International Journal of Nutrition Sciences

Journal Home Page: ijns.sums.ac.ir

Review Article

Dose-Response Effects of Coconut Oil on Glycemic Control: An Updated Meta-Analysis of Randomized Trials

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ARTICLE INFO

Keywords:
Coconut oil
Fasting glucose
Insulin
Glycemic control

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Received: February 1, 2026
Revised: April 25, 2026
Accepted: May 1, 2026

ABSTRACT

Background: Previous meta-analyses have not examined intake thresholds for coconut oil's effect on glycemic markers. This study addressed that gap by investigating the non-linear relationship between coconut oil consumption and fasting glucose and insulin levels.

Methods: PubMed, Scopus, Embase, CENTRAL, ISI Web of Science, and Google Scholar were systematically searched for randomized controlled trials (RCTs) published through August 2025. Eligible studies examined coconut oil's effect on glycemic markers in adults over at least two weeks. Pooled effect sizes were calculated using a random-effects model, followed by subgroup and sensitivity analyses. Distinct from prior reviews, a restricted cubic spline model was applied to evaluate dose-response patterns.

Results: Twelve RCTs with 518 participants were included. Coconut oil intake showed a modest but significant increase in fasting plasma glucose [mean difference (MD): 3.18 mg/dL; 95%CI: 0.22-6.13; I^2=90.8%; n=11]. No significant effect was observed on serum insulin (MD: 0.17 µIU/mL; 95%CI: −2.28-2.63; I^2=89.1%; n=5). The dose-response analysis revealed a non-linear trend, indicating that moderate consumption of 30-50 grams per day may attenuate glucose elevations compared to lower doses, a novel finding highlighting the need for dosage-specific guidelines (*p* for non-linearity=0.533).

Conclusion: Coconut oil appears to exert a modest effect on fasting glucose, with minimal impact on insulin. The observed non-linear dose-response suggests that moderate intake may offer a more favorable glycemic profile. Further high-quality trials are needed to clarify optimal dosing and clinical relevance.

Please cite this article as: Malmir H, Gaeini Z, Mirmiran P. Dose-Response Effects of Coconut Oil on Glycemic Control: An Updated Meta-Analysis of Randomized Trials. Int J Nutr Sci. 2026;11(2): doi:

Introduction

Diabetes mellitus is a chronic progressive metabolic disorder characterized by persistent hyperglycemia due to either deficits in insulin secretion, insulin action, or both. It now ranks among the greatest public health challenges in the 21st century. Moreover, the burden of diabetes on a global scale is still on the rise, with estimates of nearly 600 million people living with diabetes in 2035, which will impose a significant medical,

social, and economic burden worldwide (1, 2). Pharmacological therapies, including insulin analogs and oral hypoglycemic agents, are the mainstay of glycemic management for diabetes. Unfortunately, neither alone may significantly affect the progression of the disease, and increasingly, both treatment options are ineffective in reducing the complications of diabetes, especially where patient adherence to treatment is poor and/or access to resources is limited. Consequently, the focus is directed toward finding complementary therapeutic options to enhance glycemic control, including exploration of modifiable lifestyle factors such as nutrition, exercise, and weight loss (3-6).

From a nutrition perspective, in particular, coconut oil has become of great interest over the last several decades as an included food component that is related to the metabolic health. As a food and traditional medicine, coconut oil, derived from the fruit of the coconut palm (*Cocos nucifera*), has been used for centuries in tropical and subtropical regions. During this time, the unique fatty acid composition of coconut oil, especially the medium-chain triglycerides (MCTs) of phosphate ether lauric acid, has generated hypotheses about the health benefits of coconut oil. Proponents of the health claims of coconut oil assert that the MCTs in coconut oil are metabolized and utilized faster than long-chain triglycerides, which can lead to increased energy expenditure, increased satiety, and increased sensitivity to insulin (7, 8). While often advocated for benefits like weight loss and cardiovascular health, recent interest has focused on its role in managing diabetes (9, 10).

While systematic reviews have assessed coconut oil's impact on glycemic control, none have explored non-linear dose effects. This limits practical recommendations, as metabolic responses may depend on intake quantity (7-15). This limits practical recommendations, as metabolic responses may depend on intake quantity. A dose-response meta-analysis could provide critical insight into the dosage needed to achieve meaningful effects on serum glucose and insulin. Therefore, our study addressed this gap by conducting the first dose-response meta-analysis, evaluating whether coconut oil's effects on glucose and insulin are dose-dependent and a critical question for developing evidence-based dietary guidance. Furthermore, clarifying this relationship has significant public health implications, as it could inform more precise nutritional recommendations for populations at risk of dysglycemia, potentially offering a complementary dietary strategy to mitigate the growing global burden of diabetes.

Materials and Methods

This study is a meta-analysis and systematic review of the literature, and eligibility reporting followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (16). To promote transparency and methodological rigor, the systematic review protocol was registered before data extraction at the International Prospective Register of Systematic Reviews (PROSPERO; registration ID: CRD42024548562)

Search Strategy

To identify eligible trials, an electronic literature search was performed using five electronic databases including PubMed, Scopus, Embase, the Cochrane Central Register of Controlled Trials (CENTRAL), ISI Web of Science, and Google Scholar. The search was performed on all published records from inception until April 2024, with no restrictions on geography or language. Search terms included terms in combination for "coconut oil", "virgin coconut oil", glycemic markers (e.g., "fasting blood sugar", "fasting plasma glucose", "HbA1c", "glycemic control", "insulin") and terms related to study design (e.g., "randomized controlled trial", "intervention"). Sensitivity was increased in PubMed by using [tiab] and [MeSH] tags. The bibliographies of relevant reviews and articles were manually searched to identify additional eligible trials.

Inclusion Criteria

Two authors (HM and ZG) independently assessed study eligibility based on initial inclusion and exclusion criteria. Studies were included if they met the criteria of (i) Design: Randomized controlled studies, non-randomized controlled studies, and crossover studies (minimum two weeks of intervention); (ii) Population: Adults aged 18 years and older; (iii) Intervention: Administration of coconut oil (e.g. virgin or extra virgin) as the primary dietary exposure, measured in grams or milliliters per day; (iv) Comparator: Control group receiving no intervention, a placebo, or another type of oil (e.g. soybean, corn, or olive); (v) Outcomes: Quantitative measures of fasting glucose or insulin in serum or plasma, with measures reported as change-from-baseline, end-of-intervention values, or provided sufficient data to enable estimates of mean differences; and (vi) Data Availability: Studies had to have supplied enough data to calculate effect sizes, standard deviations or allow estimation through established waiver methods.

Exclusion Criteria

Studies were excluded if they were (a) In vitro,

or animal studies; (b) Observational, cross-sectional, or ecological studies; (c) Reviews, meta-analyses, editorials, or conference abstracts; and (d) Unable to isolate effects of coconut oil.

Study Selection and Data Extraction

There were two reviewers who independently screened study titles and abstracts, subsequently engaging in full-text reviews for studies deemed potentially eligible for inclusion. Disagreements were resolved through discussion or by third reviewer adjudication. A pre-designed data extraction form was utilized to extract study characteristics, including: author, year of publication, country, design, sample size, participant characteristics, intervention (dose, duration), control group, and outcomes (glucose and insulin). When changes from baseline data were unavailable, pre- and post-means and standard deviations were extracted; and mean differences were estimated according to Cochrane Handbook guidance. Standard deviations were calculated from standard errors where appropriate.

Quality Assessment of Studies

Using the Cochrane RoB 2 tool, two reviewers assessed methodological quality across the following six domains including randomization process, allocation concealment, blinding of participants and personnel, blinding of outcome assessment, incomplete outcome data, and selective reporting. No disagreements were resolved by bringing together reviewers to come to consensus over their bias assessments (17).

Data Synthesis and Statistical Analysis

Data were summarized using effect size estimates in the form of mean differences (MD) with 95% confidence intervals (95%CI). In light of anticipated heterogeneity a random effects meta-analysis model (DerSimonian and Laird) was used. Heterogeneity was assessed by using the I^2 statistic for the level of heterogeneity and the Cochran's Q statistic. Subgroup analyses were performed based on sex, intervention duration (less than or equal to four weeks vs. greater than four weeks), type of coconut oil, baseline health status, and quality of study (good, fair, or poor). The sensitivity analysis was completed by systematically removing studies. A non-linear dose-response meta-analysis using restricted cubic splines was conducted to examine the relationship between the dose of coconut oil and glucose. Publication bias was assessed with funnel plots (visual inspection) and Egger's test ($p<0.05$), with all analyses performed in Stata (version 17.0; StataCorp, College Station, TX, USA).

Results

Systematic Search and Study Selection Process

Totally, 2542 records were generated from database searching. After excluding duplicates (n=412), 2130 articles were screened for unique articles by title and abstract. Of the 2130 titles and abstracts screened, 2105 studies were excluded after title and abstract review for not being relevant, lacking interventional design, or having no outcomes for consideration. Out of 25 articles, 13 studies were excluded because of short-term effects of coconut consumption and did not report serum glucose or insulin levels in all phases (18), or the trials reporting two times (19, 20). Twelve full-text articles met the inclusion criteria and were included in the systematic review. Of the 12 included trials, 11 trials contributed to the glucose outcome meta-analysis, and five studies contributed to the insulin outcome analysis. The study selection and inclusion process were represented in the PRISMA flowchart (Figure 1).

Findings of Systematic Reviews

The 12 trials included in this study spanned between 1992 and 2023. The studies were located in Asia (20-23), Brazil (24-26), the UK (27, 28), and the USA (29, 30). Sample sizes for the studies ranged from 9 to 114 participants; and interventions lasted from 2 to 12 weeks. Study design included randomized parallel (20, 22, 24, 26, 31), nonrandomized trials (25, 27, 31), crossover (21, 23, 30), and multiple-arm ones (28). Six studies had both sexes included (20, 22, 25, 29, 30, 31), five studies had males only (21, 23, 26, 27, 29) and one study had females (24). Participants were either healthy or had cardiometabolic diseases such as type 2 diabetes (22), metabolic syndrome (20), cardiovascular disease (25), and obesity (24, 26). All intervention groups received coconut oil, extra virgin coconut oil (20, 23, 26), or fresh coconut (31); while control groups received nothing or oil other than coconut (e.g., palm oil, soybean oil, olive oil). Outcomes included serum glucose, insulin, hemoglobin A1c (HbA1c), Homeostatic Model Assessment of insulin sensitivity (HOMA-S%) and beta-cell function (HOMA-B%).

Detailed results of the assessment of risk of bias were presented in Table 1. Full risk-of-bias assessments were provided in Table 1 too. All included studies had a low risk of bias for missing outcome data, outcome measurement, and selective reporting. Five of the 12 studies had a low risk of bias for randomization, and one of the 12 had a low risk for deviation from the intended intervention. Of the studies, overall quality was assessed as good (1), fair (12), and poor (7).

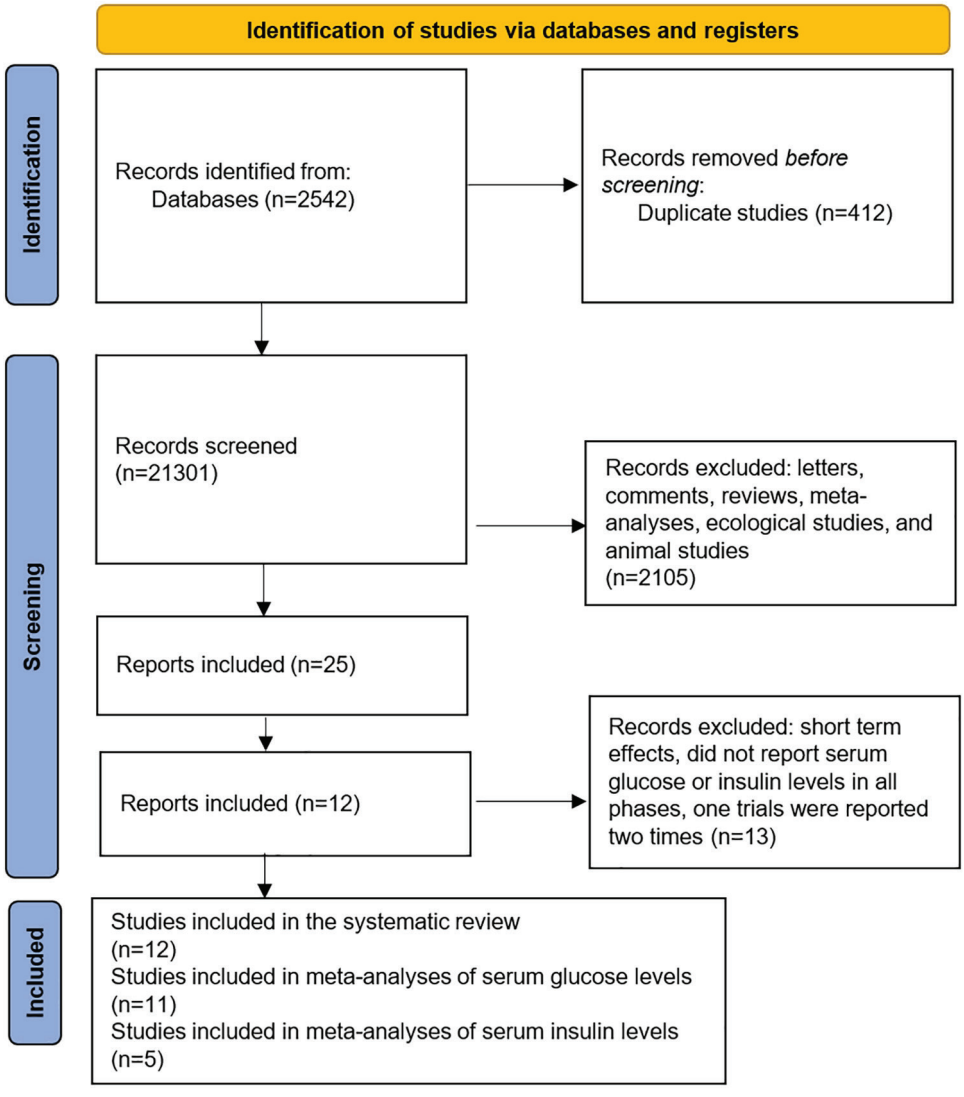

Figure 1: Flow chart for study search, selection, and screening.

Glucose Serum Level

Combining 11 effect sizes from 11 studies, we found that coconut oil consumption significantly increased glucose serum level [mean difference (MD): 3.18 (0.22, 6.13), I-square=90.8%, $p<0.0001$] (Figure 2). Subgroup analyses were performed to find the source of heterogeneity. Considering subgroup analyses by gender, type of coconut oil, duration of intervention, baseline health status and quality; the results were not significant and heterogeneity remained high (Table 2). Sensitivity analysis revealed that the overall effect was not affected by any individual (Figure 1a). Funnel plots and Egger's regression tests indicated no evidence of substantial publication bias for serum glucose level ($p=0.398$) (Figure 1b). A nonlinear dose-response meta-analysis was performed using a restricted cubic spline. Although the findings showed that consumption of amounts less than 30 grams per day of coconut oil was associated with an increased glucose serum level, and taking 30 to 50 grams of coconut oil led to a decrease in glucose serum level. It should be noted that the above findings were not statistically significant (p nonlinearity=0.533) (Figure 3).

Serum Insulin Level

Combining four effect sizes from four studies, we did not observe a statistically significant effect on serum insulin level, with substantial heterogeneity between the eligible studies [MD: 0.17 (-2.25, 2.63), $I^2=89.1\%$, $p<0.0001$] (Figure 4). Subgroup analyses by gender, type of coconut oil, duration of intervention, and baseline health status did not change the findings (Table 3). Subgroup analyses by considering the quality of studies indicated that serum insulin level might be reduced significantly when we considered fair quality studies [MD:-1.71 (95%CI: -2.65, -0.77)]. Sensitivity analysis revealed that the overall effect not to be affected by any individual study (Figure 2a). Funnel plots and Egger's regression tests indicated no evidence of substantial publication bias for serum glucose level ($p=0.901$) (Figure 2b). We could not perform a dose-response analysis because of the insufficient number of articles.

Table 1: Characteristics of clinical trial studies included in the systematic review.

Number	Reference	Study design	Country	Age range	Gender	Sample size	Duration (w)	Participants	Intervention groups		Outcome variable	Outcome assessment	Comparison	Findings
									Treatment	Control				
1	(29)	Non randomized trial	USA	22-43	M	13	13 weeks (3 weeks int/2 weeks washout)	Healthy	Palm oil, coconut oil, or hydrogenated soybean oil (muffins and cookies)	-	Insulin (microunits/mL) Glucose levels (mg/dL)	Radioimmunoassay Enzymatic methods (glucose oxidase method)	Insulin (Post- vs. pre-) Glucose (Post- vs. pre-)	14±2 vs. 11±3 (p=0.07) 78±2 vs. 80±5 (p=0.77)
2	(27)	Non randomized trial	UK	20-59	M	12	4 weeks int (4-8 weeks washout)	Healthy	Sea buckthorn berry oil, coconut oil 5 g per day [(500-mg capsules (10 capsules per day)]	-	Glucose levels (mmol/L)	Standard spectrophotometric methods	Glucose (Post- vs. pre-)	5.36 (0.13) vs. 5.11 (0.17) (p<0.01)
3	(22)	RCT, parallel	India	45.16± 2.20	Both	20 int 20 ctrl	2 month	Newly diagnosed type 2 diabetes	Sesame oil, 35 ml of the oil per day	Coconut oil	Glucose levels (mg/dL)	Standard technique	Glucose (Post- vs. pre-)	165±5 vs. 162±6
4	(24)	RCT, parallel	Brazil	20-40	F	20 int 20 ctrl	12 weeks	Abdominal obesity as defined by waist circumference [>88 cm]	30 mL of soy bean oil	30 mL of coconut oil	Glucose (mg/dL) Insulin (microunits/mL)	Standard laboratory kits	Glucose (Post- vs. pre-) Insulin (lU/mL) HOMA-S% HOMA b%	82.8±5.4 vs. 83.5±7.8 (p=0.81) 9.8±4.1 vs. 9.0±4.5 (p=0.09) 2.0±0.9 vs. 1.8±0.09 (p=0.11) 39.4±18.0 vs. 36.1±20.1 (p=0.20)

5	(25)	Non randomized trial	Brazil	45-85	Both	114 92 int 22 crtl	3 month	Coronary artery disease patients	Diet+coconut oil sachets containing 13 mL/d	Diet	Glucose level (mg/dL)	Spectrophotometric method using the glucose oxidase/ peroxidase.	G Glucose (Post- vs. pre-)	1.4±23.6 vs. 118.0±34.1 (p=0.57)
											HgA1c (mg/dL)	Turbidimetric immunoassay	HgA1c	0.1±0.6 vs. 6.2±1.1 (p=0.05)
6	(30)	RCT, crossover	USA	18-79	Both	24	4 weeks	Healthy	(Muffins or rolls) containing corn oil	(Muffins or rolls) containing coconut oil	Glucose (mg/dL)	Enzymatic assay	Glucose (Post vs. pre)	6.0 (−3.0, 13.2) vs. 92.5 (76.5, 136)
											Insulin	Electrochemiluminescence immunoassay		
													HOMA-S%	0.7 (−25.0, 28.1) vs. 109 (64.8, 213)
													HOMA b%	0.7 (−19.0, 11.0) vs. 90.0 (70.0, 117)
7	(31)	RCT, parallel	India	23.8± 4.8	Both	80	3 months	Healthy	100 g fresh coconut	An equivalent 45 g of groundnuts and 22 g of groundnut oil per day	Glucose (mmol/L)	Fully automated biochemistry analyzer	(Post- vs. pre-)	4.23±0.37 vs. 4.63±0.32 (p<0.001)
											Insulin (pmol/L)	Enzyme immunoassay ELISA kits	(Post- vs. pre-)	41.57±24.07 vs. 72.66±125.2
8	(28)	RCT, multiple arm	UK	50-75	Both	28 coconut oil 33 butter 30 olive oil	4 weeks	Healthy	50 gram/d coconut oil or olive oil	Butter	Glucose (mmol/L)	Hexokinase-glucose-6-phosphate dehydrogenase method	Change	−0.05 (0.49)

9	(21)	RCT, crossover	India	28-50	M	9	8 weeks (6 weeks wash out)	Healthy	Addition 35 g of coconut oil (CO) or peanut oil every day		Glucose (mmol/L)	The manufacturers' instructions/ protocols	Post- vs. pre-	5.6±0.49 vs. 5.2±0.34
											Insulin (mU/mL)	Radioimmuno assay (RIA) method	Post- vs. pre-	10.4±2.36 vs. 16.6±3.27
10	(20)	RCT, parallel	Iran	20-50	Both	22 int 22 ctrl	4 weeks	Metabolic syndrome	Virgin coconut oil 30mg/d as an alternative to the same amount of oil in the habitual diet	Usual diet	Glucose (mg/dL)	Commercial diagnostic kits	Post- vs. pre-	102.5 (95 to 113) vs. 105 (97 to 112) p=0.605
11	(26)	RCT, parallel	Brazil	20-59	M	29 15 coconut 14 soy	6 weeks	Obesity	1 tablespoon (12 mL) of extra virgin coconut oil	1 tablespoon (12 mL) soybean oil	Glucose (mmol/L)	Enzymatic method	Post- vs. pre-	78.73±10.97 vs. 84.40±10.46 (p=0.02)
											Insulin (mU/mL)	ELISA	Post- vs. pre-	5.13±3.79 vs. 8.28±7.13 (p=0.06)
12	(23)	Non randomized, crossover	India	28-50	Male	22	8 weeks	Healthy	Diet with virgin coconut oil (~35g/d)	Diet with peanut oil (~35g/d)	Glucose (mg/dL)	Manufacturers' instructions	Post- vs. pre- 95+2.9 vs. 91+2.1	97±3.1 vs. 89±2.4
											Insulin (mU/mL)	RIA	Post- vs. pre- 8.0+0.90 vs. 7.7 +2.1	18.2±2.67 vs. 19.5±3.28

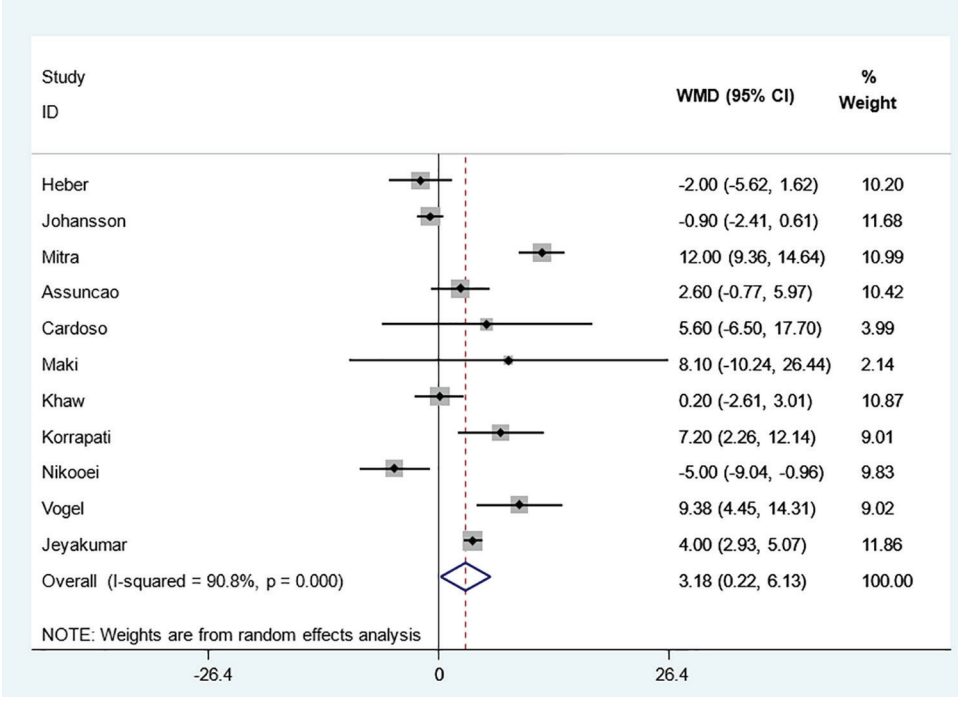

Figure 2: Combined effect sizes of coconut oil consumption on serum glucose levels.

Table 2: Subgroup analyses of coconut oil (mL) and serum glucose level (mg/dL).

Characteristics	n	Mean difference (95%CI)	I^2 (%)	*P* heterogeneity
All studies	11	3.18 (0.22, 6.13)	90.8	<0.0001
Sex				
Women	1	2.60 (-0.77,5.97)	0	<0.0001
Men	4	3.02 (-1.93,7.97)	87.7	<0.0001
Men and women	6	3.50 (-1.34, 8.34)	91.9	<0.0001
Study duration				
≤4 weeks	6	-0.24 (-2.88, 2.41)	68.7	0.007
>4 weeks	5	6.77 (2.56, 10.98)	88.7	<0.0001
Intervention				
Coconut oil	8	3.49 (-0.76, 7.75)	91.3	<0.0001
Virgin coconut oil	3	2.72 (-3.75, 9.19)	91.4	<0.0001
Health status of participants				
Healthy	6	1.64 (-1.31,4.58)	87.0	<0.0001
Unhealthy	5	4.88 (-2.03,11.80)	92.6	<0.0001
Quality of studies				
Poor	1	2.60 (-0.77, 5.97)	0	<0.0001
Fair	7	4.72 (0.638.81)	91.0	<0.0001
Good	3	-0.98 (-2.36, 0.41)	0.0	0.484

Discussion

This systematic review and dose–response meta-analysis combined results from 12 intervention studies examining the effects of consumption of coconut oil on glycemic control among adults, specifically for fasting glucose and insulin. The results of the pooled analysis indicated a small but statistically significant increase in fasting glucose level, whereas there were no consistent effects for fasting insulin level. These analyses suggest that coconut oil may have a small effect on glucose metabolism but the effect on insulin sensitivity seems equivocal based on fasting biomarkers. Subgroup analyses revealed that the reduction in fasting insulin level was only measured in studies with fair methodological quality, indicating that methodological nuances can influence results and may help explain the null effects that previous reviews concluded.

The dose-response analysis provided a new viewpoint that indicated a potentially non-linear relationship between coconut oil intake and fasting glucose level.

Figure 3: Nonlinear dose-response effects of coconut oil consumption on serum glucose levels.

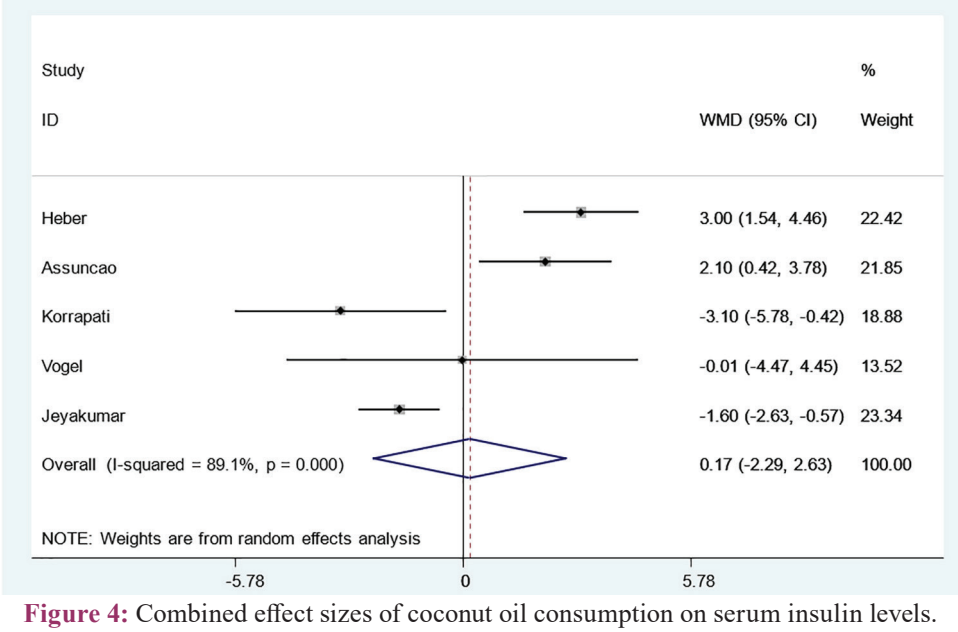

Figure 4: Combined effect sizes of coconut oil consumption on serum insulin levels.

More specifically, low intake (0-30 g/day) suggested an increase in glucose, while moderate intake (30-50 g/day) suggested neutral or perhaps beneficial effects. However, the current association was not statistically significant (*p* nonlinearity=0.533) and only had a small number of data points. Therefore, these findings should be considered hypothesis-generating and should not be used for clinical recommendations. The existence of a "therapeutic window" in terms of coconut oil is an open question. The clinical importance of the observed increase in fasting glucose level (mean difference ~3.18 mg/dL) is tenuous at best, the change in magnitude is small and probably falls within the range of analytical error of many assays, and it is unlikely that this would lead to a meaningful increase in individual diabetes risk (7, 9).

The proposed mechanisms are also speculative. MCTs have a rapid hepatic metabolism and may stimulate hepatic gluconeogenesis, or temporary hepatic insulin resistance, and thus lead to small elevations in circulating glucose level (32). The lack of a corresponding increase in insulin level may express either a depressed pancreatic beta-cell response or a small enough magnitude that the compensatory mechanisms were not called upon. The drop in insulin value from higher quality studies is intriguing; however, this requires confirmation from high quality, rigorously designed randomized controlled studies (33-35).

Table 3: Subgroup analyses of coconut oil (mL) and serum insulin level (mU/mL).

Characteristics	n	Mean difference (95%CI)	I^2 (%)	*P* heterogeneity
All studies	5	0.17 (-2.28, 2.63)	89.1	<0.0001
Sex				
Women	1	2.10 (0.42, .3.78)	-	
Men	3	0.07 (-4.26,4.40)	87.4	<0.0001
Both	1	-1.60 (-2.29, -0.57)	-	
Study duration				
≤4 weeks	2	0.06 (-5.92,6.05)	93.5	<0.0001
>4 weeks	3	0.13 (-2.77, 3.04)	85.3	0.001
Intervention				
Coconut oil	3	0.88 (-2.13, 3.88)	87.2	<0.0001
Virgin coconut oil	2	-1.52 (-2.52, -0.52)	0	0.496
Health status of participants				
Healthy	3	-0.47 (-3.90, 3.05)	93.3	<0.0001
Unhealthy	2	1.84 (0.27, 3.41)	0	0.385
Quality of studies				
Poor	1	2.10 (0.42, 3.78)	-	
Fair	3	-1.71 (-2.65, -0.77)	0.0	0.441
Good	1	3.00 (1.54, 4.46)	-	

This review and meta-analysis is the first to include a dose–response format for coconut oil and glycemic markers. Rigorous methodology with PRISMA adherence, risk-of-bias, sensitivity analyses, and subgroup analysis increases internal validity. Including both refined and virgin coconut oil trials allows for adjustment of composition and any effects it might have. There are also some limitations. High heterogeneity (I^2>90%) that was not explained by subgroup analysis limits our confidence in the pooled results. The relatively short duration (2-12 weeks; median 6 weeks) of trials examined limit our ability to assess long-term metabolic adaptations. The small sample sizes, and number of trials used for insulin outcomes reduced the statistical power. Low-to-moderate methodological quality in all included studies (limitations in blinding, randomization and selective reporting) raised the risk of bias. The lack of mechanistic data, such as insulin sensitivity indices (HOMA-IR, clamp studies) limited our ability to interpret metabolic pathways too.

This review also highlights the need for dietary recommendations to be matched with the metabolic characteristics of patients even at cellular level (36, 37). The responses to coconut oil are likely to differ in those who are metabolically healthy, versus those with metabolic disorders, supporting the need for stratified research. Moreover, the quality of existing evidence based on our methodological appraisal, is variable, with most studies rated as fair or poor. Common methodological zone were inadequate blinding, small sample sizes, non-randomized designs, and short follow-up. Altogether, these weaknesses limit confidence in the strength of the observed effects, and applicability of results (13-15).

A major limitation of this evidence base is the considerable heterogeneity amongst studies (I^2>90%) despite considerable subgroup analyses, which were not sufficient to explain the sources of heterogeneity. Sources of heterogeneity are likely attributed to variety in metabolic health profile of participants (healthy, type 2 diabetes, metabolic syndrome, and cardiovascular diseases), variation in the type of coconut oil (refined vs. virgin), short duration of interventions (mostly 2-12 weeks), and variability in quality of study. Virgin coconut oil has polyphenols, phytosterols and tocopherols, which may have antioxidant and anti-inflammatory properties, therefore potentially ameliorating the effects of saturated fats, whereas refined coconut oil is stripped of its polyphenols. Additionally, people with insulin resistance or poor beta-cell function may have a unique metabolic response compared with those with insulin sensitivity, which is difficult to capture with pooled estimates. From a methodological perspective, variability in these elements has limited the interpretability of summary effect estimates (10, 25).

Conclusion

Our evidences suggest that coconut oil intake has a small, statistically significant effect on fasting glucose level, but it does not have a consistent effect on fasting insulin level. The findings on these outcomes have considerable heterogeneity and the trials were short in duration and low

on methodological quality. A non-linear dose–response relationship was presented; but this was not statistically significant and requires validation in a greater number of trials. Overall, there is not enough evidence to support a case for coconut oil use in glycemic control, and this is not justified in claims for the effects of coconut oil. Overall, there needs to be a future well-designed randomized controlled trial with long-term follow-up, standardization of coconut oil used between studies, and sufficient stratification of participants regarding their metabolic health status to clarify coconut oil's effects on glucose and insulin homeostasis.

Acknowledgement

We appreciate financial support of Shahid Beheshti University of Medical Sciences, Tehran, Iran to perform the project.

Funding

This work was supported by Shahid Beheshti University of Medical Sciences, Tehran, Iran (43003699).

Authors' Contribution

HM conceptualized and designed the study; HM and ZG analyzed and interpreted the data; HM, ZG, and PM drafted the initial manuscript; PM supervised the project; and all authors read and approved the final version of the manuscript

Conflict of Interest

The authors declare no competing interests.

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