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ORIGINAL ARTICLE

# Efficacy and Safety on *Moringa oleifera* on Blood Glucose and Lipid profile: A Meta-analysis

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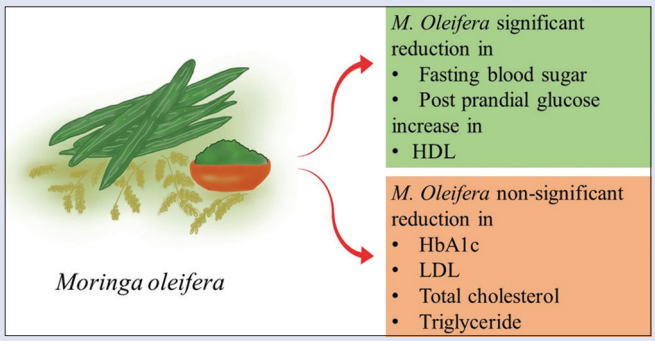
## ABSTRACT

**Background:** Results from previous clinical trials in which the effects of *Moringa oleifera* (MO) on blood glucose and lipid profile were investigated are controversial. **Objectives:** The main objective of this study was to assess the effects of MO consumption on blood glucose level and lipid profile in randomized controlled trial (RCTs) and non-RCTs. **Materials and Methods:** A comprehensive systematic review was performed by searching the PubMed, ScienceDirect, Scopus, and Thai Library Integrated System databases up to December 2019 without any language restrictions by two independent authors. The DerSimonian and Laird random-effects model method was used to pool the results. **Results:** Seven trials with 257 participants and treatment duration of 28–90 days were included. The pooled results showed a significant reduction in fasting blood sugar (FBS; weighted mean difference [WMD]: −14.81 mg/dL; 95% confidence interval [CI]: −27.99, −1.63; $I^2$ = 97.8%), postprandial glucose (PPG) (WMD − 64.73 mg/dL; 95% CI: −102.87, −26.59; $I^2$ = 93%) and no significant change in $HbA_1C$ (WMD: 0.70%; 95% CI: −1.42, 0.69; $I^2$ = 99%), low-density lipoprotein (WMD − 11.20 mg/dL; 95% CI: −34.12, 11.72; $I^2$ = 8.08%), total cholesterol (WMD − 4.73 mg/dL; 95% CI: −24.96, 15.49; $I^2$ = 80%), and triglycerides (WMD − 3.29 mg/dL; 95% CI: −9.95, 3.36; $I^2$ = 29%). Moreover, MO treatment increased high-density lipoprotein (HDL) level significantly (WMD 2.15 mg/dL; 95% CI: 1.92, 2.39; $I^2$ = 0%). No serious adverse effects of the intervention were reported. **Conclusion:** The results of our study suggested that MO treatment decreased FBS, PPG levels and increase HDL level. However, the long-term benefits and safety of the treatment remain to be determined.

**Key words:** Blood glucose, efficacy, lipid profile, meta-analysis, *Moringa oleifera,* safety

## SUMMARY

- The results of this study showed that *Moringa oleifera* treatment decreased fasting blood sugar and postprandial glucose and had no serious adverse effects.




**Abbreviations used:** MO: *Moringa oleifera*; RCT: Randomized controlled trial; ThaiLis: Thai Library Integrated System; FBS: Fasting blood sugar; PPG: Postprandial glucose; LDL: Low-density lipoprotein; HDL: High-density lipoprotein; TC: Total cholesterol; TG: Triglyceride; PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses; MeSH: Medical Subject Heading; WMD: Weighted mean difference.

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## INTRODUCTION

*Moringa oleifera* (MO) is one of a widely used herbal medicine in Asia. Moreover, Ayurvedic medicine considered MO is a useful medicinal plant and has potential for application in the development of medicine in the modern era.[1,2] The major constituents of MO are moringinine, quercetin, chlorogenic acid, niaziminin, and aurantiamide.[3] MO has a wide spectrum of biological activities including antibacterial, antifungal, antiviral, anti-inflammatory, antioxidant, anti-hyperglycemic, and anti-hyperlipidemic effects, and has been used in traditional medicine.[3-5]

Several studies have shown the anti-hyperglycemic and anti-hyperlipidemia effects of MO in different preclinical models of hyperglycemia and dyslipidemia.[6-9] The multitude of mechanisms that underlie the effects of MO mostly include improved insulin sensitivity, glucagon synthase activity, and glucose uptake, and inhibition of α-amylase, α-glucosidase, β-hydroxy β-methylglutaryl-CoA (HMG-CoA) reductase, and enhanced endocytosis of low-density lipoprotein cholesterol (LDL) by activation of LDL receptor.[5,10-12] Some clinical trials demonstrated that MO improved fasting blood sugar (FBS) levels and lipid profile significantly,[13,14] whereas some others reported that it has no or negative effects.[15,16]

In this regard, several clinical trials have demonstrated that MO reduces blood glucose and lipid level, even if the results relatively varied across trials. Therefore, to resolve the inconsistencies in MO's effects on blood glucose levels and lipid profile, we proposed performing a systematic review and meta-analysis of published clinical trials both randomized controlled trials (RCTs) and non-RCTs.



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## MATERIALS AND METHODS

This systematic review was conducted according to the Cochrane Collaboration framework guidelines[17] and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.[18]

### Search strategies and study selection

The following databases were searched for articles from their inception to December 2019: PubMed, ScienceDirect, Scopus, and Thai Library Integrated System. The search algorithms for each database were developed and modified using relevant search terms combined with related the Cochrane Handbook for Systematic Reviews of RCTs.

Search strings or strategies for searching in each database were clearly reported. For the strategy for searching, we used the Medical Subject Heading terms "*Moringa oleifera*," OR/AND "blood glucose," "lipid profile." To ensure a thorough search, we also use hand searching for the included studies' reference lists or any previous reviews. The inclusion criteria were RCTs and non-RCTs with controlled groups investigating any MO formulation's clinical effects, regardless of the length of the study, age range or average age, and dose of MO. The exclusion criteria were the studies with insufficient data. Title and the article abstracts were searched to define the studies that evaluated MO's effect on blood glucose or lipid profile. Then, two researchers (WP, KW) assessed the full-text of potential trials independently. If there are disagreement between two researchers. It will be resolved by discussion with a third person (BS).

### Data extraction and quality assessment

The standard extraction form, which consistent with the CONSORT statement for reporting herbal medicinal interventions[19] was used for data extraction. The main information was extracted for the individual articles: Authors, year of publication, trial design, participant characteristics, intervention, sample size, treatment duration, and the measurement of outcome. The Cochrane risk-of-bias tool[17] and Jadad scale[20] were used to assess the methodological quality of the recruited studies in this meta-analysis. The risk of bias was assessed based on the Cochrane criteria for including: (1) sequence generation, (2) allocation concealment, (3) participant and personnel blinding, (4) blinded the outcome assessment, (5) reported the incomplete outcome data, (6) selective reporting, and (7) other sources of bias.[17] The overall risk of bias for individual trial was rated as low risk, high risk, and unclear risk. The Jadad scale consisting of 5 characteristics was applied to assess the quality of the recruited studies. Five characteristics of the

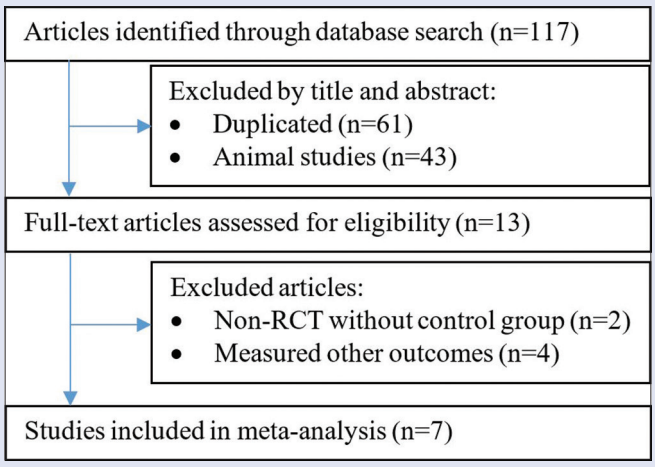


**Figure 1:** Flow diagram of studies selection

**Table 1:** Characteristics of the included studies

| Authors, years | Country | Studies design | Participants | Age (years); range or average | Duration (days) | Intervention | | Outcome measure | Jadad Scale |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | | Experimental group (*n*) | Control group (*n*) | | |
| Kumari, 2010[13] | India | Non-RCT | DM Type II | 41-60 | 40 | MO leaves powder 8 g/day (23) | Placebo (9) | FBS, PPG lipid profile | 2 |
| Nambiar *et al.*, 2010[25] | India | RCT | Hyperlipidemia | 49.5 | 50 | DDT 575 mg 8 tablets/day (20) | No supplement (20) | FBS, lipid profile | 3 |
| Giridhari *et al.*, 2011[26] | India | Non-RCT | DM Type II | 40-58 | 90 | Sulfonylurea drug+MO leaf tablets/day (30) | Sulfonylurea drug (30) | FBS, $HbA_{1C}$ | 2 |
| Sandoval and Jimeno 2013[15] | The Philippines | RCT | LDL >100 (mg/dL) | 18-55 | 30 | MO capsule 2100 mg/day (33) | Placebo (35) | FBS, lipid profile, AEs | 4 |
| Baipakdee, 2013[27] | Thailand | Non-RCT | Pre-DM | 30-60 | N/A | MO capsule 1500 mg/day (15) | Placebo (15) | FBS, PPG | 2 |
| Kushwaha *et al.*, 2014[14] | India | RCT | Menopause | 45-60 | 90 | MO powder 7 g/day (30) | Placebo (30) | FBS, $HbA_{1c}$ | 3 |
| Taweerutchana *et al.*, 2017[16] | Thailand | RCT | DM Type II | 54.5 | 28 | MO leaf capsule 8 g/day (16) | Placebo (16) | FBS, $HbA_{1c}$ | 4 |

RCT: Randomized control trial; MO: *Moringa oleifera*; DDT: Dehydrated drumstick leaf tablets; N/A: Not available; FBS: Fasting blood sugar; LDL: Low-density lipoprotein, DM: Diabetes mellitus; AE: Adverse events, PPG: Postprandial glucose, $HbA_{1C}$: Hemoglobin A1c

**Table 2:** Meta-analysis of effects of *Moringa oleifera* on lipid outcomes

| Outcomes | WMD (95% CI) | $P^a$ | $I^2$ (%) | $P^b$ | References |
|---|---|---|---|---|---|
| LDL | −11.20 (−34.12-11.72) | 0.34 | 8.08 | 0.0003 | [13,15,25] |
| HDL | 2.15 (1.92-2.39) | 0.01 | 0.0 | <0.00001 | [13,15,25] |
| Total cholesterol | −4.73 (−24.96-15.49) | 0.65 | 80.0 | 0.007 | [13,15,25] |
| Triglyceride | −3.29 (−9.95-3.36) | 0.33 | 29.0 | 0.24 | [13,15,25] |

[a]*P* value of WMD; [b]*P* value of heterogeneity. WMD: Weighted mean difference, CI: Confidence interval, LDL: Low-density lipoprotein, HDL: High-density lipoprotein

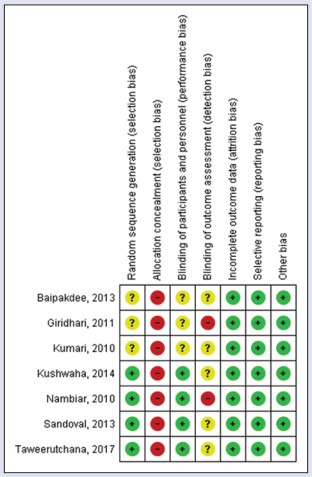


**Figure 2:** Risk of bias summary from individual studies (+ = low risk, − = high risk and ? = unclear)

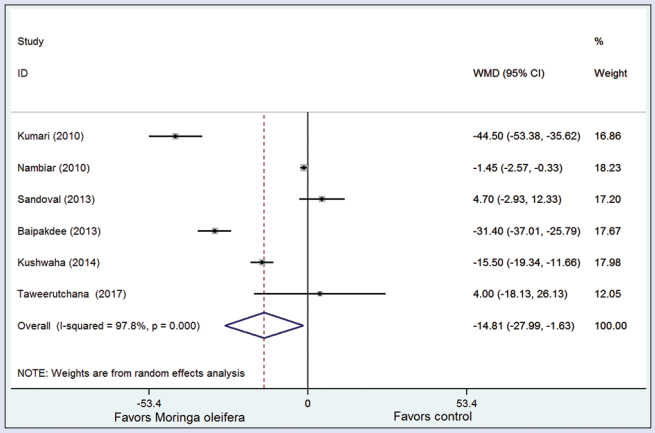


**Figure 3:** Forest plot detailing weighted mean difference and 95% confidence interval for impact of *Moringa oleifera* on fasting blood sugar

RCTs were considered: (1) randomization process statement, (2) a randomized sequence was generate appropriately, (3) double-blinding process was used, (4) the double-blinding method was described, and (5) reported the withdrawals and dropouts details. The recruited trials with Jadad scale ranged from three to five score were consider as good quality.[20]

## Outcome measurement

The primary outcomes of interest included: (1) fasting plasma glucose, $HbA_{1C}$, and postprandial glucose (PPG) levels and lipid profile including LDL, high-density lipoprotein (HDL), total cholesterol (TC), and triglyceride (TG). (2) The secondary outcome was adverse events.

## Statistical analysis

Pooled effects were calculated and stratified according to blood glucose and lipid profile control associated with MO and its comparators. Weighted mean difference (WMD) was used for analyzing the continuous outcome. The Chi-squared test and $I^2$ test were used for the heterogeneity evaluation among the included studies. The Chi-squared test and $I^2$ value of 0%–50% and 51%–100% were classified as homogeneity and statistical heterogeneity, respectively.[21] In case of statistical heterogeneity found, we performed a subgroup analysis to explore the underlying reason, if applicable. The visual inspection of funnel plots was used to examine publication bias.[22,23] All primary outcomes were analyzed by the DerSimonian and Laird random-effects model. Statistical analyses were performed with (StataCorp. Stata Statistical Software: Release 14. College Station, TX, USA) and Review Manager (Revman®) version 5.3 (Cochrane Collaboration, Oxford, UK). A sensitivity analysis was analyzed using a fixed-effect model[24] and data from low-quality studies were excluded to ensure the robustness of results. In addition, a subgroup analysis was performed based on the duration of treatment.

# RESULTS

The diagram of PRISMA flow of study analysis is illustrated in Figure 1. The 117 related studies were retrieved according to search strategy and selection through the above-mentioned database. After removal of duplicate trials, 43 trials were recruited for the screening step. Based on the screening titles and abstracts process, 13 studies were selected for a full-text review. A total of six studies were excluded from the full-text review because two studies were non-RCTs without controlled group and four studies evaluated other clinical outcomes. Therefore, seven studies were included in this meta-analysis.

## Characteristics of the recruited studies

Table 1 shows the characteristics of the seven recruited studies. The studied included a total of 257 participants, of which 89 had type 2 diabetes mellitus and 108 had dyslipidemia. All the studies were single-blinded RCTs published between 2010 and 2017 and included four studies[13,14,25,26] conducted in India, two in Thailand,[16,27] and one in the Philippines.[15] The participants' mean age ranged from 18 to 60 years; the follow-up duration was 28–90 days. The dosage preparation and dose of MO used different among included studies. Three trials examined powder,[13,14,26] three others used capsules,[15,16,27] and one used tablets as interventions. The control group of all trials received a placebo.[25] Only one study reported the quantity of active constituent of MO extract used, 3700 μg of beta carotene and 1775 mg of total phenols.[25]

## Quality of included studies

Based on the criteria of the Cochrane risk of bias, most trials (4/7) were classified as low risk of bias on random sequence generation and blinding of participants and personnel. All studies were rated as high risk

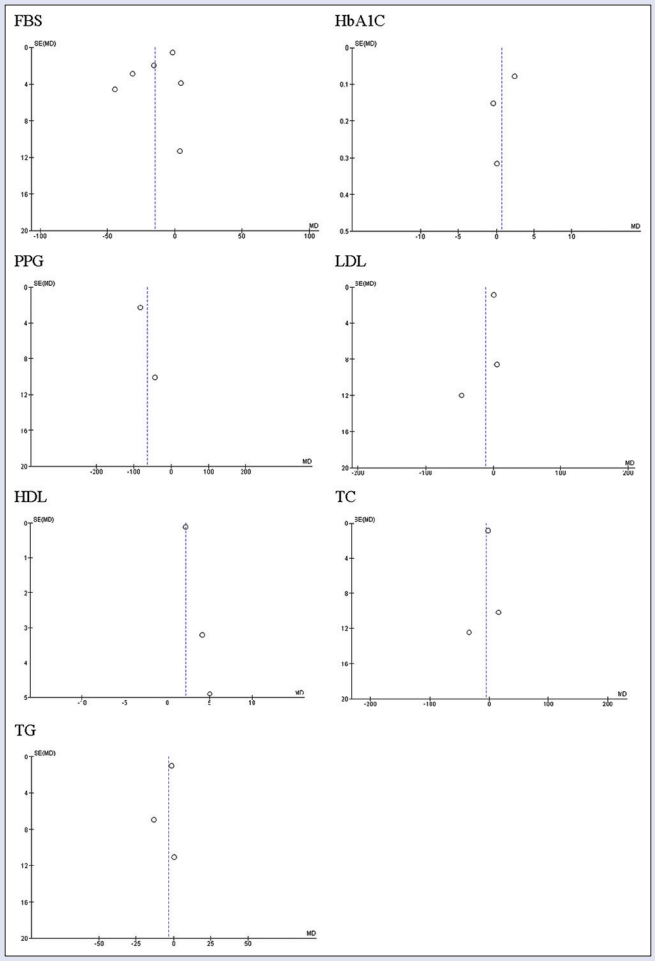


**Figure 4:** Funnel plots detailing publication bias

of bias on allocation concealment [Figure 2]. Moreover, four studies were rated as unclear and three trials were not described on blinding outcome assessment, therefore, they were classified as high risk on this domain. The Jadad scale score for most studies (4/7) ranged from 3 to 5 for a total of five scores [Table 1].

## Effect of *Moringa oleifera* on blood glucose level

Pooled effect size based on six studies including 257 participants indicated that MO treatment significantly decreased FBS (WMD: −14.81 mg/dL; 95% confidence interval [CI]: −27.99, −1.63) compared to that in the comparator group [Figure 3]. However, there was no difference in the $HbA_{1C}$ outcome between the MO intervention and placebo groups (WMD: 0.70%; 95% CI: −1.42, 0.69). Moreover, the meta-analysis demonstrated that MO treatment tended to decrease (PPG; −64.73 mg/dL; 95% CI: −102.87, −26.59). Heterogeneity was observed in these outcomes ($I^2 > 50\%$).

## Effect of *Moringa oleifera* treatment on lipid profile

The meta-analysis showed that MO treatment tended to reduce LDL (−11.20 mg/dL; 95% CI: −34.12, 11.72; $I^2 = 88\%$), TC (−4.73 mg/dL; 95% CI: −24.96, 15.49; $I^2 = 80\%$), TG (−3.29 mg/dL; 95% CI: −9.95, 3.36; $I^2 = 29\%$) to a greater extent that did placebo; however, the intergroup differences were not significant. However, MO could increase HDL 2.15 mg/dL (95% CI: 1.92, 2.39; $I^2 = 0\%$) higher than placebo group significantly [Table 2].

**Table 3:** Subgroup analysis

| | Number of studies (*n*); WMD; 95% CI; $I^2$ | | | | | | |
|---|---|---|---|---|---|---|---|
| | FBS | $HbA_{1C}$ | PPG | LDL | HDL | TC | TG |
| MO dosage form | | | | | | | |
| Tablets or capsules | 2; −6.86 mg/dL; (−24.22-10.51); $I^2$=97% | 1; 0.07%; (−0.55-0.69); $I^2$=N/A | N/A | 2; 0.45 mg/dL; (−1.31-2.20); $I^2$=0% | 2; 2.15 mg/dL; (1.91-2.39); $I^2$=0%* | 2; 4.47 mg/dL; (−11.94-20.87); $I^2$=67% | 2; −1.26 mg/dL; (−3.30-0.79); $I^2$=0% |
| Powders | 4; −29.71 mg/dL; (−58.13-−1.30); $I^2$=97%* | 2; 1.01%; (−1.73-3.74); $I^2$=100% | 2; −64.73 mg/dL; (−102.87-−26.59); $I^2$=93% | 1; −47.00 mg/dL; (−70.44-−23.56); $I^2$=N/A* | 1; 5.00 mg/dL; (−4.59-14.59); $I^2$=N/A | 1; −34.00 mg/dL; (−58.36-−9.64); $I^2$=N/A* | 1; −13.00 mg/dL; (−26.59, 0.59); $I^2$=N/A |
| Treatment period (days) | | | | | | | |
| ≤30 | 3; −8.29 mg/dL; (−36.90-20.31); $I^2$=97% | 1; 0.07%; (−0.55-0.69); $I^2$=N/A | N/A | 1; 5.52 mg/dL; (−11.34-22.38); $I^2$=N/A | 1; 4.16 mg/dL; (−2.12-10.44); $I^2$=N/A | 1; 16.23 mg/dL; (−3.73-36.19); $I^2$=N/A | 1; 0.30 mg/dL; (−21.44-22.04); $I^2$=N/A |
| >30 | 3; −19.84 mg/dL; (−37.22-−2.45); $I^2$=98%* | 2; 1.01%; (−1.73-3.74); $I^2$=100% | 2; −64.73 mg/dL; (−102.87-−26.59); $I^2$=93% | 2; −21.81 mg/dL; (−68.15-24.54); $I^2$=94% | 2; 2.15 mg/dL; (1.91-2.39); $I^2$=0% | 2; −15.38 mg/dL; (−46.90-16.14); $I^2$=85% | 2; −5.13 mg/dL; (−15.94-5.67); $I^2$=64% |
| Study design | | | | | | | |
| Non-RCT | 2; −37.48 mg/dL; (−50.28-−24.67); $I^2$=83% | 1; −0.39%; (−0.69-−0.09); $I^2$=N/A | 2; −64.73 mg/dL (−102.87-−26.59); $I^2$=93% | 1; −47.00 mg/dL; (−70.44-−23.56); $I^2$=N/A* | 2; 2.15 mg/dL; (1.91-2.39); $I^2$=0% | 2; −15.38 mg/dL; (−46.90-16.14); $I^2$=85% | 2; −5.13 mg/dL; (−15.94-5.67); $I^2$=64% |
| RCT | 4; −3.40 mg/dL; (−13.14-6.35); $I^2$=94% | 2; 1.25%; (−1.03-3.54); $I^2$=98% | N/A | 2; 0.45 mg/dL; (−1.31-2.20); $I^2$=0% | 1; 4.16 mg/dL; (−2.12-10.44); $I^2$=N/A | 1; 16.23 mg/dL; (−3.73-36.19); $I^2$=N/A | 1; 0.30 mg/dL; (−21.44-22.04); $I^2$=N/A |

*Statistical significant; $P \leq 0.05$. N/A: Not available, MO: *Moringa oleifera*, RCT: Randomized control trial, ; FBS: Fasting blood sugar; LDL: Low-density lipoprotein, HDL: High-density lipoprotein; TC: Total cholesterol; TG: Triglyceride, PPG: Postprandial glucose, WMD: Weighted mean difference, CI: Confidence interval, $HbA_{1C}$: Hemoglobin A1c

**Table 4:** Sensitivity analysis outcomes compare main analysis

| Outcomes | *n*; WMD (95% CI); $I^2$ | | References |
|---|---|---|---|
| | Main analysis | Sensitivity analysis | |
| FBS | 257; −14.81 mg/dL (−27.99-−1.63); $I^2$=97.8%* | 257; −3.95 mg/dL (−4.98-−2.91); $I^2$=97.8%* | [13-16,25-27] |
| $HbA_{1C}$ | 152; 0.7% (−1.42-2.82); $I^2$=99% | 152; 1.72%; (1.59-1.86); $I^2$=99%* | [13,15,26] |
| PPG | 92; −64.73 mg/dL (−102.84-−26.59); $I^2$=93%* | 92; −81.11 mg/dL (−85.45-−76.76); $I^2$=93%* | [13,27] |
| LDL | 135; −11.20 mg/dL (−34.12-11.72); $I^2$=88% | 135; 0.18 mg/dL (−1.57-1.93); $I^2$=88% | [13,15,25] |
| HDL | 135; 2.15 mg/dL (1.92-2.39); $I^2$=0.0% | 135; 2.15 mg/dL (1.92-2.39); $I^2$=0.0% | [13,15,25] |
| TC | 135; −4.73 mg/dL (−24.96-15.49); $I^2$=80% | 135; −1.52 mg/dL (−3.26-0.22); $I^2$=80% | [13,15,25] |
| TG | 135; −3.29 mg/dL (−9.95-3.36); $I^2$=29% | 135; −1.52 mg/dL (−3.54-0.50); $I^2$=29% | [13,15,25] |

FBS: Fasting blood sugar, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, TC: Total cholesterol, TG: triglyceride, PPG: Postprandial glucose, WMD: Weighted mean difference, CI: Confidence interval, $HbA_{1C}$: Hemoglobin A1c

## Adverse effects

Only two studies[15,27] descriptively mentioned the adverse events associated with MO use. The frequent urination, headache, cough, and urine color change were the most common adverse events reported among recruited studies. Moreover, only Sandoval study[15] monitored some laboratory tests' vital results, including complete blood count, liver function test, and renal function test. All test results were found normal.

## Subgroup analysis

Most of the results of the subgroup analysis did not differ from those of the main analysis. Significant WMDs in FBS, LDL, HDL, and TC were detected in the subgroups of studies categorized according to the MO dosage form (tablets or capsules vs. powders), treatment period (≤30 days vs. >30 days), and study design (RCT vs. non-RCT as inclusion criteria [Table 3].

## Sensitivity analysis

Sensitivity analysis was performed using a fixed-effects model. Most results illustrated no differences from the findings obtained using the random-effects model. Only the results for the $HbA_{1C}$ outcome changed from non-significant to significant when analyzed using the fixed-effects model [Table 4].

## Publication bias

We also generated funnel plots for all of the outcomes analyzed, using visual inspection of the plots to detect publication bias. The funnel plot test was nearly symmetrical, indicating no potential publication bias in these studies [Figure 4].

# DISCUSSION

Our study performed a systematic review and meta-analysis of four RCTs and three non-RCTs to obtain a clinical summary of MO's effect on blood glucose level and lipid profile. This study pooled the effect of MO treatment on blood glucose and lipid profile by conducting a meta-analysis. Existing evidence suggests that compared to the comparator treatment, MO had greater benefits in terms of FBS and PPG between 28 and 90 days of treatment. The results were consistent across included trials with a low risk of bias and high quality of data (Jadad score ≥3).

MO treatment decreased FBS and PPG significantly. Previous animal studies have shown that MO treatment decreased hyperglycemia. Bamagous *et al.*[7] found that treatment with MO leaf extract (200 mg/kg) in diabetic rats was induced by streptozotocin significantly decreased blood glucose and $HbA_{1C}$. In a recent study, MO leaf powder administration to diabetic rats induced by alloxan significantly decreased blood sugar concentration.[28] The possible mechanisms of action underlying the hypoglycemic effect of MO were: (1) increased insulin secretion,[29] (2) improved insulin sensitivity via stimulation of the insulin-dependent Akt pathway and upregulation of the expression of the glucose transporter GLUT4 in the muscles,[30] (3) improved glucagon synthase activity and glucose uptake in the muscles,[10] and (4) inhibition of α-amylase and α-glucosidase in the intestine.[11,12] However, there was no significant decrease in the $HbA_{1C}$ outcome, which may be attributable to the small number of participants in individual studies and short study duration.

In a study performed in rabbit models of high-cholesterol diet-induced dyslipidemia, Chumark *et al.*[31] showed that MO could decrease LDL by approximately 50%, TG by 75%, and carotid plaque formation by 97%. Results of a study performed in rat models fed a high-fat diet showed that the methanolic extract of MO leaves decrease LDL, TC, and TG significantly.[9] The possible underlying mechanisms included reduction of total intracellular cholesterol and inhibition of HMG CoA reductase activity and enhanced LDL receptor binding activity.[5] However, our meta-analysis revealed no significant results for the effects of MO on LDL, TC, and TG. These results might be attributable to the included studies being performed in normocholesterolemic and borderline hyperlipidemia participants and short study duration (<3 months). Subgroup analysis showed that MO powder significantly decreased FBS and TC level. Moreover, MO treatment for >30 days could significantly decrease FBS. This suggests that to achieve a reduction in blood sugar levels, MO treatment should be performed for >1 month.

We believed that, this is the first meta-analysis summarizing the clinical benefits of MO treatment with regard to glycemic and lipid profile. Patients in the MO-treated group demonstrated the non-different rate of adverse effects as that in the placebo group. There were no any serious adverse events among both groups. Therefore, MO seems to be an acceptable and tolerable herbal medicine.

Nevertheless, there are some limitations of this study. First, the numbers of trials and participants included were small. Second, variations in product formulations, standardized methods and dose regimens were observed among the studies included. Only one study used a MO product was standardized and reported the quantity of active compound in the MO preparation. Third, the MO intervention duration in all the included studies was short.

# CONCLUSION

The results of our meta-analysis support the use of MO preparations as an alternative or additional medicines for lowering blood glucose levels and increase HDL level. However, due to the limitations described above, these results should be updated when more RCTs are available and well-designed RCTs with standardized MO for long-term intervention should be performed to confirm the efficacy of MO in terms of lowering glycemic profile and improving the lipid parameters.

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## Conflicts of interest

There are no conflicts of interest.

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