 0.50, 0.3240.356	3       157       0.54, 0.12)       0.202       0.767 $\binom{0.0\%}{(NA)}$ 5       383 $\stackrel{-0.36}{_{-0.67, -}}$ 0.020       0.064 $55.0\%$ 3       157 $\stackrel{-0.36}{_{-0.66, -}}$ 0.020       0.064 $55.0\%$ 3       157 $\stackrel{-0.22}{_{-0.54, 0.12}}$ 0.202       0.767 $0.0\%$ 3       157 $\stackrel{-0.36}{_{-0.12}}$ 0.202       0.767 $0.0\%$ 5       383 $\stackrel{-0.36}{_{-0.12}}$ 0.202       0.767 $0.0\%$ 5       383 $\stackrel{-0.36}{_{-0.7, -}}$ 0.202       0.767 $0.0\%$ 5       383 $\stackrel{-0.36}{_{-0.67, -}}$ 0.202       0.767 $0.0\%$ 5       383 $\stackrel{-0.36}{_{-0.67, -}$ 0.020       0.064 $55.0\%$ <b>Analysis and subgroup results of Okra supplementation on TC</b> 8       540 $\stackrel{-0.74, -}{_{0.74, -}}$ 0.003       0.008 $\binom{63.3\%}{_{(0.0\%, 84.1\%}}$ 5       378 $\stackrel{-0.59}{_{0.21}$ 0.002       0.010 $\binom{69.6\%}{_{(NA)}}$ 3       162 $\stackrel{-0.17}{_{0.50, 0}$ 0.324       0.356 $3.3\%$	3       157 $0.54, \\ 0.12)$ $0.202$ $0.767$ $0.0\% \\ (NA)$ NA         5       383 $-0.36(-) \\ 0.67, - \\ 0.06)$ $0.020$ $0.064$ $55.0\% \\ (NA)$ NA         3       157 $0.66, \\ 0.06)$ $0.020$ $0.064$ $55.0\% \\ (NA)$ NA         3       157 $-0.22(- \\ 0.54, \\ 0.12)$ $0.202$ $0.767$ $0.0\% \\ (NA)$ NA         5       383 $-0.22(- \\ 0.54, \\ 0.12)$ $0.202$ $0.767$ $0.0\% \\ (NA)$ NA         5       383 $-0.36(- \\ 0.67, - \\ 0.06)$ $0.020$ $0.064$ $55.0\% \\ (NA)$ NA         5       383 $-0.36(- \\ 0.67, - \\ 0.06)$ $0.020$ $0.064$ $55.0\% \\ (NA)$ NA         Analysis and subgrowp results of Okra supplementation on TC $0.066$ $0.003$ $0.008$ $63.3\% \\ (0.0\%, \\ 84.1\%)$ $0.111$ 8       540 $-0.45(- \\ 0.74, - \\ 0.16)$ $0.002$ $0.010$ $69.6\% \\ (NA)$ NA         5       378 $-0.59(- \\ 0.97, - \\ 0.21)$ $0.002$ $0.010$ $69.6\% \\ (NA)$ NA         3       162 $-0.17(- \\ 0.50, $ $0.324$

			0.49.7					
Prediabetic or Diabetic	6	438	-0.48 (- 0.85, - 0.10)	0.013	0.002	73.4% (NA)	NA	0.676
Without Diabetes	2	102	-0.36 (- 0.78, 0.07)	0.097	0.726	0.0% (NA)	NA	
Age (year)								
50 >	4	232	-0.39 (- 0.87, 0.08)	0.102	0.029	66.6% (NA)	NA	0.746
50 ≤	4	308	-0.50 (- 0.92, - 0.08)	0.021	0.019	70.0% (NA)	NA	
Baseline BMI			I					I
Healthy weight (≤25 kg/m2)	1	60	-0.67 (- 1.19, - 0.15)	0.012	-	-	NA	
Overweight or Obese (>25 kg/m2)	5	350	-0.41 (- 0.76, - 0.07)	0.018	0.051	57.6% (NA)	NA	0.721
Not given	2	130	-0.43 (- 1.48, 0.63)	0.428	0.003	88.6% (NA)	NA	
Duration (weeks)								
8≥	4	289	-0.36 (- 0.88,	0.174	0.003	78.8%	NA	0.428

			0.16)			(NA)		
8 <	4	251	-0.59 (- 0.85, - 0.33)	< 0.001	0.476	0.0% (NA)	NA	
Dose (mg/d)							1	
2000 ≥	3	157	-0.45 (- 0.78, - 0.12)	0.008	0.740	0.0% (NA)	NA	0.979
2000 <	5	383	-0.46 (- 0.90, - 0.01)	0.045	0.001	78.4% (NA)	NA	
Sample Size		I	I			I		I
60 >	3	157	-0.45 (- 0.78, - 0.12)	0.008	0.740	0.0% (NA)	NA	0.979
60 ≤	5	383	-0.46 (- 0.90, - 0.01)	0.045	0.001	78.4% (NA)	NA	
	Ar	alysis and sub	ogroup results of	of Okra su	pplementation	n on TG		
Overall effect	8	540	-0.24 (- 0.50, 0.02)	0.069	0.035	53.5% (0.0%, 79.8%)	0.073	
Country	I	<u>I</u>			<u> </u>		<u> </u>	

Iran	5	378	-0.30 (- 0.68, 0.07)	0.112	0.011	69.5% (NA)	NA	0.438
Other Countries	3	162	-0.11 (- 0.43, 0.22)	0.511	0.520	0.0% (NA)	NA	
Health Status						I		
Prediabetic or Diabetic	6	438	-0.24 (- 0.57, 0.09)	0.160	0.011	66.5% (NA)	NA	0.940
Without Diabetes	2	102	-0.26 (- 0.68, 0.16)	0.228	0.742	0.0% (NA)	NA	
Age (year)				1	- 1	I		I
50 >	4	232	-0.18 (- 0.45, 0.09)	0.188	0.597	0.0% (NA)	NA	0.659
50 ≤	4	308	-0.30 (- 0.78, 0.18)	0.217	0.005	77.0% (NA)	NA	
Baseline BMI		_						1
Healthy weight (≤25 kg/m2)	1	60	-0.59 (- 1.10, - 0.07)	0.027	-	-	NA	0.378
Overweight or Obese	5	350	-0.23 (-	0.233	0.024	64.5%		

(- <u>AE1</u> - / - A)			0.00				NT A	
(>25 kg/m2)			0.60,			(NA)	NA	
			0.15)					
Not given			-0.12 (-			35.3%		
not given	2	130	0.55,	0.584	0.214	(NA)	NA	
			0.31)			(INA)		
Duration (weeks)	·	·		·	·	·	·	·
0 >			-0.1 (-			(0.2%)		
8 ≥	4	289	0.52,	0.650	0.021	69.2%	NA	
			0.33)			(NA)		
								0.200
8 <			-0.42 (-			0.0%		
	4	251	0.68, -	0.001	0.787	(NA)	NA	
			0.16)					
Dose (mg/d)		I	I					I
2000 >			-0.35 (-			0.00/		
2000 ≥	3	157	0.68, -	0.037	0.735	0.0%	NA	
			0.02)			(NA)		
								0.528
2000 <			-0.19 (-			70.6%		
	5	383	0.57,	0.323	0.009	(NA)	NA	
			0.19)					
Sample Size	I	I .	1					I
			-0.35 (-			0.00/		
60 >	3	157	0.68, -	0.037	0.735	0.0%	NA	
			0.02)			(NA)		0.528
<b>60</b> ≤	5	383	-0.19 (-	0.323	0.009	70.6%		
			0.57,				NA	

			0.19)			(NA)		
	Analy	sis and subgrou	up results of	f Okra sup	oplementation on A	ALP		
Overall effect	4	266	0.03 (-	0.834	0.209	33.8% (0.0%,	0.034	
Overall effect	4	200	0.28, 0.34)	0.834	0.209	(0.0%, 78.8%)	0.034	
	Analy	sis and subgrou	up results of	f Okra sup	plementation on A	<b>ALT</b>		
Overall effect	6	425	-0.29 (- 0.71, 0.12)	0.164	0.001	76.3% (0.0%, 90.9%)	0.200	
Country		1	,			,		
Iran	3	263	-0.44 (- 1.19, 0.30)	0.241	< 0.001	88.5% (NA)	NA	0.427
Other Countries	3	162	-0.12 (- 0.48, 0.25)	0.531	0.286	20.0% (NA)	NA	- 0.437
Health Status	-	1		-				
Prediabetic or Diabetic	4	323	-0.29 (- 0.88, 0.30)	0.339	< 0.001	85.5% (NA)	NA	- 0.954
Without Diabetes	2	102	-0.31 (- 0.73, 0.11)	0.148	0.569	0.0% (NA)	NA	0.934
Age (year)								
50 >	4	232	-0.44 ( - 1.09, 0.22)	0.195	0.001	82.4% (NA)	NA	0.287
<b>5</b> 0 ≤	2	193	-0.05 (-	0.750	0.674	0.0%		

			0.33,			(NA)	NA	
			0.24)					
Baseline BMI (kg/m <sup>2</sup> )			I	-			1	<sup>1</sup>
Healthy weight (≤25 kg/m²)	-	-	-	-	-	-	NA	
<b>Overweight or Obese</b>			-0.13 (-			0.0%		
(>25 kg/m2)	4	295	0.362,	0.285	0.672	(NA)	NA	0.570
			0.11)					0.570
Not given			-0.55 (-			93.8%		
rot given	2	130	2.00,	0.455	< 0.001	(NA)	NA	
			0.90)					
Duration (weeks)								
8≥			-0.40 (-			89.3%		
0 -	3	229	1.23,	0.346	< 0.001	(NA)	NA	
			0.43)					0.565
8 <			-0.14 (-			0.0%		0.505
	3	196	0.43,	0.345	0.466	(NA)	NA	
			0.15)					
Dose (mg/d)								
<b>2000</b> ≥			-0.31 (-			0.0%		
	2	102	0.73,	0.148	0.569	(NA)	NA	
			0.11)			(1 (1 2)		0.954
2000 <			-0.29 (-			85.5%		
	4	323	0.88,	0.339	< 0.001	(NA)	NA	
			0.30)					
Sample Size								
			-0.31 (-			0.0%		
60 >	2	102	0.73,	0.148	0.569	(NA)	NA	0.954
			0.11)			()		

		1	1		1	1		1
60 ≤	4	323	-0.28 (- 0.88, 0.30)	0.339	< 0.001	85.5% (NA)	NA	
	Analy	sis and subgro	oup results o	f Okra su	pplementation on	AST		
Overall effect	5	365	-0.45 (- 0.73, - 0.17)	0.002	0.159	39.4% (0.0%, 78.4%)	0.040	
Country								
Iran	3	263	-0.48 (- 0.93, - 0.03)	0.037	0.039	69.1% (NA)	NA	- 0.838
Other Countries	2	102	-0.41 ( - 0.84, 0.01)	0.055	0.744	0.0% (NA)	NA	0.838
Health Status			1			I		
Prediabetic or Diabetic	3	263	-0.48 (- 0.93, - 0.03)	0.037	0.039	69.1% (NA)	NA	0.929
Without Diabetes	2	102	-0.41 (- 0.84, 0.01)	0.055	0.744	0.0% (NA)	NA	- 0.838
Age (year)					· ·			·
50 >	3	172	-0.63 (- 1.00, - 0.25)	0.001	0.258	26.1% (NA)	NA	0.150
50 ≤	2	193	-0.27 (- 0.57, 0.03)	0.074	0.293	9.7% (NA)	NA	— 0.150
Baseline BMI								

Healthy weight (≤25 kg/m2)	-	-	-	-	-	-	NA	
Overweight or Obese (>25 kg/m2)	4	295	-0.32 ( - 0.55, - 0.08)	0.008	0.682	0.0% (NA)	NA	0.024
Not given	1	70	-0.95 (- 1.44, - 0.45)	< 0.001	-	-	NA	
Duration (weeks)		I						
8≥	2	169	-0.66 (- 1.18, - 0.15)	0.011	0.105	61.8% (NA)	NA	0.170
8 <	3	196	-0.26 (- 0.55, 0.03)	0.082	0.583	0.0% (NA)	NA	0.179
Dose (mg/day)		I						I
2000 ≥	2	102	-0.41 (- 0.84, 0.01)	0.055	0.744	0.0% (NA)	NA	0.838
2000 <	3	263	-0.48 (- 0.93, - 0.03)	0.037	0.039	69.1% (NA)	NA	0.838
Sample Size								
60 >	2	102	-0.41 (- 0.84, 0.01)	0.055	0.744	0.0% (NA)	NA	0.929
60 ≤	3	263	-0.48 (- 0.93, - 0.03)	0.037	0.039	69.1% (NA)	NA	0.838

	Analys	sis and subgro	oup results of O	kra suppl	ementation on	Creatinine			
Overall effect	5	326	-0.12 (- 0.36, 0.12)	0.327	0.346	10.6% (0.0%, 67.9%)	0.008		
Country		I							
Iran	2	164	-0.25 (- 0.71, 0.21)	0.288	0.139	54.4% (NA)	NA	0.220	
Other Countries	3	162	0.03 (- 0.29, 0.35)	0.854	0.747	0.0% (NA)	NA	— 0.329	
Health Status		I	1		1	I	_	I	
Prediabetic or Diabetic	3	224	-0.17 (- 0.50, 0.16)	0.301	0.213	35.4% (NA)	NA		
Without Diabetes	2	102	0.04 (- 0.38, 0.46)	0.864	0.446	0.0% (NA)	NA	— 0.439	
Age (year)			1		1	I	_	I	
50 >	4	232	0.02 (- 0.25, 0.29)	0.880	0.898	0.0% (NA)	NA	0.040	
50 ≤	1	94	-0.47 (- 0.88, - 0.06)	0.025	-	-	NA	— 0.049	
Baseline BMI	1	1	1		1	1	1	1	
Healthy weight (≤25 kg/m2)	-	-	-	-	-	-	NA	0.479	
<b>Overweight or Obese</b>	3	196	-0.18 (-	0.375	0.178	42.1%			

(>25 kg/m2)			0.58,			(NA)	NA	
(~25 Kg/112)			0.30,					
			0.01 ( -					
Not given	2	130	0.33,	0.956	0.952	0.0%	NA	
	_		0.35)		0.702	(NA)		
Duration (weeks)						I		I
			0.01 ( -			0.00/		
8≥	2	130	0.33,	0.956	0.952	0.0%	NA	
			0.35)			(NA)		0.479
8 <			-0.18 (-			42.1%		0.479
0<	3	196	0.58,	0.375	0.178	42.1% (NA)	NA	
			0.22)			(INA)		
Dose (mg/d)	·							
2000 ≥			0.04 (-			0.0%		
2000 2	2	102	0.38,	0.864	0.446	(NA)	NA	
			0.46)					0.439
2000 <			-0.17 (-			35.4%		0.437
2000 <	3	224	0.50,	0.301	0.213	(NA)	NA	
			0.16)					
Sample Size								
			0.04 (-			0.0%		
60 >	2	102	0.38,	0.864	0.446	(NA)	NA	
			0.46)			(111)		0.439
<b>60</b> ≤			-0.17 (-			35.4%		0.139
00 2	3 224	0.50,	0.301	0.213	(NA)	NA		
			0.16)					

Footprint: NA, not applicable

Outcomes	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Number (INT/CON)	SMD (95%CI)	Quality of evidence
BMI	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Not serious	Publication bias strongly suspected <sup>c</sup>	258 / 207	-0.70 (- 1.23, - 0.16)	⊕ Very low
FFM	Very serious <sup>a</sup>	Not serious	Not serious	Very serious	None	130 / 82	-0.13 (- 0.41, 0.15)	How Very low
FM	Very serious <sup>a</sup>	Not serious	Not serious	Serious <sup>e</sup>	None	130 / 82	-0.74 (- 1.13, - 0.36)	
НС	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Serious <sup>e</sup>	None	130 / 82	-0.85 (- 1.41, - 0.28)	How Very low
WC	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Very serious	None	180 / 131	-0.57 (- 1.15, 0.01)	How Very low
Weight	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Serious <sup>e</sup>	Publication bias strongly suspected <sup>c</sup>	190 / 137	-0.77 (- 1.42, - 0.11)	⊕ Very low

DBP	Very serious <sup>a</sup>	Not serious	Serious <sup>f</sup>	Very serious	None	158 / 150	-0.15 (- 0.37, 0.08)	⊕ Very low
SBP	Very serious <sup>a</sup>	Serious <sup>b</sup>	Serious <sup>f</sup>	Very serious	None	158 / 150	-0.13 (- 0.56, 0.30)	⊕ Very low
Fasting insulin	Very serious <sup>a</sup>	Not serious	Not serious	Serious <sup>e</sup>	None	108 / 106	-0.35 (- 0.63, - 0.07)	
FBS	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Not serious	None	292 / 243	-1.07 (- 1.75, - 0.38)	
HbA1c	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Not serious	Publication bias strongly suspected <sup>c</sup>	228 / 187	-0.38 ( - 0.71, - 0.05)	⊕ Very low
HOMA- IR	Very serious <sup>a</sup>	Not serious	Not serious	Serious <sup>e</sup>	Publication bias strongly suspected <sup>c</sup>	108 / 106	-0.56 (- 0.84, - 0.29)	⊕ Very low
HDL-C	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Serious <sup>d</sup>	None	293 / 247	0.13 (- 0.15, 0.41)	⊕ Very low
LDL-C	Very serious <sup>a</sup>	Not serious	Not serious	Not serious	None	293 / 247	-0.32 (- 0.52, - 0.11)	⊕⊕⊕⊖ Moderate

TC	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Not serious	None	293 / 247	-0.45 (- 0.74, - 0.16)	
TG	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Serious <sup>d</sup>	None	293 / 247	-0.24 (- 0.50, 0.02)	⊕ Very low
ALP	Very serious <sup>a</sup>	Not serious	Not serious	Very serious	None	153 / 113	0.03 (- 0.28, 0.34)	⊕ Very low
ALT	Very serious <sup>a</sup>	Serious <sup>b</sup>	Not serious	Serious <sup>d</sup>	None	233 / 192	-0.29 (- 0.71, 0.12)	⊕ Very low
AST	Very serious <sup>a</sup>	Not serious	Not serious	Serious <sup>e</sup>	None	203 / 162	-0.45 (- 0.73, - 0.17)	
Creatinine	Very serious <sup>a</sup>	Not serious	Not serious	Very serious	None	183 / 143	-0.12 (- 0.36, 0.12)	How Very low

Footprint: ALP, Alkaline Phosphatase; ALT, Alanine Aminotransferase; AST, Aspartate Aminotransferase; BMI, Body Mass Index; CI, Confidence Interval; CON, Control Group; DBP, Diastolic Blood Pressure; FBS, Fasting Blood Sugar; FFM, Fat-Free Mass; FM, Fat Mass; HbA1c, Hemoglobin A1C; HC, Hip Circumference; HDL-C, High-Density Lipoprotein Cholesterol; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance; INT, Intervention Group; LDL-C, Low-Density Lipoprotein Cholesterol; SBP, Systolic Blood Pressure; SMD, Standardised Mean Difference; TC, Total Cholesterol; TG, Triglycerides; WC, Waist Circumference.

#### **Explanations**

Explanations

- a. Downgraded since more than 50% of the participants were from high risk of bias studies.
- b. The I2 value was >50% (or Heterogeneity among the studies was high)
- c. Publication Bias was detected through Egger and Begg's test. (p-value < 0.05)
- d. Downgraded since the 95% CI crosses the threshold of interest.
- e. Downgraded since the participants included were less than 400 persons.
- f. Downgraded for indirectness in country.

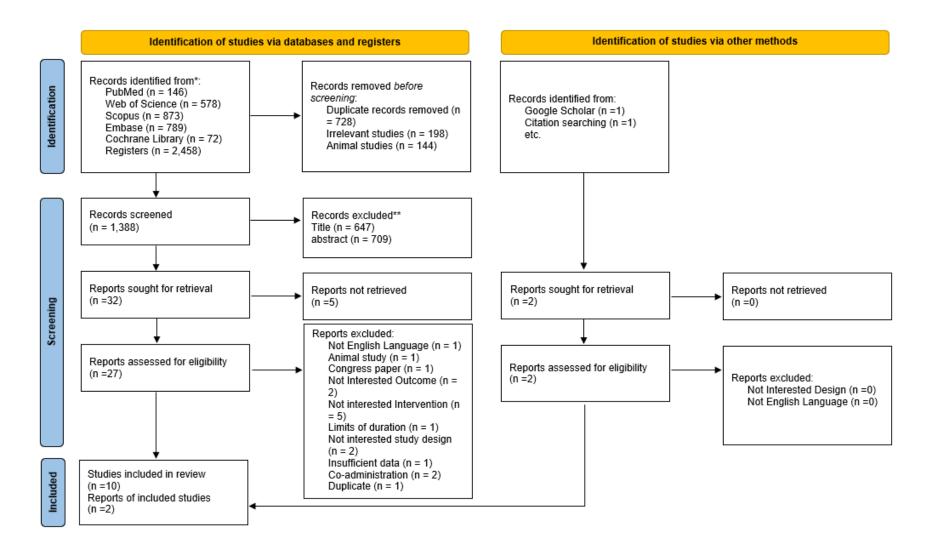
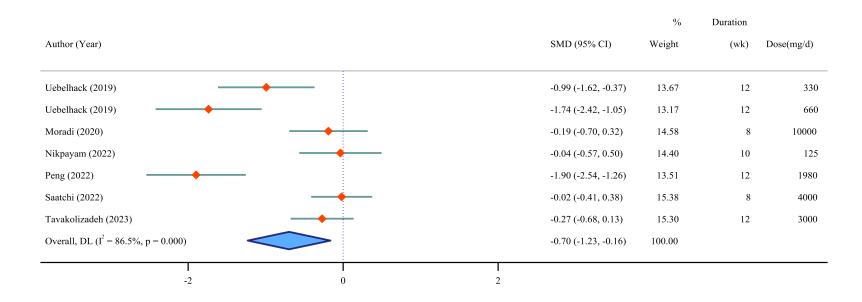
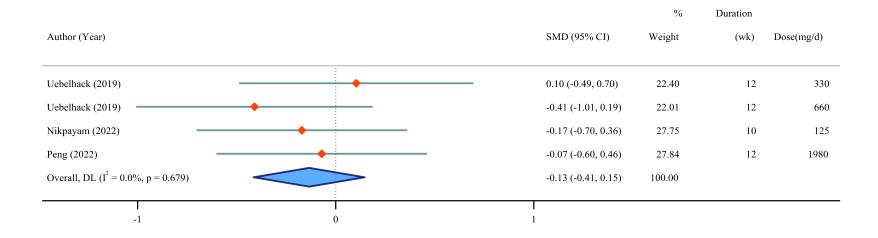
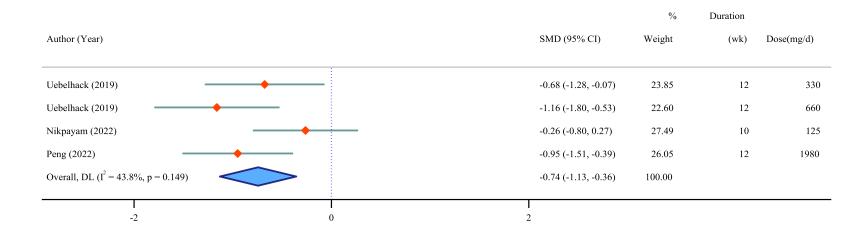


FIGURE 1. Flowchart of study selection for inclusion trials in the systematic review.

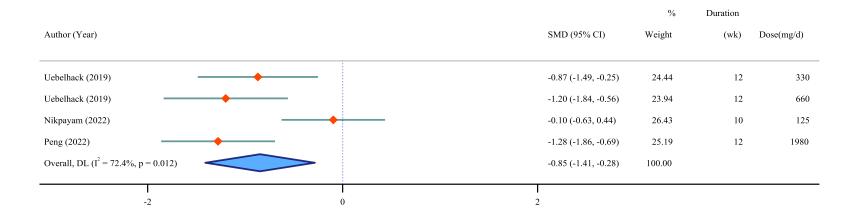
(a)

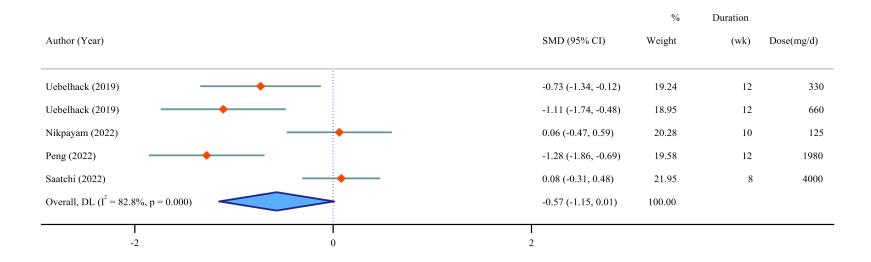




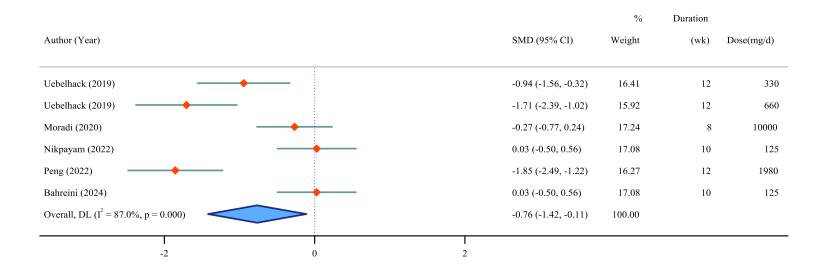


# (d)

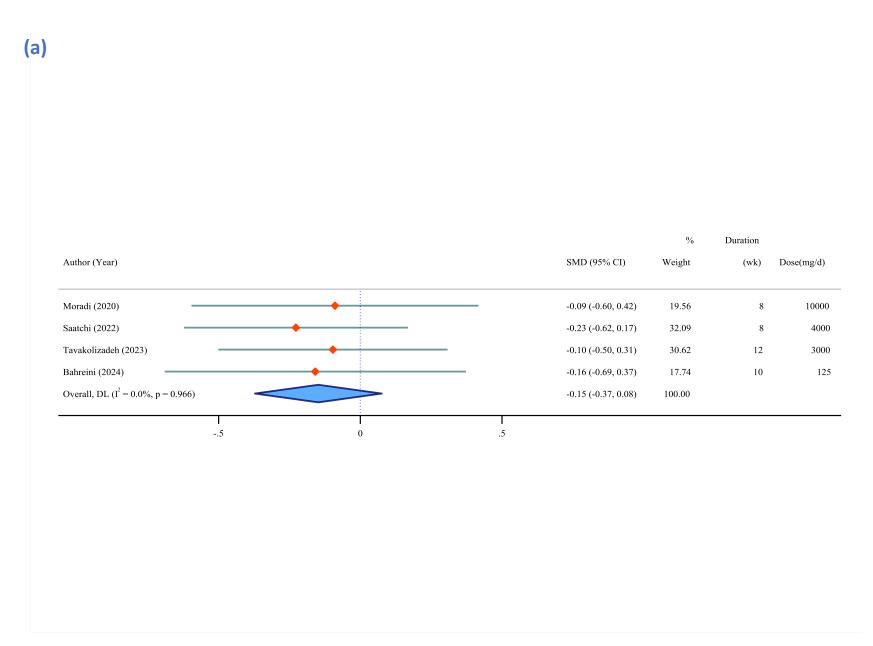








#### Figure 2. Forest plot of the effects of okra supplement on anthropometric measures (a: BMI, b: FFM, c: FM, d: HC, e: WC, f: Weight)





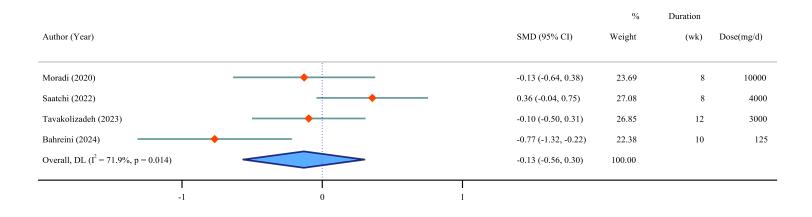
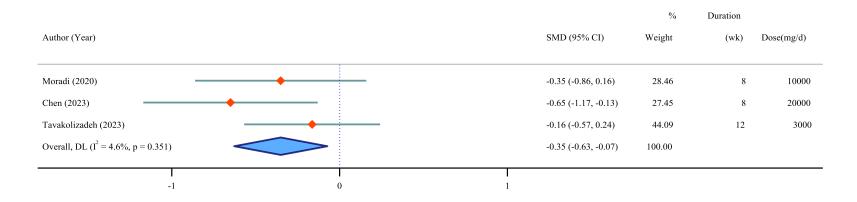


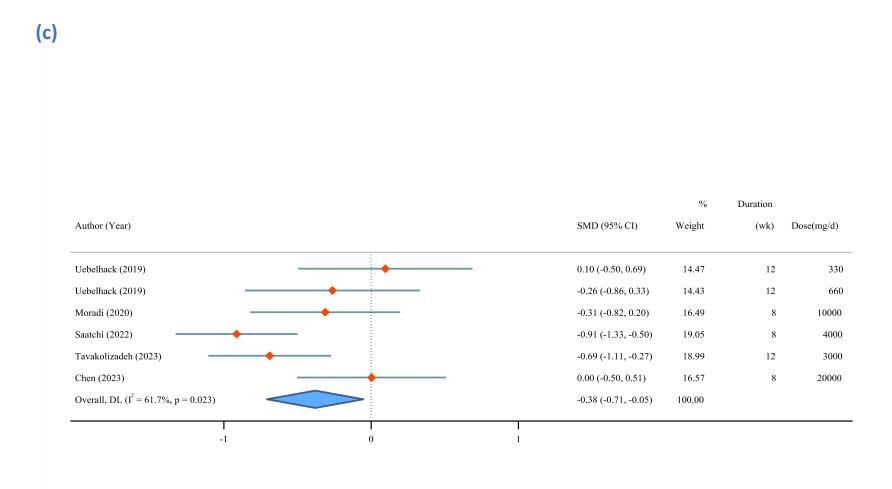
Figure 3. Forest plot of the effects of okra supplement on blood pressure (a: DBP, b: SBP)





(b)

		%	Duration	
Author (Year)	SMD (95% CI)	Weight	(wk)	Dose(mg/d)
Uebelhack (2019)	0.25 (-0.35, 0.84)	10.39	12	330
Uebelhack (2019)	0.21 (-0.39, 0.80)	10.39	12	660
Khodija (2020)	-1.86 (-2.94, -0.78)	8.80	2	40000
Khodija (2020)	-1.80 (-2.87, -0.73)	8.84	2	40000
Moradi (2020)	-0.55 (-1.06, -0.03)	10.59	8	10000
Saatchi (2022)	-1.10 (-1.52, -0.67)	10.80	8	4000
Salarfard (2023)	-4.16 (-5.07, -3.25)	9.40	4	6000
Chen (2023)	-0.38 (-0.89, 0.13)	10.60	8	20000
Tavakolizadeh (2023)	-1.82 (-2.30, -1.34)	10.67	12	3000
Damayanthi (2024)	0.12 (-0.76, 0.99)	9.51	4	3800
Overall, DL ( $I^2 = 91.7\%$ , p = 0.000)	-1.07 (-1.75, -0.38)	100.00		
-5 0	5			



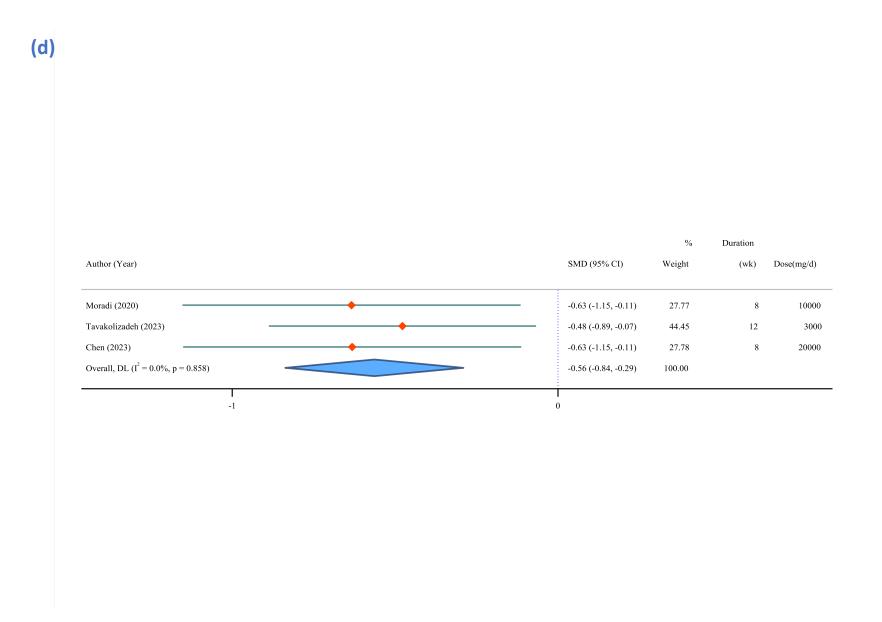
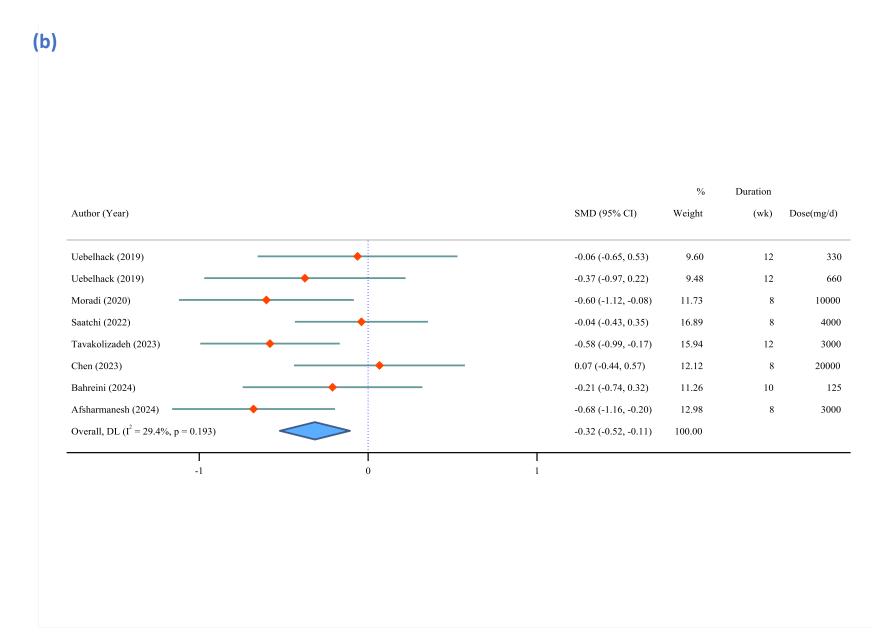


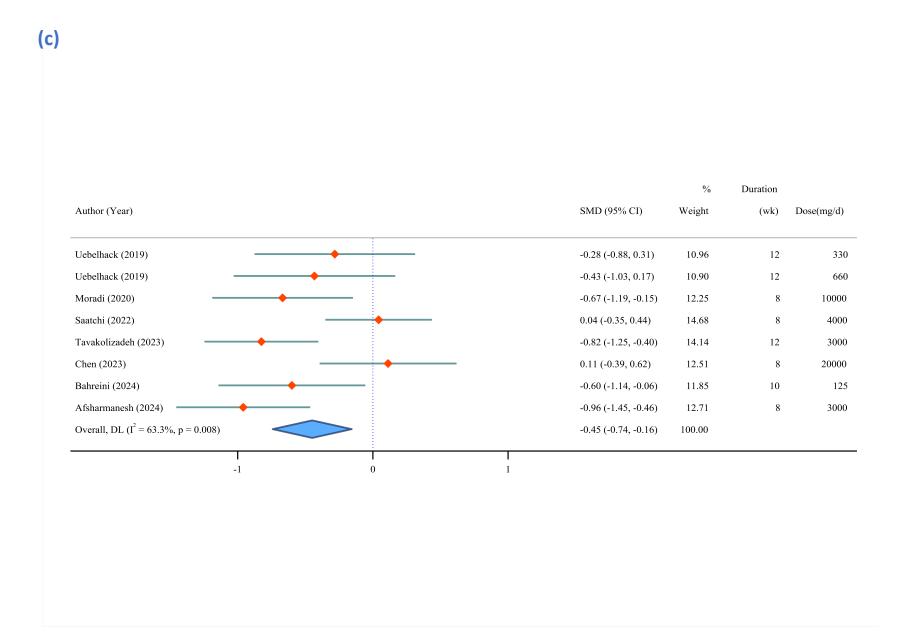
Figure 4. Forest plot of the effects of okra supplement on glycemic profile (a: Fasting insulin, b: FBS, c: HbA1c, d: HOMA-IR)



		%	Duration	
Author (Year)	SMD (95% CI)	Weight	(wk)	Dose(mg/d)
Uebelhack (2019)	-0.05 (-0.64, 0.54)	10.74	12	330
Uebelhack (2019)	0.23 (-0.36, 0.83)	10.70	12	660
Moradi (2020)	0.02 (-0.49, 0.52)	12.38	8	10000
Saatchi (2022)	-0.40 (-0.80, 0.00)	14.72	8	4000
Tavakolizadeh (2023)	0.30 (-0.11, 0.70)	14.53	12	3000
Chen (2023)	0.04 (-0.47, 0.54)	12.38	8	20000
Bahreini (2024)	0.06 (-0.47, 0.59)	11.88	10	125
Afsharmanesh (2024)	0.89 (0.40, 1.38)	12.67	8	3000
Overall, DL ( $l^2 = 59.7\%$ , p = 0.015)	0.13 (-0.15, 0.41)	100.00		



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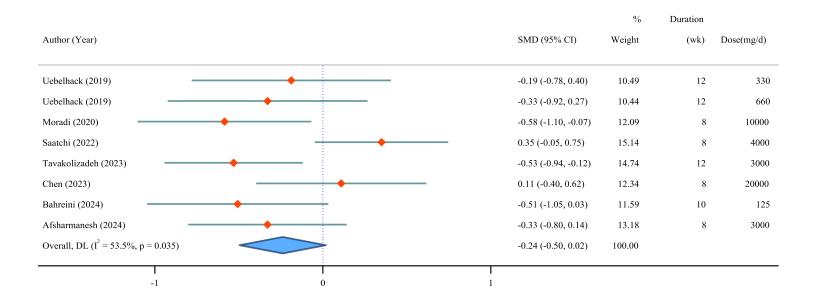


Figure 5. Forest plot of the effects of okra supplement on lipid profile (a: HDL-C, b: LDL-C, c: TC, d: TG)

