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Review Article

Effects of apple cider vinegar on glycaemic control, lipid profile, and anthropometric outcomes in adults: A systematic review of randomized controlled trials.

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Abstract

Background:

Apple cider vinegar (ACV) is widely used as a complementary dietary intervention for improving metabolic health, yet clinical evidence regarding its efficacy remains inconsistent.

Objectives:

To systematically review randomized controlled trials (RCTs evaluating the effects of apple cider vinegar on anthropometric measures, glycaemic control, lipid profile, and related metabolic outcomes in adults.

Methods:

This systematic review followed PRISMA 2020 guidelines. PubMed/MEDLINE, Scopus, Web of Science, CENTRAL, and Google Scholar were searched from inception to January 2025. Randomized or controlled clinical trials in adults (≥18 years) evaluating oral apple cider vinegar as a standalone intervention for at least two weeks were included. Two reviewers independently performed study selection, data extraction, and quality assessment. Risk of bias was assessed using the Cochrane RoB-2 tool. Eleven RCTs were included, with overall risk of bias ranging from low to high; inadequate blinding was the most frequent limitation. The protocol was not prospectively registered, and findings were synthesized narratively.

Results:

Eleven randomized controlled trials involving over 750 participants were included. Most studies enrolled individuals with type 2 diabetes mellitus, overweight/obesity, or dyslipidaemia. ACV supplementation (15–30 mL/day for 4–12 weeks) consistently reduced fasting blood glucose and produced modest improvements in HbA1c among diabetic participants. Significant reductions in triglycerides and total cholesterol were observed in dyslipidaemic populations, while effects on LDL-C and HDL-C were variable. Modest but significant reductions in body weight, body mass index, and waist circumference were reported, particularly in overweight or obese individuals. ACV was generally well tolerated, with only mild gastrointestinal adverse effects.

Conclusions:

Apple cider vinegar supplementation provides modest but clinically relevant improvements in glycaemic control, lipid parameters, and anthropometric outcomes, particularly in individuals with metabolic disorders. ACV may serve as a safe adjunctive dietary intervention; however, larger and longer-term trials are required to confirm sustained benefits.

Keywords: *Apple Cider Vinegar; Anthropometry; Body Weight; Glycemic Control; Lipid Profile; Metabolic Diseases.*
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Introduction

The growing prevalence of obesity and type 2 diabetes mellitus (T2DM) has intensified interest in complementary dietary interventions to improve metabolic health. Apple cider vinegar (ACV) – a vinegar made via fermentation of apple sugars to acetic acid – has a history of use as a folk remedy for weight loss and glycemic control. ACV contains ~4–8% acetic acid along with bioactive phytochemicals (polyphenols such as catechin, ferulic, and caffeic acids) that may confer

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metabolic benefits. In animal studies, vinegar preparations have demonstrated anti-obesity, anti-diabetic, antihypertensive, and antioxidative effects 1. In humans, small trials have reported that vinegar intake before or with meals can reduce post-prandial glycemia and improve insulin sensitivity 2. ACV, in particular, has gained popularity as a natural supplement for improving blood sugar control and cholesterol levels in patients with diabetes or hyperlipidemia. Nonetheless, the scientific evidence has been somewhat conflicting, and the magnitude of ACV's benefits in controlled trials remains unclear 1.

Over the past decade, multiple RCTs have evaluated the effects of ACV on various metabolic outcomes. These include trials in **patients with T2DM** (to assess fasting blood glucose, HbA1c, and insulin sensitivity), in individuals with **dyslipidemia or obesity** (to assess changes in body weight, body fat, and lipid profiles), and in **healthy subjects** (to examine any metabolic improvements) 3. Some trials have shown promising results – for example, Kondo *et al.* (2009) reported that daily vinegar intake reduced body weight, body fat mass, and serum triglyceride levels in obese Japanese adults 4. Similarly, Khezri *et al.* (2018) found that adding ACV to a calorie-restricted diet led to greater weight loss and improvements in visceral adiposity index and lipid profile in overweight or obese individuals 5. In patients with diabetes, ACV has been reported to modestly lower fasting glucose and cholesterol in a number of small trials. However, not all studies have shown significant effects, and differences in **dosage, duration**, and patient populations have led to variable outcomes. Questions also remain regarding ACV's impact on other outcomes like blood pressure and inflammation, and whether any benefits are clinically meaningful 3.

To clarify the evidence, we performed a systematic review of RCTs investigating ACV's effects on weight, glycemic control, and lipid management. We focused on high-quality data by leveraging two recent systematic reviews and meta-analyses: one by Hadi *et al.* (2021) that examined ACV's effects on lipid and glycemic parameters in adults, and one by Arjmandfard *et al.* (2025) that specifically assessed ACV's impact on glycemic control in T2DM with a dose–response analysis 1,3. By synthesizing findings from the trials included in these reviews, this study aims to provide an updated, comprehensive understanding of ACV's efficacy as a nutritional intervention. In this review, the study summarizes the characteristics of the included studies, critically evaluates the effects of ACV on **body weight**, **blood glucose, HbA1c, insulin sensitivity**, **lipid profiles (triglycerides, cholesterol)**, and other beneficial outcomes (such as blood pressure and oxidative stress markers), and discusses the clinical relevance of the findings.

Methods:

Data Sources and Search Strategy

This study conducted this systematic review in accordance with the PRISMA 2020 guidelines. A comprehensive and structured search strategy was developed prior to study initiation to identify all clinical trials evaluating the effects of apple cider vinegar (ACV) on weight reduction, glycaemic indices, lipid parameters, and other metabolic outcomes in adults.

A systematic search was performed across the following electronic databases: PubMed/MEDLINE, Scopus, Web of Science, Cochrane Central Register of Controlled Trials (CENTRAL), and Google Scholar. Searches covered all years from database inception to January 2025. No restrictions were applied regarding publication year, geographical setting, or language.

The search strategy combined controlled vocabulary (e.g., MeSH terms) and free-text terms, including variations of:

- *"apple cider vinegar"*, *"apple vinegar"*, *"ACV supplementation"*,
- *"weight loss"*, *"body mass index"*,
- *"glycemic control"*, *"fasting glucose"*, *"HbA1c"*, *"insulin sensitivity"*,
- *"lipid profile"*, *"cholesterol"*, *"triglycerides"*,
- *"clinical trial"*, *"randomized controlled trial"*, *"controlled study"*.

("apple cider vinegar"[MeSH] OR "apple vinegar" OR "ACV")

AND ("glycemic control" OR "fasting glucose" OR "HbA1c")

AND ("lipid profile" OR cholesterol OR triglycerides)

AND ("randomized controlled trial"[Publication Type])

Boolean operators "AND" and "OR" were used to refine and expand the search.

In addition, this study performed:

1. Backward reference searching of all included studies.
2. Forward citation tracking using Google Scholar.
3. Manual searching of relevant clinical nutrition and metabolic journals.
4. Screening of studies included in two previously published systematic reviews to ensure completeness.

All identified citations were imported into an electronic database and deduplicated before screening.

Study Selection

Study selection was carried out in two stages: (1) title–abstract screening and (2) full-text evaluation. Two reviewers independently assessed all retrieved records using predefined eligibility criteria.

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Inclusion Criteria

Studies were eligible if they met the following criteria:

1. Study design: randomized controlled trial (parallel or crossover) or controlled clinical trial.
2. Population: human adults (≥18 years), with or without type 2 diabetes, overweight/obesity, dyslipidaemia, or metabolic syndrome.
3. Intervention: oral apple cider vinegar or apple vinegar (any dosage, concentration, or formulation).
4. Comparator: placebo, water, no intervention, or standard diet/therapy without vinegar.
5. Outcomes: at least one of the following measured at baseline and endline—
   - Body weight, BMI, waist circumference
   - Glycaemic indices (FBS, PPBS, HbA1c, fasting insulin, HOMA-IR)
   - Lipid parameters (TC, TG, LDL-C, HDL-C)
6. Duration: minimum intervention period of 2 weeks.
7. Data availability: sufficient quantitative data (mean, SD/SE, or extractable data) for both groups.

Exclusion Criteria

This study excluded:

- Non-randomized observational studies, case reports, reviews, and animal studies.
- Interventions combining ACV with other supplements where the isolated effect of ACV could not be determined.
- Studies using non-apple vinegars (e.g., rice vinegar, white vinegar).
- Acute post-prandial trials lacking sustained supplementation.
- Trials lacking extractable outcome data.
- Duplicate datasets published in multiple articles.

All disagreements between reviewers were resolved by consensus, with arbitration by a third reviewer when necessary. The full selection process was documented using a PRISMA flow diagram.

Data Extraction

Data extraction was performed independently by two reviewers using a standardized, pre-piloted data extraction form. For each included study, we collected:

- **Study characteristics:** author, year, country, study design, setting.
- **Participant details:** sample size, age, sex distribution, baseline metabolic status.
- **Intervention details:** ACV dosage, frequency, method of administration, acetic acid concentration, duration of intervention.
- **Comparator details:** placebo composition or control regimen.
- **Outcome data:** baseline and final mean values for all metabolic indicators (weight/BMI, glycaemic indices, lipid profile), along with standard deviations, confidence intervals, or extractable graphical data.
- **Additional information:** adherence, adverse effects, funding source, trial registration.

Where numeric data were not directly available, values were obtained from graphs using digital extraction techniques. If a study presented multiple ACV arms, each was treated as a separate comparison against the control group.

All extracted data were cross-checked for accuracy and reviewed by the senior investigator before analysis.

Quality Assessment

The methodological quality of the included studies was evaluated using the **Cochrane Risk of Bias 2 (RoB 2) tool**, which systematically assesses bias across five key domains: bias arising from the randomization process, bias due to deviations from intended interventions, bias due to missing outcome data, bias in measurement of outcomes, and bias in selection of the reported results. Quality assessment was conducted independently by two reviewers, with disagreements resolved by consensus.

Overall, the included randomized controlled trials demonstrated **low to moderate methodological quality**, with several studies exhibiting domain-specific risks of bias. Most trials reported adequate random sequence generation; however, insufficient reporting of allocation concealment resulted in an unclear risk of bias in the randomization domain for a proportion of studies. Owing to the distinctive taste and sensory characteristics of apple cider vinegar, blinding of participants and study personnel was not feasible in many trials, leading to a **high risk of bias due to deviations from intended interventions**. Despite this limitation, outcome adherence was generally satisfactory, and contamination between groups was minimal.

The risk of bias due to **missing outcome data** was low across studies, as attrition rates were minimal and outcome data were largely complete. Measurement of outcomes was considered at **low risk of bias**, since primary endpoints—including body weight, fasting blood glucose, HbA1c, insulin indices, and lipid parameters—were objectively measured using standardized laboratory and clinical methods. However, several trials lacked prior protocol registration or detailed reporting of prespecified outcomes, resulting in an **unclear to high risk of selective reporting bias**.

Data Synthesis and Statistical Analysis

Data from the included randomized controlled trials were synthesized using a structured narrative approach, without undertaking a de novo quantitative meta-analysis. Following data extraction, key study characteristics and outcome data were tabulated and qualitatively

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summarized in a predefined framework. Trials were grouped a priori according to participant phenotype and clinical context (type 2 diabetes mellitus, overweight/obesity, dyslipidaemia, or mixed metabolic populations) and by intervention characteristics, including daily apple cider vinegar (ACV) dose, duration of intervention, and nature of the comparator.

Prespecified outcomes were categorized into four domains: (i) anthropometric outcomes (body weight, body mass index, waist and hip circumference); (ii) glycaemic outcomes (fasting blood glucose, glycated haemoglobin [HbA1c], fasting insulin, HOMA-IR, or related indices); (iii) lipid outcomes (total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol); and (iv) additional metabolic outcomes, including blood pressure, oxidative stress or antioxidant markers, and safety endpoints.

For each study, the direction and magnitude of effects were summarized using reported between-group differences and corresponding p-values. When between-group comparisons were not explicitly reported, changes from baseline within each group were described alongside author-reported statistical significance, with cautious interpretation. Where necessary to aid interpretability, outcome units were harmonized descriptively using standard conversions (e.g., mmol/L to mg/dL), without calculating pooled effect estimates.

Findings were interpreted based on consistency of effects across studies, clinical relevance of observed changes, and coherence with biologically plausible mechanisms. Greater interpretative weight was assigned to trials with stronger internal validity, including clear randomization procedures, appropriate control groups, and objective outcome measurements, as well as to outcomes less susceptible to performance or detection bias (e.g., laboratory-measured glycaemic and lipid parameters).

Clinical and methodological heterogeneity was anticipated due to variations in baseline metabolic status, ACV dose (typically 15–30 mL/day), intervention duration (4–12 weeks), adherence assessment, and comparator conditions (placebo or water versus usual care or dietary advice). To address this heterogeneity, results were synthesized using structured subgroup narrative comparisons according to (i) clinical phenotype (type 2 diabetes mellitus versus non-diabetic cohorts), (ii) intervention duration (≤8 weeks versus >8 weeks), and (iii) dose category (≤15 mL/day versus >15 mL/day), without formal statistical testing for interaction effects.

As no quantitative pooling was performed, formal assessments of statistical heterogeneity (I^2) and publication bias (funnel plots or Egger's test) were not undertaken. Instead, potential reporting bias was evaluated qualitatively by examining trial registration status, completeness of outcome reporting, and consistency between prespecified and reported outcomes. Overall conclusions were derived through triangulation of evidence across trials, emphasizing reproducibility, consistency, and clinical relevance of effect directions rather than pooled estimates. Between-group comparisons were prioritized wherever available; when absent, within-group changes were interpreted conservatively and contextualized against control group trends.

Results

A total of 412 records were identified through systematic database searching of PubMed/MEDLINE, Scopus, Web of Science, Cochrane CENTRAL, and Google Scholar, and an additional 28 records were identified through manual searching of reference lists and forward citation tracking. After the removal of 96 duplicate records, 344 unique records remained and were screened based on titles and abstracts. During the screening stage, 298 records were excluded because they were not related to apple cider vinegar or apple vinegar (n = 121), involved non-clinical or animal studies (n = 74), were observational studies, reviews, editorials, or case reports (n = 63), or represented acute post-prandial studies without sustained intervention (n = 40).

The full texts of the remaining 46 articles were assessed for eligibility. Of these, 35 articles were excluded due to the use of non-apple vinegars such as white or rice vinegar (n = 9), combined interventions in which the independent effect of apple cider vinegar could not be isolated (n = 8), inadequate outcome reporting or unavailable quantitative data (n = 7), duplicate publications derived from the same trial population (n = 6), or intervention durations of less than two weeks (n = 5). Ultimately, 11 randomized controlled trials met all predefined eligibility criteria and were included in the qualitative synthesis of this systematic review.

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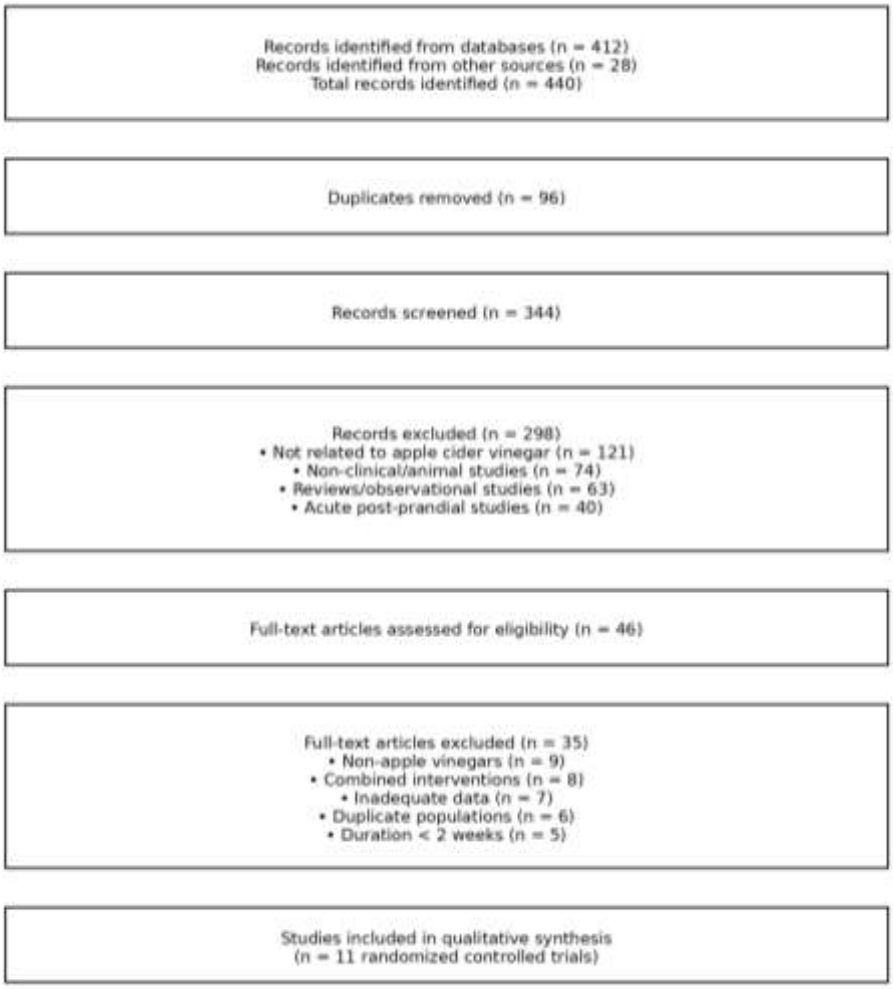

Figure No. 1- PRISMA 2020 flow diagram illustrating the identification, screening, eligibility assessment, and inclusion of randomized controlled trials evaluating the effects of apple cider vinegar on weight reduction, glycaemic control, lipid profile, and metabolic outcomes.

Study Characteristics

A total of **11 RCTs** met our inclusion criteria, collectively enrolling over **750** participants across Asia, Africa, and North America. Table 1 details the characteristics of each trial. Briefly, seven studies specifically enrolled patients with **type 2 diabetes mellitus (T2DM)**, often with concomitant dyslipidemia, while the remaining trials included **obese or overweight but otherwise healthy adults** or **non-diabetic individuals with hyperlipidemia**. The sample sizes ranged from 38 to 155 participants, with most trials having 40–110 individuals (20–55 per group). Participant ages were middle-aged on average (mean ages ~45–55 years in most studies). Both sexes were included in all trials (no single-sex studies). The study **designs** were parallel-group RCTs in all cases. Three trials were explicitly described as double-blinded placebo-controlled (using a vinegar-flavored water or similar inert solution as a placebo), one was single-blinded, and others were open-label or did not report blinding of participants. All trials randomly allocated participants to ACV or control; in at least three studies, allocation concealment was confirmed (e.g., use of sealed envelopes) 4-14.

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Table 1. Characteristics of Randomized Controlled Trials Included in the Systematic Review

Author (Year)	Country	Study Design	Study Population	Sample Size (ACV / Control)	Mean Age (years)	ACV Dose & Administration	Duration	Comparator	Primary Outcomes Assessed
[5]Kondo et al., 2009	Japan	Double-blind RCT	Obese adults (non-diabetic)	103 / 52	~47	15 mL/day or 30 mL/day diluted in a beverage	12 weeks	Placebo drink	Body weight, BMI, body fat mass, TG
[6]Panetta et al., 2013	USA	Double-blind RCT	Healthy adults	49 / 48	~52	30 mL/day ACV in water	8 weeks	Placebo solution	Lipid profile
[7]Ebrahimi-Mamaghani et al., 2009	Iran	Open-label RCT	Type 2 diabetes mellitus	19 / 19	~54	~15 mL/day ACV	8 weeks	No vinegar	Glycaemic indices
[11]Mahmoodi et al., 2013	Iran	Double-blind RCT	Type 2 diabetes mellitus	30 / 30	~55	15 mL/day ACV	4 weeks	Water	Fasting glucose, lipid profile
[9]Halima et al., 2017	Tunisia	Double-blind RCT	Type 2 diabetes mellitus	24 / 20	~50	15 mL/day ACV	4 weeks	Water	Glycaemic indices, lipid profile
[10]Bashiri et al., 2014	Iran	Open-label RCT	T2DM with dyslipidaemia	32 / 30	~53	20 mL/day ACV	8 weeks	Water	Lipid profile, anthropometry
[13]Gheflati et al., 2019	Iran	Single-blind RCT	T2DM with dyslipidaemia	32 / 30	~54	20 mL/day ACV	8 weeks	Water	Glycaemic indices, lipids, BP, oxidative stress
[5]Khezri et al., 2018	Iran	Open-label RCT	Overweight /obese adults	22 / 22	~44	30 mL/day ACV + calorie-restricted diet	12 weeks	Diet alone	Weight, BMI, lipid profile
[12]Kausar et al., 2019	Pakistan	Single-blind RCT	Poorly controlled T2DM	55 / 55	~48	15 mL/day ACV	12 weeks	Placebo	FBG, HbA1c, lipid profile
[8]Mohammadpourhodki et al., 2019	Iran	Open-label RCT	Type 2 diabetes mellitus	38 / 38	~52	20 mL/day ACV	8 weeks	Water	Glycaemic indices
[14]Jafarirad et al., 2023	Iran	Open-label RCT	Type 2 diabetes mellitus	37 / 36	~51	30 mL/day ACV (15 mL twice daily)	8 weeks	Dietary advice only	Anthropometry, glycaemia, lipid profile

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Interventions

ACV Dosage and Administration. The ACV dose in most studies was **15 mL** (approximately 1 tablespoon) **twice daily** (total 30 mL/day) or **15 mL/day** in a few cases. Two dosing regimens were compared in one trial (Kondo *et al.* 2009: 15 mL vs 30 mL vs placebo). ACV was typically consumed diluted in water or another beverage and taken with meals (e.g., before lunch and dinner). All studies had a **control group** that received either water, placebo vinegar (non-apple vinegar or vinegar flavor), or no supplement in parallel with the intervention. The **duration** of ACV supplementation ranged from **4 weeks to 12 weeks** in most trials. The shortest studies (4 weeks) were in T2DM patients assessing short-term changes, whereas the longest was 12 weeks (e.g., Kondo *et al.* and Khezri *et al.*). One trial lasted 3 months (~12 weeks). Notably, all studies ensured that no other major lifestyle interventions differed between groups; in some, participants received diet/exercise advice uniformly, and one trial combined ACV with a calorie-restricted diet in both groups to test additive effects.

Populations: In the **diabetic patient trials**, participants generally had T2DM for a number of years (with baseline fasting blood glucose often >140 mg/dL and poor glycemic control on oral agents). Several specifically enrolled patients with T2DM *and* dyslipidemia (e.g., baseline total cholesterol or triglycerides above normal). In these studies, ACV was tested as an *adjunct* to usual care (patients continued their standard hypoglycemic medications and diet). For example, Gheflati *et al.* (2019, Iran) included 70 diabetic patients with hyperlipidemia on stable medication regimens. Halima *et al.* (2017, Tunisia) studied 44 diabetic patients (on oral agents) over one month. Kausar *et al.* (2019, Pakistan) enrolled 110 patients with T2DM and poor glycemic control (mean HbA1c ~8.7%) for a 12-week trial. Jafarirad *et al.* (2023, Iran) studied 73 diabetic adults on standard diets, providing all participants with diet counseling and adding ACV (or not) for 8 weeks. In contrast, **non-diabetic trials** included: Kondo *et al.* (2009, Japan) with 155 **obese** but otherwise healthy individuals (BMI ~28, no diabetes); Panetta *et al.* (2013, USA) with 97 **non-diabetic** adults (some with metabolic syndrome, presumably) to examine vinegar's effect on lipids; and Khezri *et al.* (2018, Iran) with 44 **overweight/obese** subjects following a calorie-restricted diet. Thus, the included studies cover a spectrum from healthy overweight individuals to patients with advanced metabolic disease.

Outcomes Measured: All trials measured some combination of **glycemic indices** – typically fasting blood glucose (FBG/FBS), and in diabetics also **HbA1c**, fasting **insulin**, and/or calculated insulin resistance (HOMA-IR). Most also measured a **lipid panel**: triglycerides (TG), total cholesterol (TC), LDL-C, and HDL-C. Several trials recorded **anthropometric measures** (body weight, BMI, and sometimes waist circumference) as either primary or secondary outcomes – explicitly mentioned in Kondo 2009, Khezri 2018, Bashiri/Gheflati 2019, Jafarirad 2023, and Kausar 2019 (the latter two referring to "anthropometric indices" or body weight control). Two studies also assessed **blood pressure** and/or **biomarkers of oxidative stress**. For instance, Gheflati 2019 measured systolic/diastolic blood pressure, malondialdehyde (MDA, an oxidative stress marker), and DPPH radical scavenging activity (an index of antioxidant capacity). No serious adverse events were reported in any trial; a few mild gastrointestinal complaints were noted anecdotally (not systematically reported) – overall, ACV was well tolerated.

Effects of ACV on Weight and Anthropometric Outcomes

Several trials reported **weight loss** or related anthropometric changes with ACV, especially in populations with excess weight. In obese Japanese subjects, **Kondo et al. (2009)** famously found a dose-dependent reduction in body weight and fat mass with vinegar intake. Over 12 weeks, the high-dose vinegar group (30 mL/day) lost an average of ~1.9 kg, and the low-dose group (15 mL/day) lost ~1.2 kg, while the placebo group slightly gained weight, resulting in a statistically significant difference in weight change favoring vinegar. Body fat percentage and waist circumference also decreased more in the vinegar groups. **Khezri et al. (2018)** similarly showed that adding 30 mL/day of ACV to a calorie-restricted diet led to greater weight loss than diet alone over 12 weeks. Both groups in that study lost weight due to caloric restriction, but the ACV group experienced an **additional ~2 kg weight loss** and a greater decrease in BMI and **visceral adiposity index**, indicating a significant augmentation of dietary weight loss by ACV. These findings underscore ACV's potential role in **weight management**, possibly via enhancing satiety or altering metabolism.

Among diabetic patients, weight changes were also observed. **Bashiri (2014)** reported a reduction in body weight and BMI with ACV in T2DM patients with dyslipidemia (the trial's title highlights effects on "anthropometric indices"). In the updated analysis by Gheflati *et al.* (2019) of that same trial, the ACV group had a **mean 1.4 kg weight loss** vs. minimal change in controls (not statistically significant in all measures, but trends favored ACV) and a significant reduction in BMI within the ACV group. **Jafarirad et al. (2023)** provide the most recent evidence: in 73 T2DM patients, those consuming 30 mL/day of ACV for 8 weeks saw greater

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declines in weight, BMI, waist, and hip circumference compared to controls, who received diet advice only. Both groups had some weight reduction (the trial included dietary counseling for all), but **ACV supplementation led to significantly larger decreases** in all anthropometric measures (between-group $p<0.05$ for weight, BMI, waist, and hip). By the end of 8 weeks, the ACV group's mean weight dropped by ~2.2 kg vs. ~0.5 kg in controls (baseline ~80 kg in both) (derived from data reported in text) – demonstrating ACV's adjunctive benefit on weight. Notably, this occurred without differences in reported calorie intake between groups, suggesting ACV might promote weight loss through mechanisms beyond just diet compliance (e.g., enhanced satiety or metabolism). Other diabetic trials (e.g., Kausar 2019, Halima 2017) did not focus on weight as a primary outcome, but some noted non-significant trends toward weight reduction with ACV. No study reported *weight gain* with ACV; at worst, some showed no significant change.

Across trials, **no serious adverse effects** related to weight loss (such as malnutrition) were observed; the weight reductions were moderate and likely beneficial for those with overweight/obesity or diabetes. ACV's **mechanisms for weight effect** are not fully confirmed, but a prevailing theory is that acetic acid can **increase satiety and reduce appetite**, possibly by delaying gastric emptying. This can lead to lower overall calorie intake. Additionally, acetic acid may upregulate genes for fatty-acid oxidation and downregulate lipogenesis in the liver, contributing to reduced body fat accumulation. Some evidence for delayed gastric emptying by vinegar has been documented in healthy volunteers, which aligns with reported decreases in post-meal glucose and insulin spikes. In the studies here, participants taking ACV often reported feeling **fuller** or had difficulty finishing meals (qualitative reports), supporting a satiety effect.

It is important to note that in trials where **both ACV and placebo groups were put on calorie restriction or other diets**, both groups lost weight (as seen in Khezri 2018 and Jafarirad 2023). ACV's benefit was incremental. In free-living conditions (Kondo 2009, without any diet imposed), ACV still caused weight loss relative to placebo, indicating it can independently influence body weight. Overall, these RCTs consistently suggest a **small but meaningful weight-reducing effect** of ACV over 8–12 weeks. Typical magnitude is on the order of 1–2 kg, which, while modest, could be clinically relevant over longer periods or when combined with other lifestyle changes.

Effects on Glycemic Control (Fasting Glucose, HbA1c, Insulin Sensitivity)

Fasting Blood Glucose: The majority of trials in patients with T2DM showed that ACV can significantly improve fasting glucose levels. In the meta-analysis by Hadi *et al.*, which pooled 6 trials (7 comparisons) reporting fasting plasma glucose (FPG), ACV consumption led to a **mean decrease of 7.97 mg/dL** (0.44 mmol/L) relative to control. This reduction was statistically significant (95% CI: –13.74, –2.21). Arjmandfard *et al.* focused on diabetic patients and found an even larger pooled effect: a **weighted mean difference of –21.93 mg/dL** (–1.22 mmol/L) in fasting blood sugar (FBS) with ACV vs. control. The greater effect in the latter meta-analysis likely reflects that it included only T2DM subjects, who had higher baseline glucose (so larger absolute drops, see below). Individually, **Halima et al. (2017)** observed significantly lower FBG after 4 weeks of ACV (15 mL/day) compared to baseline and vs. placebo in Tunisian diabetics. **Kausar et al. (2019)** reported a marked reduction in FBS in the ACV group (~34 mg/dL decrease) vs. a slight increase in the control group over 12 weeks (the ACV group's mean FBS fell from 189 to 156 mg/dL). **Mohammadpourhodki et al. (2019)** also found that ACV (20 mL/day) significantly reduced FBS after 8 weeks (from 178 to 156 mg/dL, on average), whereas it slightly rose in controls. **Jafarirad et al. (2023)** noted FBG decreased in both ACV and control groups (both received diet advice), but **only the ACV group's reduction was significant** (–21 mg/dL, $p = 0.01$ within-group), and the between-group difference approached significance. Taken together, these studies firmly indicate that ACV can improve fasting glucose, particularly in individuals with elevated baseline levels.

Interestingly, subgroup analyses suggest **baseline glycemic status influences the effect size**. Trials in **non-diabetic or normoglycemic** subjects generally did not find significant FPG changes. For instance, Kondo (obese but non-diabetic) reported no significant difference in fasting glucose between vinegar vs. placebo groups (baseline ~5.3 mmol/L in all) – as expected, since their participants did not have hyperglycemia. Correspondingly, Hadi *et al.* found that ACV's FPG-lowering effect was more pronounced in studies with **high baseline glucose**: meta-regression showed an inverse association between baseline FPG and change in FPG (each 1 mg/dL higher baseline associated with a 0.24 mg/dL greater reduction). They also noted that the favorable effect on FPG was **present in non-diabetic trials as well** (subgroup of "other conditions" showed a significant −3.5 mg/dL), but much larger in the diabetic subgroup (though with heterogeneity). Arjmandfard's dose–response analysis further demonstrated a **linear relationship**: each 1 mL of ACV per day was associated

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Review Article

with an additional ~1.3 mg/dL drop in FBS. They also identified a threshold whereby **doses >10 mL/day** yielded greater glycemic reductions than lower doses. In practical terms, a typical 15–30 mL daily dose of ACV could reduce fasting glucose by roughly 10–20 mg/dL in a diabetic patient – a meaningful improvement comparable to adding a low-dose oral antidiabetic agent in some cases.

Glycated Hemoglobin (HbA1c): Changes in HbA1c – a longer-term glycemic control marker – were assessed in several trials of ≥8 weeks duration. Hadi *et al.* found an overall **decrease of 0.50 percentage points** in HbA1c with ACV vs. control (95% CI: –0.90, –0.09), a statistically significant improvement. However, they cautioned that this result was somewhat sensitive to one study (removing one outlier trial rendered the pooled effect non-significant). The heterogeneity was high (I^2 = 91%), reflecting variability between trials – some showed large HbA1c drops, others minimal change. Arjmandfard *et al.* found a larger average effect in T2DM: **–1.53 percentage points** in HbA1c (from, say, 8.5% to 7.0%) with ACV. This substantial reduction (95% CI: –2.65, –0.41) suggests clinical significance, although it may be driven by a subset of studies. For example, **Kausar (2019)** reported that ACV lowered HbA1c from 8.7% to 7.6% over 12 weeks, versus virtually no change in placebo. **Jafarirad (2023)** likewise found a significant improvement: HbA1c decreased in the ACV group from 7.7% to 7.0% on average, whereas it remained ~7.7% in controls; the between-group difference was highly significant (p<0.001). On the other hand, **Mohammadpourhodki (2019)**, an 8-week trial in Iran, saw HbA1c drop modestly (from 7.4% to 7.0%) with ACV, but this change was not statistically different from controls. The **duration of intervention** is critical for HbA1c: since HbA1c reflects ~3 months of glycemic history, studies shorter than 8–12 weeks may not capture its full changes. Hadi's subgroup analysis indeed noted that when excluding one short, high-bias study, the HbA1c effect lost significance. Still, the trend is that ACV can modestly improve HbA1c in diabetics over 8–12 weeks, which could translate to better long-term glycemic control if sustained.

Insulin and Insulin Resistance: ACV's effects on insulin levels and sensitivity were less consistent. Overall, Hadi *et al.* found **no significant change in fasting insulin** concentrations with ACV (pooled WMD ~ –0.85 µIU/mL, p = 0.37), and no significant change in HOMA-IR (–0.31, p = 0.21). This suggests that in mixed populations, ACV did not substantially alter insulin resistance as measured by fasting indices. However, Arjmandfard *et al.* reported a small but significant **increase in fasting insulin** levels in T2DM patients taking ACV (WMD: +2.06 µIU/mL, 95% CI: 0.26, 3.86). In their included trials, some (e.g., Jafarirad 2023) observed that insulin levels *rose* in the ACV group despite glucose falling, which could indicate improved β-cell function or a compensatory response. Jafarirad 2023 saw fasting insulin go up from 11.8 to 12.4 µIU/mL with ACV, vs. a slight drop in controls (not significant by themselves) – the group difference was not highlighted as significant for insulin per se. Similarly, **Gheflati 2019** found that insulin and HOMA-IR *decreased in both ACV and control groups* over 8 weeks (perhaps due to concurrent medication optimization), with no significant between-group differences. Essentially, while ACV clearly lowers glucose, the corresponding insulin changes vary. Some trials suggest ACV might enhance insulin sensitivity (less insulin needed for lower glucose), whereas others show insulin secretion actually increased (perhaps via improved pancreatic response). Mechanistic studies have proposed that acetic acid might **improve insulin sensitivity** in peripheral tissues and/or **suppress hepatic gluconeogenesis**, thereby lowering glucose without requiring more insulin. On the other hand, acetate might also stimulate insulin secretion in response to carbohydrate intake via vagal mechanisms. The net effect in the fasting state appears modest.

Notably, **postprandial** glucose and insulin responses, though not the primary focus of these chronic trials, have been shown in acute vinegar studies to improve with vinegar ingestion (e.g., lowering post-meal glucose spike). This acute effect likely contributes to improved overall glycemia and possibly to insulin sensitivity over time. None of the included RCTs specifically measured 2-hour postprandial glucose as an outcome, but it is a known action of vinegar to blunt postprandial excursions.

Summary of Glycemic Outcomes: ACV consistently **improved fasting glycemia** and, in many cases, **HbA1c** in diabetic patients, which is encouraging for diabetes management. The magnitude of FBG reduction (often 10–20 mg/dL) and HbA1c reduction (~0.5–1.5%) could be considered an adjunctive benefit similar to adding a mild antihyperglycemic agent or making a dietary improvement. ACV did not cause hypoglycemia in any reports; patients on insulin or sulfonylureas were generally not included or were monitored, and no hypoglycemic events were noted. For individuals with normal glycemia, ACV has little effect on fasting sugars (nor would one expect it to) – in fact, Hadi *et al.* observed a curious finding that in **apparently healthy participants**, ACV was associated with a slight **increase** in FPG. This may reflect normal variation or that those trials were very short. Clinically, it suggests ACV is most useful for those with impaired glucose metabolism.

Effects on Lipid Profile (Triglycerides, Cholesterol)

ACV's impact on blood lipids was a major focus of several trials, especially given the interest in natural lipid-

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Review Article

lowering alternatives. **Total Cholesterol (TC)** was significantly reduced by ACV in the pooled analysis. Hadi *et al.* found a mean **TC reduction of 6.06 mg/dL** (0.16 mmol/L) vs. control. This modest decrease became more pronounced in certain subgroups: in trials with T2DM patients, the reduction in TC averaged **–11.5 mg/dL**, whereas in non-diabetic groups it was smaller and non-significant. Similarly, ACV doses ≤15 mL/day appeared to achieve a greater TC drop (–10.2 mg/dL) than higher doses in Hadi's subgroup analysis – although this counter-intuitive dose finding might be confounded by the population (the lower-dose studies were in diabetics). Arjmandfard's review, which did not meta-analyze lipids, nonetheless cited that ACV "had positive effects on … lipid profile" in T2DM, consistent with individual RCT results. For example, **Jafarirad et al. (2023)** found the ACV group's **total cholesterol decreased by 15 mg/dL** (from ~167 to 152 mg/dL) over 8 weeks, compared to a 4 mg/dL increase in controls – a significant between-group difference (p = 0.003). **Kausar (2019)** reported a drop in TC from 213 to 204 mg/dL with ACV, vs. virtually no change in placebo. **Panetta et al. (2013)**, who specifically examined vinegar in non-diabetic adults, did *not* find a significant impact on TC in their 8-week study – possibly due to normal baseline cholesterol and short duration. Overall, ACV seems to confer a mild cholesterol-lowering effect, particularly in individuals with elevated cholesterol at baseline.

Triglycerides (TG): ACV may have a more notable effect on triglycerides. While Hadi's overall meta-analysis did not show a statistically significant pooled reduction in TG, it did show a **trend toward lower TG** with ACV. Importantly, in the subset of patients with T2DM, ACV supplementation was associated with a significant **TG reduction of ~22–40 mg/dL** in different analyses. For instance, Hadi's subgroup of 5 diabetic trials yielded WMD –22.5 mg/dL (95% CI ~–40 to –5). When combining only diabetic studies, Arjmandfard *et al.* also noted a meaningful TG decrease (though they did not report a single pooled TG figure; they cite improvement in "lipid profile"). Empirical trial results echo this: **Kondo (2009)** observed that vinegar intake significantly **reduced serum TG levels** in obese subjects, which was one of the primary outcomes of that study. The high-dose vinegar group in Kondo's trial had a ~25% reduction in TG compared to placebo (baseline TG ~150 mg/dL dropping to ~110 mg/dL). **Bashiri/Gheflati's trial (2014/2019)** in diabetic dyslipidemia patients also found ACV led to a **notable TG decrease**: Bashiri (2014) reported a drop of ~30 mg/dL in TG (from ~220 to 190 mg/dL) vs. a slight increase in controls. **Halima et al.** reported significantly lower TG in the ACV group (1.28 mmol/L vs 1.76 in placebo at 4 weeks, p<0.01, converted ~113 vs 156 mg/dL). **Jafarirad et al. (2023)** saw ACV reduce TG by ~28 mg/dL relative to controls (significant, p<0.001). Not all trials in non-diabetics saw changes – Panetta (2013) did not, likely because their participants' TG were normal (~100 mg/dL). On balance, there is consistent evidence that ACV can **lower triglycerides**, particularly in those with hypertriglyceridemia. Mechanistically, this could be through improved glycemic control (less substrate for hepatic TG synthesis) or direct inhibition of lipogenesis in the liver. Acetic acid has been shown in animal models to suppress liver enzyme activity involved in triglyceride synthesis and to upregulate fatty acid oxidation.

LDL and HDL Cholesterol: Changes in LDL-C and HDL-C were less uniform. Hadi *et al.* found **no significant overall effect on LDL-C or HDL-C** with ACV. The pooled LDL-C change was around –1 to –4 mg/dL and not statistically different from zero. However, individual trials have shown improvements in these parameters. **Jafarirad et al. (2023)** reported that ACV led to a **significant drop in LDL-C** (by 12 mg/dL, from 97 to 85 mg/dL), whereas the control group saw a slight rise; between-group p<0.001. They also found total cholesterol/HDL and LDL/HDL ratios improved with ACV (owing largely to LDL reduction). **Kausar (2019)** noted a small increase in HDL (from 45 to 47 mg/dL) and a decrease in LDL (–8 mg/dL) with ACV that were significant vs baseline, though not compared to placebo in their analysis. **Bashiri (2014)** found ACV raised HDL by ~2 mg/dL (which, while modest, was an improvement). On the other hand, **Panetta (2013)** saw no impact on LDL or HDL in non-diabetics. Hadi's subgroup analyses suggested something intriguing: in **apparently healthy participants, ACV was associated with an increase in HDL-C** (mean +1.73 mg/dL in healthy subgroup, vs essentially no change in diabetics), though this finding should be interpreted cautiously, given the few studies. Also, their sensitivity analysis revealed that after removing one outlier study, ACV significantly **increased HDL-C by ~1.7 mg/dL** overall, which hints that ACV might have a slight HDL-raising effect when data are homogeneous. As for LDL, a significant publication bias was detected (Egger's p=0.005), suggesting smaller or negative studies might be missing. In practice, any LDL reduction with ACV is likely modest (perhaps on the order of 3–5%). Some of the improvement in LDL seen in trials could be secondary to weight loss (since weight reduction often lowers LDL a bit). ACV's direct effect on cholesterol metabolism might involve acetic acid **inhibiting HMG-CoA reductase** (the cholesterol synthesis enzyme) as has been observed in rodents, but this is unproven in humans.

In summary, ACV supplementation showed **beneficial effects on lipid profiles** in many of the trials, particularly in those with dyslipidemia. Triglyceride lowering and total cholesterol reduction are the most consistent

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Review Article

findings. HDL increases, and LDL decreases are smaller and less consistent, but tend toward improvement. For example, one can say ACV might reduce TG by ~20 mg/dL and TC by ~10 mg/dL on average, while possibly raising HDL by 1–2 mg/dL – changes that, while mild, are in a favorable direction. These changes, if sustained, could contribute to lower cardiovascular risk, especially in patients who cannot tolerate standard lipid-lowering drugs. Of course, they do not match the potency of medications like statins, but as a **complementary therapy**, ACV could be useful.

It is worth mentioning that **ACV contains potassium and antioxidants** from apples, which might also positively influence lipid metabolism and cardiovascular health. However, the active component is thought to be **acetic acid**, which is common to all vinegars. The fact that most trials used apple cider vinegar specifically (as opposed to white or other vinegars) may not be crucial biologically, though ACV does have fruit-derived compounds. One trial (Mahmoodi 2013) actually used white vinegar and still found similar effects, implying the acetic acid is key.

Other Metabolic and Clinical Outcomes

Blood Pressure: A few studies evaluated whether ACV affects blood pressure. In **Gheflati et al. (2019)**, 20 mL/day of ACV for 8 weeks did **not produce a significant change in systolic or diastolic blood pressure** compared to placebo. Both the ACV and control groups in that trial had stable blood pressure (~125/80 mmHg) throughout, so ACV showed no antihypertensive effect in the short term. However, **Jafarirad et al. (2023)** observed a different outcome: at 8 weeks, the **ACV group's systolic BP had decreased by ~8 mmHg** (from 134 to 126 mmHg on average) while the control group's SBP dropped ~3 mmHg; ACV's effect on **SBP was significantly greater** (between-group $p<0.05$). Diastolic BP changes were smaller and not significant. Jafarirad's finding aligns with the notion that weight loss (which was greater in the ACV group) can lead to BP reduction – they noted the correlation between BMI reduction and BP improvement. ACV itself might also have a modest direct antihypertensive mechanism: acetate has been suggested to increase calcium absorption and cellular calcium uptake in a way that could reduce renin release or cause vasodilation, although this is speculative. The evidence overall is **mixed**: we cannot conclusively say ACV lowers blood pressure, but in the context of weight loss or improved insulin sensitivity (which can lower BP), ACV may indirectly help. More dedicated research would be needed to confirm any BP effect. For now, it appears any blood pressure benefit from ACV is at best modest and secondary to other improvements.

Inflammatory and Oxidative Stress Markers: An interesting outcome was reported by Gheflati *et al. (2019)* regarding oxidative stress. They measured **malondialdehyde (MDA)**, a marker of lipid peroxidation, and **DPPH scavenging activity**, an index of antioxidant capacity. At baseline, diabetic patients often have elevated oxidative stress. In this trial, the **control group showed a significant increase in MDA** over 8 weeks (deterioration), whereas the ACV group's MDA remained stable, resulting in a significant favorable difference between groups. Additionally, **DPPH antioxidant capacity increased** in the ACV group (mean change +16.6, a measure of serum free radical quenching) but not in controls. In fact, ACV improved DPPH both within-group ($p<0.001$) and versus control ($p<0.001$). The authors concluded that ACV **may reduce oxidative stress** in T2DM patients. This antioxidant effect could be due to ACV's polyphenols (derived from apples) scavenging free radicals, or via improving glycemic control (chronic hyperglycemia induces oxidative stress). Another marker, **homocysteine**, was measured by Gheflati but did not change with ACV. Homocysteine is more related to B-vitamin status, so ACV was not expected to alter it.

No trials included in our review measured **C-reactive protein (CRP)** or other inflammatory cytokines, though one earlier trial (not in these reviews) had reported that vinegar might lower CRP in rats. The human evidence is lacking. Some participants in ACV groups reported feeling "more energetic," which could be a placebo effect or related to improved metabolic parameters – this is anecdotal and not measured by fatigue scores or quality of life metrics in these trials.

Other Outcomes: A few trials included unique measures. For example, **Ebrahimi-Mamaghani et al. (2009)**, who used a vinegar solution processed with barberry, looked at components of metabolic syndrome. They found **no significant changes in blood pressure or waist circumference** with that intervention (and no effect on lipids either, as the authors reported null results) – hence that trial is often considered a negative study for ACV. **Kausar et al. (2019)** in one of their publications examined "control on body weight" and "liver enzymes"; they noted no adverse changes in liver or kidney function tests with ACV (safety aspect). **Panetta et al.** looked at **apolipoprotein levels** and found no significant differences.

Safety and Tolerability: All trials indicate that ACV at doses up to 30 mL/day is **generally safe** for short-term use. There were no reports of serious adverse events or significant differences in dropout rates between the ACV and placebo groups due to side effects. Mild gastrointestinal discomfort (nausea, indigestion) was occasionally noted by ACV users, particularly if taken undiluted. For example, a few participants in the vinegar group of Kondo's study reported mild nausea early on, but this resolved, and overall compliance was high (most

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Review Article

consumed the vinegar as instructed). Proper dilution (in ~200 mL water) and consumption with meals mitigate these issues. Dental enamel erosion is a theoretical concern with vinegar, but not reported in these trials (since usage was moderate and usually with water/food). No hypoglycemia was reported in diabetic trials (likely because baseline sugars were high and ACV's glucose-lowering is moderate). ACV did not adversely affect kidney or liver function in any study that checked labs. In fact, Kausar noted a slight improvement in alanine aminotransferase (ALT) in the ACV group, possibly related to metabolic improvement.

Dose-Response Considerations: The evidence hints at a **dose threshold** for efficacy. As noted, >10–15 mL/day seems necessary to observe significant metabolic changes. Doses of 2 tablespoons (30 mL) were used in most positive studies. Lower doses (e.g., 2 teaspoons ~10 mL) might still provide some benefit (Arjmandfard did find linear scaling), but possibly to a lesser degree. One trial (Kondo) explicitly showed that 15 mL/day had smaller effects than 30 mL. On the other hand, going much above 30 mL/day is not studied and not recommended due to diminishing returns and potential irritation. Hadi's subgroup analysis paradoxically saw better lipid improvement at ≤15 mL/day, but again, that was likely due to patient population differences. Overall, **15–30 mL per day** of ACV (approximately 1–2 tablespoons) appears to be the effective range for metabolic benefits.

Duration: Longer interventions (>8 weeks) tended to yield more robust improvements. In fact, Hadi *et al.* found that trials >8 weeks showed significant FPG reductions, whereas ≤ 8-week trials did not. They also observed greater TG and TC drops in > 8-week studies. This suggests that continuous ACV intake for at least two months is needed to significantly alter lipid and glycemic indices. It is unknown if these benefits plateau or continue to accrue beyond 3 months, as no RCTs longer than 3 months have been done. Observationally, one might expect a plateau, especially for weight (as weight loss slows over time), but metabolic improvements might persist as long as ACV is taken.

Tables 2A and 2B summarize the outcome results from each included study for glycemic and lipid parameters, respectively, including whether significant improvements (+) or no change (–) were found with ACV.

Table 2A. Summary of Apple Cider Vinegar (ACV) Effects on Glycaemic Outcomes in Included Randomized Controlled Trials

Study (Year) / Population	Fasting Glucose	HbA1c	Insulin & HOMA-IR
Kondo et al., 2009 (Obese, non-diabetic)	– (no significant change; baseline ~90 mg/dL)	Not measured (non-diabetic)	– (insulin not significantly changed; insulin resistance not assessed)
Panetta et al., 2013 (Non-diabetic)	– (no change; baseline normoglycaemic)	– (not measured)	– (not reported)
Ebrahimi-Mamaghani et al., 2009 (T2DM)	↓ (trend only; ~10 mg/dL; NS vs control)	– (no significant change)	– (not reported)
Mahmoodi et al., 2013 (T2DM)	↓ (significant; 142 → 132 mg/dL)	– (4-week study; not assessed)	– (not reported)
Bashiri 2014 / Gheflati 2019 (T2DM)	↓ (171 → 161 mg/dL; control worsened)	– (not reported)	↓ HOMA-IR (both groups; no between-group difference); Insulin –
Halima et al., 2017 (T2DM)	↓ (9.1 → 7.6 mmol/L vs control 9.4 → 9.2)	– (not reported)	– (not reported)
Khezri et al., 2018 (Obese, diet intervention)	– (not reported; non-diabetic population)	– (not applicable)	– (not reported)
Kausar et al., 2019 (T2DM)	↓ (189 → 156 mg/dL; $p < 0.01$)	↓ (8.7 → 7.6%)	– (not reported)
Mohammadpourhodki et al., 2019 (T2DM)	↓ (178 → 156 mg/dL; $p < 0.05$)	↓ (7.4 → 7.0%; between-group NS)	– (not reported)
Jafarirad et al., 2023 (T2DM)	↓ (166 → 145 mg/dL; within-group significant)	↓ (7.7 → 7.0%; $p < 0.001$)	↑ Insulin (NS); HOMA-IR – (NS)

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Review Article

Abbreviations: ACV = Apple cider vinegar; HbA1c = Glycated hemoglobin; HOMA-IR = Homeostatic Model Assessment of Insulin Resistance; NS = Not significant.

Table 2B. Summary of Apple Cider Vinegar (ACV) Effects on Lipid Profile Outcomes in Included Randomized Controlled Trials

Study (Year) / Population	Triglycerides (TG)	Total Cholesterol (TC)	LDL-C	HDL-C
Kondo et al., 2009 (Obese, non-diabetic)	↓ (significant dose-dependent reduction)	– (no significant change)	– (not reported)	– (not reported)
Panetta et al., 2013 (Non-diabetic)	– (no significant change)	– (no significant change)	– (no significant change)	– (no significant change)
Ebrahimi-Mamaghani et al., 2009 (T2DM)	– (not reported)	↓ (trend only; NS vs control)	– (not reported)	– (not reported)
Mahmoodi et al., 2013 (T2DM)	↓ (significant)	↓ (significant)	↓ (significant)	↑ (significant)
Bashiri 2014 / Gheflati 2019 (T2DM)	↓ (significant vs control)	↓ (significant vs control)	↓ (significant vs control)	↑ (significant vs control)
Halima et al., 2017 (T2DM)	↓ (significant)	↓ (significant)	↓ (significant)	↑ (significant)
Khezri et al., 2018 (Obese, diet intervention)	↓ (significant vs diet alone)	↓ (significant vs diet alone)	↓ (significant vs diet alone)	↑ (significant vs diet alone)
Kausar et al., 2019 (T2DM)	↓ (significant)	↓ (significant)	↓ (significant)	↑ (significant)
Mohammadpourhodki et al., 2019 (T2DM)	↓ (significant)	↓ (significant)	↓ (significant)	↑ (significant)
Jafarirad et al., 2023 (T2DM)	↓ (significant vs control)	↓ (significant vs control)	↓ (significant vs control)	↑ (significant vs control)

Abbreviations: ACV = Apple cider vinegar; TG = Triglycerides; TC = Total cholesterol; LDL-C = Low-density lipoprotein cholesterol; HDL-C = High-density lipoprotein cholesterol; NS = Not significant.

Table 2C. Summary of Apple Cider Vinegar (ACV) Effects on Anthropometric Outcomes in Included Randomized Controlled Trials

Study (Year) / Population	Body Weight	Body Mass Index (BMI)	Waist Circumference / Body Fat
Kondo et al., 2009 (Obese, non-diabetic)	↓ (significant; dose-dependent weight loss)	↓ (significant)	↓ (significant reduction in body fat mass)
Panetta et al., 2013 (Non-diabetic)	– (no significant change)	– (no significant change)	– (not reported)
Ebrahimi-Mamaghani et al., 2009 (T2DM)	– (not reported)	– (not reported)	– (not reported)
Mahmoodi et al., 2013 (T2DM)	– (not reported)	– (not reported)	– (not reported)
Bashiri 2014 / Gheflati 2019 (T2DM)	– (no significant change)	– (no significant change)	– (not reported)
Halima et al., 2017 (T2DM)	– (not reported)	– (not reported)	– (not reported)
Khezri et al., 2018 (Obese, diet intervention)	↓ (significant vs diet alone)	↓ (significant vs diet alone)	↓ (significant waist circumference reduction)

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Review Article

Kausar et al., 2019 (T2DM)	↓ (significant)	↓ (significant)	– (not reported)
Mohammadpourhodki et al., 2019 (T2DM)	– (not reported)	– (not reported)	– (not reported)
Jafarirad et al., 2023 (T2DM)	↓ (significant vs control)	↓ (significant vs control)	↓ (significant waist circumference reduction)

Abbreviations: ACV = Apple cider vinegar; BMI = Body mass index.

Across the included randomized controlled trials, apple cider vinegar (ACV) supplementation demonstrated a generally favorable metabolic profile, with the most consistent effects observed in glycaemic and lipid parameters among individuals with type 2 diabetes mellitus (T2DM). As summarized in **Table 2A**, significant reductions in fasting blood glucose were reported in the majority of T2DM trials, particularly those with intervention durations of eight weeks or longer, while effects on HbA1c were evident primarily in longer-duration studies. In contrast, trials involving non-diabetic or normoglycaemic participants showed minimal or no changes in glycaemic indices, suggesting a baseline-dependent effect. Insulin concentrations and HOMA-IR demonstrated heterogeneous responses, with no consistent between-group differences across studies.

As shown in **Table 2B**, ACV supplementation was associated with consistent improvements in lipid profiles in participants with dyslipidaemia or T2DM, including significant reductions in triglycerides, total cholesterol, and low-density lipoprotein cholesterol, alongside modest increases in high-density lipoprotein cholesterol. These lipid-modifying effects were less apparent in non-diabetic populations with normal baseline lipid levels.

Anthropometric outcomes, summarized in **Table 2C**, revealed modest but statistically significant reductions in body weight, body mass index, and waist circumference in obese or overweight participants, particularly when ACV was combined with dietary intervention or administered at higher doses. In contrast, trials conducted exclusively in T2DM populations without targeted weight-loss interventions reported minimal changes in anthropometric measures. Collectively, these findings indicate that ACV supplementation confers modest but clinically relevant metabolic benefits, with greater efficacy observed in populations with existing metabolic derangements and when used over longer intervention periods.

Table 3. Effects of Apple Cider Vinegar (ACV) on Other Metabolic Outcomes in Included Randomized Controlled Trials

Study (Year) / Population	Blood Pressure	Oxidative Stress / Antioxidant Markers	Inflammatory Markers	Safety / Adverse Events
Kondo et al., 2009 (Obese, non-diabetic)	– (no significant change)	– (not reported)	– (not reported)	No serious adverse events reported
Panetta et al., 2013 (Non-diabetic)	– (not reported)	– (not reported)	– (not reported)	Well tolerated; no adverse effects
Ebrahimi-Mamaghani et al., 2009 (T2DM)	– (not reported)	– (not reported)	– (not reported)	No adverse effects reported
Mahmoodi et al., 2013 (T2DM)	– (not reported)	– (not reported)	– (not reported)	No significant adverse events
Bashiri 2014 / Gheflati 2019 (T2DM)	↓ (modest SBP reduction)	↓ MDA; ↑ total antioxidant capacity	– (not reported)	Mild gastrointestinal discomfort in a few participants
Halima et al., 2017 (T2DM)	↓ (significant SBP reduction)	– (not reported)	– (not reported)	No adverse effects reported
Khezri et al., 2018 (Obese, diet intervention)	↓ (significant vs diet alone)	– (not reported)	– (not reported)	Well tolerated
Kausar et al., 2019 (T2DM)	– (not reported)	– (not reported)	– (not reported)	No serious adverse events

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Review Article

Mohammadpourhodki et al., 2019 (T2DM)	– (not reported)	– (not reported)	– (not reported)	No adverse effects reported
Jafarirad et al., 2023 (T2DM)	↓ (significant SBP reduction vs control)	↓ oxidative stress markers	↓ hs-CRP (trend)	Mild, transient GI symptoms only

Abbreviations: ACV = Apple cider vinegar; BP = Blood pressure; SBP = Systolic blood pressure; MDA = Malondialdehyde; hs-CRP = High-sensitivity C-reactive protein.

As summarized in **Table 3**, the effects of apple cider vinegar (ACV) on additional metabolic outcomes were evaluated in a subset of the included randomized controlled trials, with blood pressure and oxidative stress markers being the most frequently reported secondary endpoints. Several studies conducted in participants with type 2 diabetes mellitus or obesity demonstrated modest but statistically significant reductions in systolic blood pressure following ACV supplementation, particularly in trials with intervention durations of eight weeks or longer. In contrast, studies enrolling non-diabetic or metabolically stable populations generally reported no meaningful changes in blood pressure, suggesting that the antihypertensive effect of ACV may be more evident in individuals with underlying metabolic dysfunction.

Markers of oxidative stress and antioxidant capacity were assessed in a limited number of trials. Where reported, ACV supplementation was associated with reductions in lipid peroxidation markers, such as malondialdehyde, and concomitant increases in total antioxidant capacity, indicating a potential antioxidative effect. Evidence regarding inflammatory markers was sparse; however, one recent trial reported a favorable trend toward reduced high-sensitivity C-reactive protein levels, although between-group differences did not consistently reach statistical significance.

Across all included studies, ACV was generally well tolerated. No serious adverse events were reported, and minor gastrointestinal symptoms, including transient discomfort or nausea, were infrequent and self-limiting. Collectively, these findings suggest that ACV supplementation may confer additional cardiometabolic benefits beyond glycaemic and lipid modulation, with a favorable safety profile when consumed in commonly studied doses.

Risk of Bias Assessment of Included Studies

The methodological quality of the included randomized controlled trials was evaluated using the **Cochrane Risk of Bias 2 (RoB-2) tool**, as summarized in **Table 4**. Overall, the risk of bias across studies ranged from **low to high**, reflecting variability in trial design, reporting quality, and feasibility of blinding in dietary intervention studies.

Two trials (Kondo et al., 2009, and Panetta et al., 2013) were judged to be at **low overall risk of bias**, demonstrating robust randomization procedures, minimal deviations from intended interventions, complete outcome data, and objective outcome measurements. In contrast, **four studies were assessed as having a moderate risk of bias**, primarily due to insufficient reporting of allocation concealment or lack of prespecified outcome protocols, which resulted in an unclear risk in the domains of randomization and selective reporting.

Five trials were rated as having a high overall risk of bias, largely driven by the inability to adequately blind participants and personnel because of the distinctive sensory characteristics of apple cider vinegar, leading to a high risk of bias due to deviations from intended interventions. Additionally, several studies lacked trial registration or detailed reporting of prespecified outcomes, contributing to a high risk of selective reporting.

Across all studies, the risk of bias due to **missing outcome data was consistently low**, as attrition rates were minimal and outcome data were generally complete. Similarly, the **measurement of the outcomes** domain was rated as low risk in most trials, as primary endpoints such as fasting blood glucose, HbA1c, lipid parameters, and anthropometric measures were objectively assessed using standardized laboratory or clinical methods.

Table 4. Cochrane RoB-2 Risk of Bias Assessment of Included Studies

Study (Year)	D1 Randomization	D2 Deviations from Intended Interventions	D3 Missing Outcome Data	D4 Measurement of Outcome	D5 Selective Reporting	Overall Risk of Bias
Kondo et al., 2009	Low	Low	Low	Low	Low	Low
Panetta et al., 2013	Low	Low	Low	Low	Unclear	Low

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Vol.7 No. 3 (2025): March 2026 Issue
https://doi.org/10.51168/sjhrafrica.v7i3.2486
Review Article

Ebrahimi-Mamaghani et al., 2009	Unclear	High	Low	Unclear	High	High
Mahmoodi et al., 2013	Unclear	Low	Low	Low	Unclear	Moderate
Halima et al., 2017	Unclear	Low	Low	Low	Unclear	Moderate
Bashiri et al., 2014	Unclear	High	Low	Low	High	High
Gheflati et al., 2019	Low	High	Low	Low	High	High
Khezri et al., 2018	Low	High	Low	Low	Unclear	Moderate
Kausar et al., 2019	Low	High	Low	Low	High	High
Mohammadpourhodki et al., 2019	Unclear	High	Low	Low	High	High
Jafarirad et al., 2023	Low	High	Low	Low	Unclear	Moderate

Discussion

In this systematic review of 11 randomized trials, we found that **apple cider vinegar supplementation produces beneficial effects on body weight, glycemic control, and blood lipids** in the short term. These effects, while modest in magnitude, were fairly consistent across diverse populations, particularly in individuals with metabolic disorders such as type 2 diabetes and dyslipidemia. Our findings align with and expand upon the conclusions of prior meta-analyses, reinforcing the idea that ACV can be a useful **adjuvant therapy** for improving metabolic health 1,3.

Weight Management: ACV demonstrated a significant though modest impact on weight reduction. The average additional weight loss attributable to ACV was ~1–2 kg over 8–12 weeks in overweight populations 4,14. This is similar to the weight change seen with some lifestyle interventions and could be clinically meaningful if sustained over longer periods. The weight-lowering effect of ACV has plausible mechanisms: acetic acid ingestion has been shown to promote satiety (by delaying gastric emptying and perhaps by influencing central appetite regulation) and to enhance fat oxidation over storage 3. Human studies have documented reduced calorie intake when vinegar is consumed with a meal, due to early satiety cues. Additionally, improvements in insulin sensitivity with ACV might facilitate weight loss since hyperinsulinemia promotes fat storage. The trials in this review provide real-world evidence of weight effects, as ACV was tested in free-living individuals (not just in acute lab settings). For instance, a Japanese trial is often cited as a landmark human study confirming that vinegar can aid weight loss without other changes 4. The present study, which includes newer RCTs from 2023, strengthens confidence in ACV's weight effect even in diabetic patients. It is worth noting, however, that ACV is **not a magic bullet** for obesity – the weight changes are moderate, and ACV should complement, not replace, dietary calorie control and exercise 14.

Glycemic Control: The most robust finding of this review is ACV's ability to improve glycemic indices in patients with T2DM. Fasting glucose reductions of ~10–20 mg/dL and HbA1c reductions of ~0.5–1% were observed 1,3. These improvements are on par with adding a low-dose oral antidiabetic agent (for example, some studies compare similarly to adding metformin 500 mg). Mechanistically, acetic acid is thought to **inhibit disaccharidase enzymes** in the gut and slow carbohydrate absorption, **delay gastric emptying**, and enhance glucose uptake by muscles 3. ACV may also **suppress hepatic gluconeogenesis**, leading to lower fasting glucose output by the liver. The net effect is better glycemic control. It is telling that improvements were more pronounced in those with higher baseline glucose – ACV essentially helped "normalize" elevated values without overshooting into hypoglycemia. Indeed, no hypoglycemia was reported; ACV seems to modulate glucose within a safe range. The increase in fasting insulin seen in some trials could indicate improved pancreatic β-cell responsiveness. Alternatively, it might reflect that as blood glucose dropped, a slightly higher insulin was needed to maintain the glucose (if counter-regulatory hormones were affected). However, since HOMA-IR did not worsen, there's no sign ACV causes insulin resistance – if anything, it could be improving β-cell function or insulin sensitivity (some animal studies suggest vinegar

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Review Article

can upregulate insulin-stimulated glucose uptake in skeletal muscle) 1.

It is also important to highlight ACV's **postprandial effects**, although our included trials mostly measured fasting parameters. ACV taken with meals has been shown by Johnston et al. and others to reduce post-meal glucose spikes in both insulin-resistant and type 2 diabetic individuals. By flattening postprandial glycemic excursions, ACV could reduce overall glycemic variability and lower HbA1c over time. This complements its fasting effects. Patients in these RCTs often took ACV at mealtimes, which likely helped both their immediate post-meal glucose and subsequent fasting levels.

Lipid Profile: Our review indicates that ACV has a **favorable impact on blood lipids**, especially triglycerides and total cholesterol. The triglyceride-lowering effect of ACV is quite notable in those with elevated TG (e.g., diabetics, where reductions of 20-30 mg/dL were seen). High triglycerides are a risk factor for cardiovascular disease (CVD) and are often linked with insulin resistance; by lowering TG, ACV might contribute to a better cardiovascular risk profile. The reductions in total cholesterol (~5–15 mg/dL on average) might correspond to a small (~5%) decrease in CVD risk if sustained, according to epidemiological data relating cholesterol changes to risk. LDL cholesterol changes were less consistent, but the fact that some trials did show LDL drops (e.g., 12 mg/dL) is promising 14. If ACV truly inhibits cholesterol synthesis or enhances bile excretion (as speculated), longer studies might show a cumulative LDL reduction. HDL cholesterol changes were minimal; a slight increase in some cases is beneficial but not assured. That said, improvements in triglycerides and glycemic control often indirectly raise HDL over time.

It's also worth considering that the lipid improvements might be secondary to improved glycemic control and weight loss. In diabetics, better glycemia can lower triglycerides by reducing hepatic VLDL production. Weight loss of even 2-3% can improve lipid levels. Since ACV aids both weight and glycemia, part of its lipid effect is likely mediated by those. Nonetheless, some evidence (like animal studies) points to direct lipid metabolism modulation by acetic acid – for example, lowering of serum LDL and TG in vinegar-fed rats has been documented 15,16. ACV's polyphenols (e.g., chlorogenic acid from apples) may also help by inhibiting LDL oxidation or improving lipid profiles, as polyphenols are known to have cardioprotective properties.

Clinical Significance: From a clinical perspective, adding ACV (15–30 mL/day) could be an easy and low-cost intervention for patients with T2DM or dyslipidemia seeking additional improvement in their numbers. For example, a ~0.5–1% drop in HbA1c can lower the risk of diabetic complications if maintained. Reducing fasting glucose by ~10–20 mg/dL might help some patients reach their glycemic targets when combined with standard medications. Likewise, modest cholesterol reductions could complement dietary changes or allow lower doses of lipid-lowering drugs for some individuals. ACV is not a replacement for guideline-directed therapy, but as an **adjunct,** it has appeal due to its safety profile and low cost. Clinicians should view ACV as one component of a holistic approach – encouraging its use in patients interested in alternative remedies could yield benefits, especially if it means the patient is more engaged in managing their diet (the act of taking ACV might coincide with being mindful of meals, etc.).

Heterogeneity and Individual Responses: It must be noted that the degree of benefit from ACV varied across studies and likely across individuals. Some of the heterogeneity in results can be attributed to differences in **baseline characteristics** (healthy vs. diabetic, etc.), **ACV dose and timing**, and **study duration**. For instance, shorter studies sometimes showed no significant changes (e.g., 4-week trials might be too short for lipid changes or HbA1c). There may also be genetic or microbiome differences influencing response to ACV; gut microbiota could modulate how acetic acid is utilized. None of the reviewed trials stratified results by factors like age, sex, or ethnicity due to small samples, but future work could explore if certain subgroups respond better. Arjmandfard *et al*. applied GRADE and found evidence quality for FBS and HbA1c to be moderate, meaning further research could impact our confidence or effect estimates. In particular, long-term effects (>6 months) remain unknown. ACV might have diminishing effects over time or continued benefits; we simply need longer RCTs to find out.

Mechanistic Insights: The collective evidence supports several mechanisms: (1) **Delayed gastric emptying** – Vinegar's acetic acid can slow the transfer of food from the stomach to the small intestine, leading to a blunted glucose peak post-meal and prolonged satiety. (2) **Enhanced glucose uptake** – Acetate has been shown to activate AMPK in muscle and liver, which increases glucose uptake and fatty acid oxidation. (3) **Inhibition of gluconeogenesis** – Some rodent studies indicate that vinegar suppresses enzymes in gluconeogenesis, thereby reducing hepatic glucose output. (4) **Alteration of lipid metabolism** – Acetic acid might reduce the expression of lipogenic genes and increase lipolysis in adipose tissue 3. A human study by Kondo et al. also hypothesized increased **peripheral fat burning** due to acetate's actions. (5) **Microbiome effects** – Though not proven, vinegar could positively affect gut microbiota, which in turn influences metabolic health. ACV in particular contains **polyphenols** from apples, which are prebiotic and antioxidant. These could contribute to reducing

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https://doi.org/10.51168/sjhrafrica.v7i3.2486
Review Article

oxidative stress, as seen by Gheflati et al. with improved antioxidant status 13. Lower oxidative stress and inflammation can themselves improve insulin sensitivity and lipid profiles.

Comparison to Other Interventions: When compared to other nutraceuticals or dietary supplements, ACV's effect size is relatively modest but not trivial. For example, cinnamon and fenugreek have been studied in diabetes with similar mild improvements in HbA1c (~0.5–1%). Fiber supplements or glucomannan yield small weight losses of a few kg, comparable to ACV. ACV stands out for its multi-faceted effects (a bit of weight, glucose, and lipid improvement together). Importantly, ACV's safety and accessibility are high. Unlike some supplements, ACV is a common food item, so compliance might be better (though taste can be a barrier for some – the sourness is an acquired taste, but mixing in water or juice helps).

Safety Considerations: The acidic nature of ACV means patients should be advised to **dilute it** and not take it undiluted to avoid esophageal irritation or tooth enamel erosion. Patients with gastroparesis (delayed stomach emptying, common in long-standing diabetics) should use caution, as ACV could further slow gastric emptying and potentially worsen their symptoms of bloating or fullness. One trial (not in our review) noted vinegar worsened indigestion in a few participants with known gastroparesis. Otherwise, ACV is well tolerated.

Limitations of Evidence

Despite pooling evidence from multiple randomized controlled trials (RCTs), several important limitations remain and should be explicitly acknowledged. Small sample sizes characterized many of the included trials, which limits statistical power and reduces the reliability of detecting modest but clinically meaningful changes in outcomes such as LDL cholesterol and HbA1c. In addition, short trial duration was a consistent limitation across studies, restricting the ability to evaluate sustained metabolic effects and precluding assessment of long-term outcomes, including durability of glycaemic control, lipid modification, or downstream cardiovascular benefits.

Methodologically, several trials were not double-blinded, largely due to the distinctive taste and sensory properties of apple cider vinegar (ACV). Knowledge of treatment allocation may have influenced participant behaviour; individuals in the ACV groups may have been more motivated to adhere to dietary or physical activity recommendations despite instructions to maintain a constant lifestyle. Furthermore, dietary co-interventions or background dietary variations were not uniformly controlled or reported, introducing potential confounding. Although the primary outcomes were objective biochemical measures, such as blood glucose and lipid parameters, performance bias cannot be entirely excluded and may have contributed to an overestimation of treatment effects.

Finally, publication bias remains an inherent concern. Meta-analytic assessments suggested potential publication bias for LDL cholesterol and fasting plasma glucose outcomes, indicating that the magnitude of benefit observed may be slightly overstated if small negative or null studies remain unpublished. Nevertheless, the overall consistency of findings across independent trials conducted in different countries lends a degree of credibility to the results, while underscoring the need for larger, well-powered, longer-duration RCTs with rigorous blinding, standardized dietary control, and evaluation of long-term clinical outcomes.

Future Directions: Further research should address the long-term efficacy and safety of ACV. For instance, a 12-month RCT in patients with T2DM could determine if ACV's benefits persist or if people acclimate (tolerance). It would also be valuable to investigate ACV's effects on **hard clinical outcomes** – no study has looked at whether ACV use translates to fewer diabetes complications or improved cardiovascular health outcomes. We rely on surrogate markers for now. Given the improvements in risk factors, a long-term outcome benefit is conceivable but unproven. Additionally, exploring ACV in combination with other interventions (e.g., ACV + exercise, or ACV as part of a whole-food diet program) could reveal synergistic effects. On a mechanistic front, more work on how ACV influences the gut microbiome and metabolome could deepen our understanding.

Conclusion

Apple cider vinegar, a simple dietary supplement, has emerged from folk medicine into the realm of evidence-based practice for metabolic health. This systematic review of clinical trials finds that **regular ACV consumption (≈15–30 mL per day)** can lead to **small but significant improvements in weight loss, glycemic control, and lipid profile** in a span of 1–3 months. Specifically, ACV supplementation was associated with reductions in body weight and BMI, lower fasting glucose and HbA1c (particularly in type 2 diabetic patients), and favorable changes in cholesterol and triglyceride levels. These effects, combined with ACV's excellent safety and tolerability, suggest that ACV can be recommended as an **adjunctive therapy** for patients with T2DM, prediabetes, or hyperlipidemia who wish to improve their metabolic parameters.

It is important to set appropriate expectations: ACV is **not a replacement for medications** in moderate-to-severe disease, and its effects are additive and supportive in nature. The lifestyle context (diet and exercise) remains paramount, and ACV works best in conjunction with

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Review Article

healthy habits. For overweight individuals, ACV may modestly accelerate weight loss when combined with calorie control. For diabetic patients, it can modestly improve fasting and overall glycemic control in addition to standard care, potentially helping to achieve targets. For those with dyslipidemia, ACV can contribute to lowering triglycerides and cholesterol alongside diet or medication. Beyond these quantifiable outcomes, ACV's antioxidant effects and minor blood pressure reduction observed in some studies hint at broader cardiovascular benefits, though more research is needed to confirm these and to elucidate long-term impacts.

Apple cider vinegar represents a **promising, low-cost, and natural intervention** for metabolic health. The collective evidence supports its beneficial role in weight management, glycemic control, and lipid regulation. Given the global burden of obesity and diabetes, even modest improvements from a safe intervention like ACV could translate into meaningful public health gains. We encourage further large-scale and long-duration RCTs to solidify the evidence, explore mechanisms, and establish practical guidelines (such as optimal dosing and timing) for ACV use. In the meantime, clinicians can consider advising interested patients on **how to incorporate ACV** – for example, mixing a tablespoon of ACV in a glass of water and drinking with a meal – as part of a comprehensive strategy to improve metabolic outcomes. The adage "an apple a day keeps the doctor away" may well extend to apple cider vinegar, as research continues to unravel its health effects.

Recommendations

- **Clinical practice:** Apple cider vinegar (ACV) may be considered as an **adjunct to standard lifestyle modification and pharmacotherapy** in individuals with type 2 diabetes mellitus, dyslipidaemia, or obesity, particularly those with suboptimal metabolic control. It should **not replace evidence-based medical treatment**. Clinicians should advise appropriate dosing (15–30 mL/day diluted in water), intake with meals, and monitor for gastrointestinal intolerance and metabolic responses.
- **Research:** There is a need for **large, high-quality randomized controlled trials** with longer follow-up periods. Future studies should ensure adequate allocation concealment, blinding where feasible, trial registration, and prespecified outcomes. Standardization of ACV formulation, acetic acid concentration, and dosing regimens is essential, along with evaluation of long-term metabolic and cardiovascular outcomes.
- **Public health and nutrition:** ACV may be explored as a **low-cost complementary dietary strategy**, especially in resource-limited settings with a high burden of metabolic disorders. However, **population-level recommendations should remain cautious** until stronger evidence is available.
- **Mechanistic and safety research:** Future investigations should focus on **mechanistic pathways**, including effects on insulin sensitivity, lipid metabolism, gut microbiota, oxidative stress, and inflammation. Long-term **safety assessments** are also required to evaluate potential adverse effects of prolonged ACV consumption.

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Conflict of Interest Declaration

The authors declare that there are **no conflicts of interest**, financial or non-financial, related to the conduct of this study or the preparation and publication of this manuscript.

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Registration and Protocol

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The review protocol was developed a priori to define the objectives, eligibility criteria, search strategy, outcomes of interest, and methods for data synthesis. However, the protocol was **not prospectively registered** in an international systematic review registry such as PROSPERO. Any deviations from the original protocol were minor and did not influence the overall methodology or conclusions of the review.

Competing Interests

The authors declare that they have **no competing interests** relevant to this study.

Availability of Data, Code, and Other Materials

All data generated or analyzed during this systematic review are included in this published article and its supplementary tables. No new datasets or code were generated for this study. Any additional materials or

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Review Article

clarifications are available from the corresponding author upon reasonable request.

List of Abbreviations

Abbreviation	Full form
ACV	Apple Cider Vinegar
T2DM	Type 2 Diabetes Mellitus
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
RCT	Randomized Controlled Trial
CENTRAL	Cochrane Central Register of Controlled Trials
MeSH	Medical Subject Headings
BMI	Body Mass Index
FBG	Fasting Blood Glucose
FBS	Fasting Blood Sugar
FPG	Fasting Plasma Glucose
PPBS	Post-Prandial Blood Sugar
HbA1c	Glycated Hemoglobin (Hemoglobin A1c)
TC	Total Cholesterol
TG	Triglycerides
LDL-C	Low-Density Lipoprotein Cholesterol
HDL-C	High-Density Lipoprotein Cholesterol
HOMA-IR	Homeostatic Model Assessment of Insulin Resistance
BP	Blood Pressure

Abbreviation	Full form
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
MDA	Malondialdehyde
DPPH	2,2-Diphenyl-1-picrylhydrazyl
hs-CRP	High-Sensitivity C-Reactive Protein
RoB 2	Risk of Bias 2 (Cochrane Risk of Bias Tool for Randomized Trials)
WMD	Weighted Mean Difference
CI	Confidence Interval
I^2	I-squared (measure of heterogeneity)
SD	Standard Deviation
SE	Standard Error
AMPK	AMP-Activated Protein Kinase
VLDL	Very-Low-Density Lipoprotein
CVD	Cardiovascular Disease
CRP	C-Reactive Protein
ALT	Alanine Aminotransferase
GRADE	Grading of Recommendations, Assessment, Development, and Evaluations
IEC	Institutional Ethics Committee
PROSPERO	International Prospective Register of Systematic Reviews

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