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Effects of canola oil on body weight and composition in adults: an updated systematic review and meta-analysis of 32 randomized controlled trials

Abbas Mohtashamian[1,2,3], Masoumeh Mahabady[1], Fatemeh Bagheri[1], Hanieh Barghchi[2,3,4] and Azadeh Aminianfar[1*]

Abstract

Objective We aim to provide an overview and update the current documents regarding the effect of canola oil (CO) compared to other dietary oils on body weight and composition in adults.

Methods PubMed, Scopus, Google Scholar, and ISI Web of Science were searched until Sepetember 2024 for randomized clinical trials (RCTs) that assessed the effect of CO on anthropometric measures.

Results In this systematic review and meta-analysis thirty-two studies were included. CO consumption significantly increased WHR (MD: 0.003 cm, 95% CI: 0.001, 0.005, *P* value: 0.003) and significantly decreased BMI (mean difference (MD): -0.127 kg/m^2, 95% C: -0.231, -0.024, *P* value: 0.016) However, it did not significantly affect other anthropometric measures ($P > 0.05$). Based on subgroup analysis, CO supplementation significantly reduced BW in studies on T2DM patients, with parallel design, on patients over 50 years old and with a dose of more than 30 g/d. It also significantly increased WC in trials with parallel design and on hyperlipidemia patients. In addition, CO supplementation significantly increased WHR in the majority of subgroups.

Conclusions Compared to other oil supplementation, CO could decrease BW, BMI and increase WHR, and WC in general or subgroup analysis. Further studies are needed to provide additional insight into how canola oil affects BW and composition in adults.

Keywords Canola oil, Weight, Body composition, Randomized clinical trials, Systematic review, Meta-analysis

Introduction

Obesity is a well-known growing critical risk factor for chronic diseases such as cardiovascular disease and diabetes [1, 2]. This metabolic disorder is defined by the accumulation of fat caused by excess energy consumption [2]. It is reported that almost two billion people will have obesity and 671 million people will have health troubles owing to obesity by 2022 [3]. Overweight and obesity will affect 38% and 20% of the world's adults, respectively, by 2030 [4].

*Correspondence:
Azadeh Aminianfar
aaminianfar@gmail.com
[1] Research Center for Biochemistry and Nutrition in Metabolic Diseases, Basic Science Research Institute, Kashan University of Medical Sciences, Kashan, Iran
[2] Department of Clinical Nutrition, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad City, Iran
[3] Student Research Committee, Mashhad University of Medical Sciences, Mashhad City, Iran
[4] Department of Nutritional Sciences, Faculty of Medicine, Mashhad University of Medical Sciences, Mashhad City, Iran

Genetic and environmental factors such as inappropriate diet and low physical activity are the leading risk factors for obesity [5]. Previous studies have demonstrated that the composition of dietary macronutrients like carbohydrates, protein, and fatty acids is related to body weight and body composition [6]. Different fatty acids may play different roles in adiposity. For example, although higher consumption of polyunsaturated fatty acids might be related to weight loss [7], people with a higher intake of saturated fatty acids may experience weight gain [8]. According to this, plant oils with different compositions of fatty acids might affect anthropometric indices differently. Canola oil (CO) is a plant oil which is approved by the United States Food and Drug Administration as a healthy oil in 2006 [9]. It is rich in monounsaturated fats (MUFAs) such as oleic acid (61%) and polyunsaturated fats (PUFAs) such as linoleic acid (21%) and alpha-linolenic acid (11%), as well as a rich source of plant sterols and tocopherols which play an important role in health [10]. There are some documents which have shown that CO can reduce the level of plasma lipids [11]. In addition, the consumption of CO could affect the body's biological functions, and boost immune and cardiovascular health through its anti-thrombotic and anti-oxidative effects [10]. Moreover, PUFA Omega 3 could affect fat oxidation and satiety after meals in obese or overweight people during weight loss [12, 13].

Some previous clinical trials have assayed the effect of CO in comparison to other plant oils on the anthropometric indices and body composition and reached inconsistent results. For instance, in one study, CO caused a significant reduction in fat mass compared to other PUFAs [14]. In contrast, CO supplementation did not change cardiovascular health markers in another study [15]. In 2018, a systematic review and meta-analysis investigated the effect of CO consumption on some anthropometric measurements. It reported that CO supplementation could decrease body weight (BW), with no significant effect on body mass index (BMI), waist circumference (WC), fat mass (FM), waist-hip ratio (WHR), hip circumference (HC), lean body mass (LBM) [16]. Due to the controversial results and the fact that seven more studies have been published on the effects of CO on anthropometric indices, the need to update the previous study is felt. In addition, the effect of CO on visceral fat mass was assayed in the present meta-analysis for the first time. Therefore, we aimed to summarize the latest documents on the effect of CO supplementation on anthropometric indices and body composition.

Methods

The protocol of the present paper has been registered on the PROSPERO website with the registration code CRD42023438451. Also, we used the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement guidelines [17].

Search strategy

A systematic literature search was conducted in PubMed, Scopus and google scholar up to Sepetember 2024 by using the following Medical Subject Headings (MeSH) and non-MeSH keywords: 1) Canola OR colza OR rapeseed OR "brassica rapa" OR "oilseed rape" OR "brassica napus" OR "Brassica juncea" OR "canola oil" OR "rap oil" OR "rapeseed oil" 2) "body composition" OR "fat mass" OR "fat percentage" OR "body fat" OR "lean mass" OR "body lean" OR "body mass" OR weight OR Overweight OR Obesity OR "body mass index" OR BMI OR "Visceral adipose tissue" OR "adipose tissue" OR "Perinephric fat" OR "muscle mass" OR "waist circumference" OR WC OR "waist-hip ratio" OR WHR OR "fat percent" OR "lean body mass" OR LBM OR "weight loss" OR "weight reduction" OR "weight change" 3) "Randomized Controlled Trial" OR "clinical trial" OR "controlled trial" OR "intervention" OR "Randomised" OR "Randomized" OR "randomly" OR "placebo" OR "trial" OR "assignment" OR "RCT" OR "cross-over" OR "parallel" OR "single-blind" OR "double-blind" OR "Controlled Clinical Trial". In addition, the reference list of the included studies was reviewed to find other relevant articles. Appendix S1 shows the search strategy used for online databases.

Study selection

The eligibility of studies for the present systematic review and meta-analysis was determined by reviewing titles and abstracts of articles by A.M and F.B. Then, A.M and H.B reviewed the full text of selected articles. We resolved the discrepancies by discussing with A.A. We calculated the kappa statistic to determine the level of agreement between reviewers for study selection using SPSS software (ver. 26). To this end, the following interpretation of kappa was used: chance agreement (≤ 0), slight agreement (0.01–0.20), fair agreement (0.21–0.40), moderate agreement (0.41–0.60), substantial agreement (0.61–0.80), almost perfect agreement (0.81–0.99). In this stage, there was perfect agreement in study selection between the reviewers (K statistic, 0.82; $p < 0.001$).

The original articles included in this systematic review if: 1) were randomized controlled clinical trials (RCTs); 2) were done in adults (over 18 years); 3) the subjects involved were given canola oil supplement; 4) the authors reported sufficient information about BW, BMI,

HC, WC, WHR, VFM, FM and LBM. Exclusion criteria included: 1) intervention period < 2 weeks; 2) performed in children or adolescents; 3) CO consumption lower than values defined as reasonable based on previous research (< 10 g/d) [18].

Data collection

The required data were collected according to the guidelines of the PRISMA statement. Screening forms were used to identify eligible articles for this research having the inclusion criteria. The data of selected articles were independently reviewed by two authors (A.M. and F.B.). The continuance data collection process included extracting the following data from each study using Microsoft Office Excel 2016 MSO (16.0.4266.1001) software spreadsheet: publication characteristics (first author's full name, year of publication, and country where the study was conducted), participants data (age, health status, body mass index, and gender), characteristics of the study (number of participants, type of control treatment, duration of intervention, dose of intervention and placebo, study design), outcomes (BW, BMI, WHR, FM, LBM, VFM, WC, HC) and how to measure body composition.

We extracted the mean values and standard deviations for the outcomes at baseline, post-intervention, and the changes between them. If data were collected at several time points, just the last measurement values were utilized. Both authors (A.M. and F.B.) separately summarized the data from the included studies and resolved any discrepancies by consulting with A.A. Finally, Κ statistic was calculated to determine the agreement level between reviewers for data extraction using SPSS software (ver. 26).

Quality assessment

Two researchers (A.M. and F.B.) evaluated the methodological quality of the chosen full texts using the Cochrane criteria, independently [19]. As a result, the assessment of the studies' quality was done by considering allocation concealment, adequacy of sequence generation, blinding, disclosure of attrition (incomplete outcome data), selective reporting of results, and other sources of bias. The studies were categorized as having low, high, or unclear bias risk in each domain following the Cochrane Manual guidelines, as shown in Table 1.

Also, the Κ statistic was calculated to determine the level of agreement between reviewers for assessing the quality of included studies using SPSS software (ver. 26). Additionally, GRADE evidence profiles were applied to evaluate the overall evidence quality regarding body composition (Table 2).

Statistical analysis

We evaluated the effect of consuming *canola oil* on body weight and composition. The effect sizes were expressed as weighted mean differences (WMDs) along with 95% confidence intervals. We computed the net changes in body composition by extracting the mean (± SD) of pre- and post-intervention periods for both the *canola oil* and control groups: the value change between the end of the study and the beginning of the study is to subtract the value at baseline from the value at the end. The mean difference was calculated using the following method: (value at the end of follow-up in the treatment group—value at baseline in the treatment group) minus (value at the end of follow-up in the control group—value at baseline in the control group). When there was no informed standard deviation of the mean difference, the result was determined through a mathematical calculation using the following technique: SD = square root [(SD pre-treatment)^2 + (SD post-treatment)^2—(2 R × SD pre-treatment × SD post-treatment)], assuming a correlation coefficient of 0.5, as a conservative estimate for R which ranges between 0 and 1 [20]. In the case of medians and ranges or 95% CIs, mean and SD values were calculated utilizing the method developed by Hozo et al. [20]. Heterogeneity was tested using Cochran's Q-test (with significance set at $p < 0.1$) and the I^2 test to estimate the percentage of heterogeneity (I^2 value ≥ 50% representing significant heterogeneity). When heterogeneity existed, a random effects model was applied; otherwise, a fixed-effects model was applied. Furthermore, a leave-one-out sensitivity analysis was performed to evaluate each study's effect on the total effect size [20]. The potential publication bias was identified using the funnel plot, Begg's rank correlation, and Egger's weighted regression tests. Also, the analysis of the effects of publication bias was adjusted using the Duval & Tweedie "trim and fill" and "failsafe N" methods [21].

Fixed effect analysis was employed for all subgroup analyses. The Comprehensive Meta-analysis version 3.0 was used for all statistical analyses [22]. Statistically significant *P* value lower than 0.05 was considered.

Results

Results of the search and trial flow

Two authors independently screening the title, abstract and full text of the articles. In this stage, there was perfect agreement in study selection between the reviewers (Κ statistic, 0.86; $p < 0.001$).

From a total of 3094 articles found in various databases including PubMed-MEDLINE, Scopus, Cochrane Library, Web of Science, and Google Scholar, 312 duplicate articles were removed. We additionally removed

Table 1 Assessment of the studies' quality included according to Cochrane guidelines

Study	Random sequence generation	Allocation concealment	Blinding of participants and personal	Blinding of outcome assessment	Incomplete outcome data	Selective outcome reporting	Other potential threats to validity	General risk of bias
Kanikowska et al. 2019 [58]	U	H	U	H	L	L	H	High
Moghtaderi et al. 2020 [76]	L	U	L	L	L	L	H	Low
Nicol et al. 2020 [47]	L	U	H	L	L	H	L	Moderate
Moszak et al. 2020 [53]	L	U	L	H	L	L	L	Low
Raeisi-Dehkordi et al. 2021 [65]	U	H	L	L	L	L	L	Low
Kruse et al. 2020 [50]	U	U	H	H	L	L	L	Moderate
Chauhan et al. 2020 [73]	H	H	H	H	L	H	H	High
Dus-Zuchowska et al. 2019 [56]	L	U	L	H	L	L	L	Low
Bowen et al. 2018 [45]	L	L	L	U	L	H	L	Low
Atefi et al. 2018 [69]	U	H	H	H	L	H	L	High
Małgorzata et al. 2020 [54]	U	U	L	H	L	L	L	Low
Salar et al. 2015 [67]	U	U	H	H	L	H	L	High
Study	**Random sequence generation**	**Allocation concealment**	**Blinding of participants and personal**	**Blinding of outcome assessment**	**Incomplete outcome data**	**Selective outcome reporting**	**Other potential threats to validity**	**General risk of bias**
Azemati et al. 2012 [68]	U	U	H	H	L	L	L	Moderate
Iggman et al. 2011 [47]	U	H	H	H	L	H	L	High
Liu et al. 2016 [14]	U	U	L	L	L	L	H	Low
Baxheinrich et al. 2012 [60]	U	U	H	H	L	L	L	Moderate
Saedi et al. 2017 [71]	U	H	U	H	L	L	L	Moderate
Seppanen-Laakso et al. 1993 [61]	H	H	H	H	L	H	L	High
Öhrvall et al. 2001 [55]	U	U	L	H	L	H	L	Modeate
Jenkins et al. 2014 [54]	U	U	L	L	L	L	H	Low

Table 1 (continued)

Södergren et al. 2001 [48]	U	H	L	U	L	H	L	Moderate
Kratz et al. 2002 [51]	U	H	U	H	L	H	L	High
Uusitupa et al.1994 [44]	U	U	U	L	L	H	L	Low
Nydahl et al. 1995 [57]	U	H	H	U	L	H	L	High
Chisholm et al. 2005 [59]	U	U	U	H	L	H	L	Moderate
Noroozi et al. 2009 [72]	U	U	H	U	L	L	L	moderate
Study	**Random sequence generation**	**Allocation concealment**	**Blinding of participants and personal**	**Blinding of outcome assessment**	**Incomplete outcome data**	**Selective outcome reporting**	**Other potential threats to validity**	**General risk of bias**
Gustafsson et al. 1994 [62]	U	H	U	H	L	H	L	High
Herrmann et al. 1997 [46]	U	U	L	U	L	H	H	moderate
Wardlaw et al. 1991 [64]	U	U	L	L	L	H	L	Low
Kratz et al. 2002 [51]	U	H	H	H	L	H	L	High
Kruse et al. 2014 [53]	U	H	H	H	L	H	H	High
Bahareh Nikooyeh et.al. 2023 [75]	L	L	L	U	L	L	L	Low

Table 2 GRADE profile regarding the effect of canola oil on body composition

Quality assessment						Quality of evidence
Outcomes	**Risk of bias**	**Inconsistency**	**Indirectness**	**Imprecision**	**Publication bias**	
Body weight	No serious limitation	No serious limitation	No serious limitation	No serious limitation	No serious limitation	High
BMI	No serious limitation	No serious limitation	No serious limitation	No serious limitation	No serious limitation	High
FM	No serious limitation	No serious limitation	No serious limitation	No serious limitation	No serious limitation	High
HC	No serious limitation	No serious limitation	No serious limitation	No serious limitation	No serious limitation	High
LBM	No serious limitation	No serious limitation	No serious limitation	No serious limitation	No serious limitation	High
VFM	No serious limitation	No serious limitation	No serious limitation	No serious limitation	No serious limitation	High
WC	No serious limitation	serious limitation	No serious limitation	No serious limitation	No serious limitation	High ⊕
WHR	No serious limitation	No serious limitation	No serious limitation	No serious limitation	No serious limitation	High

2721 articles by screening the title and abstract. We examined the 53 articles that were left by reading all the content and eliminated 21 studies for various reasons: studies did not report the relevant endpoints ($n=5$) [23–27], reporting duplicate data ($n=2$) [28, 29], or having no data of interest ($n=14$) [30–43] (Fig. 1).

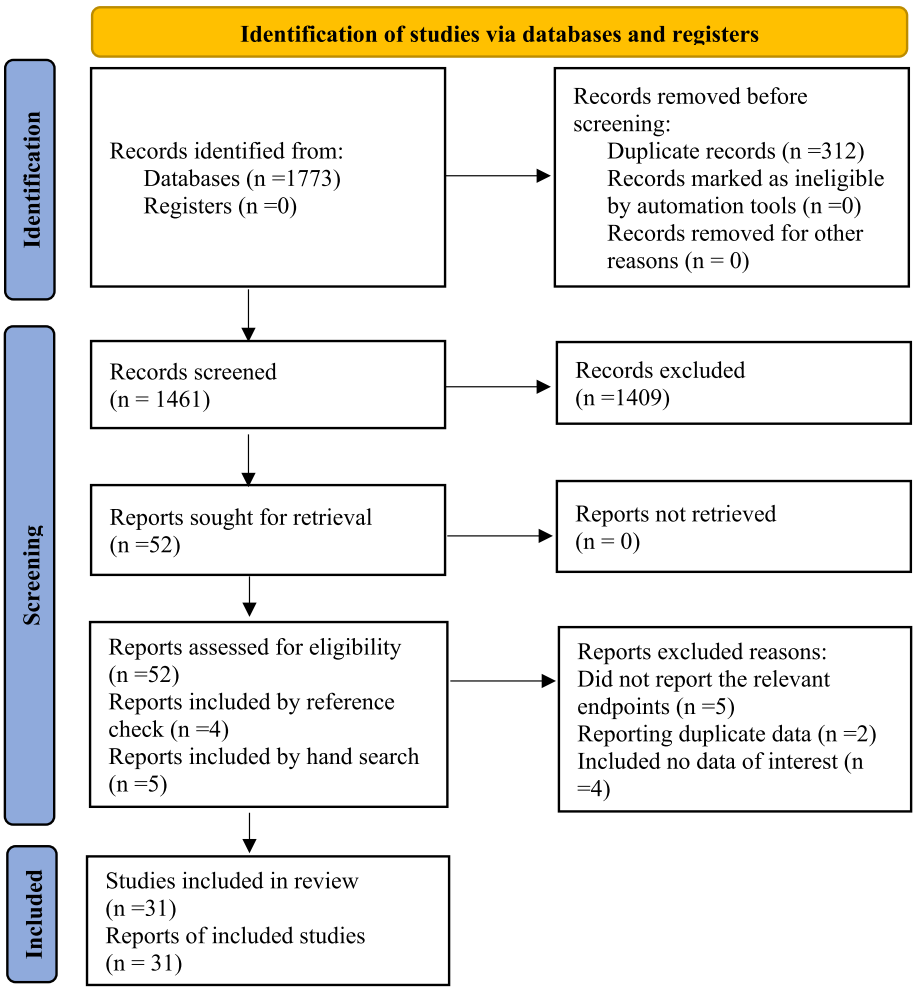

Fig. 1 Flow diagram of the study selection procedure 17 showing the number of eligible studies for the meta-analysis of the effect of canola oil on anthropometric measurements

Study characteristics

Characteristics of eligible studies are summarized in Table 3. The sample size of the included studies was between 10 [44] and 119 participants [45]. Out of the 32 included studies, 21 studies were performed in Europe [44–64], 1 in America [65] and 10 studies in Asia [66–75]. The duration of the trials was between 3 and 28 weeks. Five studies were conducted in women only [44, 64, 67, 69, 70], two in men only [51, 71] and the rest of the eligible studies involved both genders. 23 studies had a parallel design [46, 47, 49, 51–56, 59, 61–65, 67, 69–75], and nine studies had a crossover design [44, 45, 48, 50, 57, 58, 60, 66, 68]. A wide range of canola oil supplement doses between 12 g/d [46] and 50 g/d [53] were used in the intervention groups. Participant characteristics also varied between studies, many focusing on special and diseased populations: obesity [50, 53, 54, 57, 59], type 2 diabetes [55, 66, 67, 70, 75], metabolic syndrome [45, 61], NAFLD [51, 71], hyperlipidemia [48, 49, 58, 62–64, 72–74], healthy [44, 47, 52, 56, 60, 65, 68], coronary artery disease [46] and osteoporosis [69].

Meta-analysis results

Thirty studies including a total of 1772 participants reported BW as an outcome measure [44–61, 63–74]. Combined results from the fixed effects model indicated that BW did not change significantly following CO consumption (MD:—0.017 kg, 95% CI: −0.195, 0.161, *P* value: 0.85) (Fig. 2) with non-significant heterogeneity between the studies (I^2 = 0.0%, *P* value = 0.883, Mean PI = −0.01, 95% PI = −0.18, 0.16).

Twenty-one studies including a total of 1337 participants reported BMI as an outcome measure [47, 48, 51, 53, 54, 56, 57, 59–64, 66, 68–75]. The fixed effects model indicated that BMI change significantly following canola oil consumption in combined results (mean difference (MD): −0.127 kg/m^2, 95% C: −0.231, −0.024, *P* value: 0.016)(Fig. 3) with non-significant heterogeneity between studies (I^2 = 31.07%, *P* value = 0.064, Mean PI = −0.12, 95% PI = −0.43, 0.19).

Thirteen studies including a total of 659 participants reported an association between canola oil consumption and WHR [51, 53, 54, 56, 57, 59, 60, 63, 64, 66, 68, 69, 73]. Overall results from the fixed-effects model indicated that canola oil consumption resulted in a significant change in WHR (MD: 0.003 cm, 95% CI: 0.001, 0.005, *P* value: 0.003) (Fig. 4). There was no significant heterogeneity between these studies (I^2 = 36.915%, *P* value = 0.081, Mean PI = 0.003, 95% PI = −0.3, 0.31). As Azemati et al.'s study had a large deviation from the other studies with a difference in mean of 0.86 cm, we repeated the analysis once without this study. This exclusion did not alter the results (MD: 0.003 cm, *P* value:0.003).

Seven studies including a total of 434 participants reported fat mass as an outcome measure [51, 53, 54, 61, 64, 66, 68]. Combined results from the fixed effects model indicated that fat mass did.

not change significantly following canola oil consumption (MD: 0.101 kg, 95% CI: −0.191, 0.393, *P* value: 0.499) (Fig. 5) with non-significant heterogeneity between the studies (I^2 = 0.0%, *P* value = 0.981, Mean PI = 0.1, 95% PI = −0.28, 0.48).

Seven studies including a total of 505 participants reported HC as an outcome measure [54, 64, 66, 68, 69, 72, 73]. Combined results from the fixed effects model indicated that HC did not change significantly following canola oil consumption (MD: −0.135 cm, 95% CI: −0.531, 0.26, *P* value: 0.503) (Fig. 6) with non-significant heterogeneity between the studies (I^2 = 0.0%, *P* value = 0.995, Mean PI = −0.13, 95% PI = −0.64, 0.38).

Five studies including a total of 349 participants reported LBM as an outcome measure [54, 59, 61, 66, 68]. Combined results from the fixed effects model indicated that LBM did not change significantly following canola oil consumption (MD: −0.102 kg, 95% CI: −0.289, 0.086, *P* value: 0.287) (Fig. 7) with non-significant heterogeneity between the studies (I^2 = 0.0%, *P* value = 0.896, Mean PI = −0.1, 95% PI = −0.39, 0.19).

Three studies including a total of 249 participants reported VFM as an outcome measure [54, 66, 68]. Combined results from the fixed effects model indicated that VFM did not change significantly following canola oil consumption (MD: 0.014 kg, 95% CI: −0.126, 0.154, *P* value: 0.845) (Fig. 8) with non-significant heterogeneity between the studies (I^2 = 0.0%, *P* value = 0.883, Mean PI = 0.01, 95% PI = −0.89, 0.91).

Fourteen research projects, with a combined total of 1144 participants, used WC as a measurement for their results [47, 54, 55, 57, 61, 64, 66, 68–75]. The random effects model results showed that there was no significant change in WC after consuming canola oil (mean difference (MD): 0.325 cm, 95% CI: −0.47, 1.12, *P* value: 0.426) (Fig. 9) with significant heterogeneity between the studies (I^2 = 71.25%, *P* value < 0.001). As Noroozi et al. had a large deviation from the other studies with a difference in the mean of 24.4 cm, we performed the relevant analysis once again without of this study. No significant change occurred (mean difference (MD): 0.075 cm, *P* value: 0.76, Mean PI = 0.32, 95% PI = −0.8, 1.44).

Sensitivity analysis

The effect sizes for the effect of canola oil on all variables assessed in the present study were robust in sensitivity analyses, indicating that removing any trial did not significantly affect the results.

Table 3 Demographic Characteristics of the included studies

Author (yaer)	Country	Subjects	Age range or Mean SD	Mean of BMI at the baseline	Design	Intervention group	Comparator group	Duration (wk/d)	Participants	Outcomes	Outcomes measurement methods
Kanikowska et al. (2019) [58]	Poland	I: 11 C: 8	48.3 ± 16	41.1	Parallel- A randomized controlled trial	Calorie restriction diet (was based on a 25–30% reduction in caloric intake compared to total energy requirement and 20% calories from protein, 25–30% from fat and 50–55% from carbohydrates) + 20 mL/d canola oil	Calorie restriction diet (was based on a 25–30% reduction in caloric intake compared to total energy requirement and 20% calories from protein, 25–30% from fat and 50–55% from carbohydrates) + 20 mL/d amaranth oil	3 wk	Obese	BW, BMI, WHR, fat mass, fat free mass	Tanita MC 980 MA, Tokyo, Japan
Moghtaderi et al. (2020) [76]	Iran	I: 69 C: 70	47.43 ± 1.17	28.21	Triple blind, randomised, three-way cross-over clinical trial	A healthy dietary pattern (30–32% of total calorie needs from fats, 50–52% from carbohydrates, and 16–18% from proteins) + Canola oil	A healthy dietary pattern (30–32% of total calorie needs from fats, 50–52% from carbohydrates, and 16–18% from proteins) + sesame oil	9 wk	Healthy adults	BW, BMI, visceral fat, body fat, muscle mass, WC, HC, WHR	Bioimpedance analyser (Omron, mode: BF511)
Raeisi-Dehkordi et al. 2021 [65]	Iran	I: 95 C: 93	49.17 ± 0.70	28.93	Triple blind, randomised, three-way cross-over clinical trial	A healthy dietary pattern (30–32% of total calorie needs from fats, 50–52% from carbohydrates, and 16–18% from proteins) + Canola oil	A healthy dietary pattern (30–32% of total calorie needs from fats, 50–52% from carbohydrates, and 16–18% from proteins) + sesame oil	9 wk	T2DM	BW, BMI, visceral fat, body fat, WC, HC, WHR, muscle mass	Bioimpedance analyser (Omron, mode: BF511)

Table 3 (continued)

Nicol et al.(2022) [15]	Scotland	I: 21 C: 40	25–75 years	-	Parallel randomised control design	Normal diet + 20 ml/d RO oil	1.Normal diet + 20 ml/d SO oil 2.Normal diet	12 wk	Healthy	BW, BMI, WC	-
Moszak et al. (2020) [53]	Poland	I: 26 C: 55	18–70 years	39.6	Randomized, double-blind, controlled trial with 3 parallel groups	A calorie-restricted diet (70–75% of the total daily energy expenditure (TDEE) with an identical composition of macronutrients (20% protein, 25–30% fat, and 50–55% carbohydrates) and physical activity + 20 mL/d of RSO	1.A calorie-restricted diet ((70–75% of the total daily energy expenditure (TDEE) with an identical composition of macronutrients (20% protein, 25–30% fat, and 50–55% carbohydrates) and physical activity + 20 mL/d of ASO 2.A calorie-restricted diet ((70–75% of the total daily energy expenditure (TDEE) with an identical composition of macronutrients (20% protein, 25–30% fat, and 50–55% carbohydrates) and physical activity	3 wk	Obese	BW, BMI, WC, HC, WHR, FM, LBM, VFM	Bioelectrical impedance, Tanita MC 980 MA (Tanita, Tokyo, Japan)
Author (yaer)	**Country**	**Subjects**	**Age range or Mean SD**	**Mean of BMI at the baseline**	**Design**	**Intervention group**	**Comparator group**	**Duration (wk/d)**	**Participants**	**Outcomes**	**Outcomes measurement methods**
Kruse et al. (2020) [50]	Germany	I: 15 C: 11	27–72 years	-	Randomized controlled trial with an open-label, parallel-arm study design	Isocaloric diet + 50 g/d RA oil	Isocaloric diet + 50 g/d OL oil	8 wk	NAFLD	BW, BMI, total body fat, WHR	Air-displacement plethysmography system

Table 3 (continued)

Chauhan et al. (2020) [73]	India	I: 40 C: 40	30–45 years	-	Parallel-Non-randomized intervention trial	Diet and lifestyle advice for lipid lowering based on the NCEP-ATP III (2002) guidelines + 15 ml/d canola oil	Diet and lifestyle advice for lipid lowering based on the NCEP-ATP III (2002) guidelines + 15 ml/d usual edible oil(s)	12 wk	Dyslipidemic	BW, BMI, WC	-
Dus-Zuchowska et al. (2019) [56]	Poland	I: 44 C: 44	48.77 ± 10.21	31.00	A randomised, Double-Blind, Cross-Over Study	20 ml/d RA oil	20 ml/d Am oil	3 wk	Overweight and Obese	BW, WC, WHR, BMI	-
Bowen et al. (2018) [77]	Canada	I: 119 C: 119	44 ± 13	31.7	A double-blind, randomized, controlled feeding, crossover, clinical trial	Isocaloric, healthy, weight-maintenance base diet + canola oil	1.Isocaloric, healthy, weight-maintenance base diet + HOCO 2.Isocaloric, healthy, weight-maintenance base diet + control oil	6 wk	Metabolic syndrome	BW	-
Atefi et al.[69]	Iran	I: 26 C: 51	-	28	A single-centered, parallel group, and randomized controlled clinical trial	Balanced diet (55% carbohydrate, 18% protein and 27% fat) + 30 g/day CO	1.Balanced diet (55% carbohydrate, 18% protein and 27% fat) + 30 g/day OO 2.Balanced diet (55% carbohydrate, 18% protein and 27% fat) + 30 g/day of sunflower oil	8 wk	T2DM	BW, BMI, WC	-
Małgorzata et al. (2020) [54]	Poland	I: 30 C: 30	45–65 years	-	Parallel—a randomised, double-blind	30 g/d Cold-pressed canola oil	30 g/d Of cold-pressed camelina oil	6 wk	Postmenopausal women with dyslipidemia	BW, BMI, WC, HC, WHR, fat mass	Bioimpedance method using a Tanita Body Fat Analyzer (model – BC 420 S MA with a medical certificate)
Salar et al. (2015) [67]	Iran	I: 24 C: 48	-	-	Parallel—a single-center; single blinded, randomized, controlled trial	Balance diet (55% carbohydrate, 18% protein and 27% fat) + 30 g CO/d	Balance diet (55% carbohydrate, 18% protein and 27% fat) + 30 g SO/d	8 wk	T2DM	BW	-

Table 3 (continued)

Author (yaer)	Country	Subjects	Age range or Mean SD	Mean of BMI at the baseline	Design	Intervention group	Comparator group	Duration (wk/d)	Participants	Outcomes	Outcomes measurement methods
Kruse et al. (2014) [53]	Germany	I: 9 C: 9	39–63	-	Parallel—A randomized controlled trial	50 g CO/d	50 g OO/d	4 wk	Moderate obesity	BW, BMI, body fat, WHR	Air-displacement plethysmography system
Nigam et al.[70]	India	I: 33 C: 60	20 to 50 years	-	A randomized, parallel, open-label design	Standard diet (15–21% protein (1–1.5 g/kg of desirable body weight), 55–70% carbohydrates, and 20% fats) + ≤ 20 g CO/d	1) Standard diet (15–21% protein (1–1.5 g/kg of desirable body weight), 55–70% carbohydrates, and 20% fats) + ≤ 20 g OO/d 2) Standard diet (15–21% protein (1–1.5 g/kg of desirable body weight), 55–70% carbohydrates, and 20% fats) + ≤ 20 g soybean and safflower oils/d	6 wk	NAFLD	BW, BMI, WC	-
Azemati et al. [68]	Iran	I: 20 C: 20	50.7 ± 6.1	-	Parallel- A randomized controlled trial	Normal diet + 40 g CO/d	Normal diet + 40 g SO/d	12 wk	Postmenopausal women with osteoporosis	BW, BMI, WC, HC, WHR	-
Iggman et al. [47]	Sweden	I: 20 C: 20	25–68 years	26.3	Randomized, controlled, two-period, cross-over intervention	Isocaloric diet (containing about 50% of energy from carbohydrates, 35% from fat and 14% from protein) based on CO	Isocaloric diet (containing about 50% of energy from carbohydrates, 35% from fat and 14% from protein) based on dairy fat (SFAs)	3 wk	Hyperlipidemia	BW, BMI	-

Table 3 (continued)

Liu et al. (2016) [14]	Multicenter	I: 101 C: 101	49.5 ± 1.2	29.4	A randomized, crossover, five-period, controlled feeding study	Weight-maintaining diet (50% of energy from carbohydrate, 35% of energy from fat (18% from treatment oils), and 15% of energy from protein) based on CO (18% of total calories)	1) Weight-maintaining diet (50% of energy from carbohydrate, 35% of energy from fat (18% from treatment oils), and 15% of energy from protein) based on corn and safflower oils (18% of total calories) 2) Weight-maintaining diet (50% of energy from carbohydrate, 35% of energy from fat (18% from treatment oils), and 15% of energy from protein) based on flax and safflower oils (18% of total calories)	4 wk	Subjects with central obesity	BW, fat mass, lean mass	DXA According to the manufacturer's recommendations (Lunar Prodigy Advance, Madison, WI; QDR-4500W; Hologic Corp, Waltham, MA)
Baxheinrich et al. [60]	Germany	I: 41 C: 40	-	-	Parallel- A randomized controlled trial	Hypoenergetic diet (Diets were calculated with 42% of total energy as carbohydrates, 20% as protein and 38% as fat. For both diets, targets for SFA were,10% and for MUFA 18% of energy) + 30 g CO/d	Hypoenergetic diet (Diets were calculated with 42% of total energy as carbohydrates, 20% as protein and 38% as fat. For both diets, targets for SFA were,10% and for MUFA 18% of energy) + 30 g OO/d	28 wk	Metabolic syndrome	BW, BMI, WC, body fat, lean mass	Bioelectric impedance analysis (Maltron International)

Table 3 (continued)

Author (yaer)	Country	Subjects	Age range or Mean SD	Mean of BMI at the baseline	Design	Intervention group	Comparator group	Duration (wk/d)	Participants	Outcomes	Outcomes measurement methods
Saedi et al.[71]	Iran	I: 52 C: 44	51.39 ± 12.85	27.71	Parallel—randomized controlled trial	CO as regular consumption	SO as regular consumption	24 wk	Hyperlipidemia	BW, BMI, WC, HC-	
Seppanen-Laakso et al. (1993) [61]	Finland	I: 23 C: 34	-	-	Parallel—randomized controlled trial	CO as water–oil emulsion (17 g/d)	1) OO as water–oil emulsion (19 g/d) 2) Breads containing margarine + butter	6 wk	Hyperlipidemia	BMI	-
Öhrvall et al. (2001) [55]	Sweden	I: 20 C: 20	50.9 ± 10	26.3	Randomized, double-blind, controlled crossover trial	Isocaloric diet based on CO	Isocaloric diet based on SFAs	3 wk	Healthy	BW, BMI, WHR	-
Jenkins et al. (2014) [54]	Canada	I: 70 C: 71	-	-	Parallel—randomized controlled trial	CO-enriched bread (4.5 slices: 31 g CO/d or 14% of total calories)	Whole-wheat bread without CO (7.5 slices/d)	12 wk	T2DM	BW, WC	-
Södergren et al. [48]	Sweden	I: 10 C: 9	50 ± 8	24.5	A randomised cross-over design	Diet based on CO	Diet based on SFAs	4 wk	Hyperlipidemia	BW	-
Kratz et al. (2002) [51]	Germany	I: 17 C: 38	25.7 ± 5.4	23	Parallel—randomized controlled trial	Diet based on CO	1) Diet based on SO 2) Diet based on OO	4 wk	Healthy	BW	-
Uusitupa et al. 1994 [44]	Finland	I: 10 C: 10	23 ± 1.6	21.5	Randomized crossover study design	Diet (40% fat, 45% carbohydrates, and 15% proteins) based on CO	Diet (40% fat, 45% carbohydrates, and 15% proteins) based on butter (SFAs) + small amount of CO	3 wk	Healthy	BW	-
Nydahl et al. (1995) [57]	Sweden	I: 22 C: 22	34.5- 69.2 years	-	Randomized crossover study design	Diet based on CO (32.9 ± 14.2 g CO/d)	Diet based on OO (32.9 ± 14.2 g OO/d)	3.5 wk	Hyperlipidemia	BW, BMI	-
Author (yaer)	**Country**	**Subjects**	**Age range or Mean SD**	**Mean of BMI at the baseline**	**Design**	**Intervention group**	**Comparator group**	**Duration (wk/d)**	**Participants**	**Outcomes**	**Outcomes measurement methods**

Table 3 (continued)

Chisholm et al. (2005) [59]	New Zealand	I: 28 C: 28	48.3 ± 10.3	26.9	Randomised cross over design	Low-fat diet (30–33% total energy from fat, 48–50% % total energy from carbohydrate, 16e18% total energy from protein) + cereal containing 15 g CO/d	Low-fat diet (30–33% total energy from fat, 48–50% % total energy from carbohydrate, 16e18% total energy from protein) + 30 g nuts/d	6 wk	Healthy	BW, BMI, WHR	-
Noroozi et a.(2009) [72]	Iran	I: 30 C: 30	39.7 ± 10.0	33.6	Parallel—randomized controlled trial	Low-calorie diet (containing 29% fats, 17% proteins and 54% carbohydrates) + 30 g CO/d	Low-calorie diet (containing 29% fats, 17% proteins and 54% carbohydrates)	4 wk	Hyperlipidemia	BW, BMI, WC, HC, WHR	-
Gustafsson et al. (1994) [62]	Sweden	I: 46 C: 49	-	-	Parallel—randomized controlled trial	Diet based on CO (≤ 30% of total calories)	Diet based on SO (≤ 30% of total calories)	3 wk	Hyperlipidemia	BW, BMI, WHR	-
Herrmann et al. (1997) [46]	Germany	I: 18 C: 35	53.9 ± 5.1	-	Parallel – a randomized, double-blind	Regular diet (45% complex carbohydrates, 25% protein, and 30% fat) + 12 g CO/d	Regular diet (45% complex carbohydrates, 25% protein, and 30% fat) + 12 g fish oil	4 wk	Coronary artery disease	BW	-
Wardlaw et al. [64]	United States	I: 16 C: 16	-	-	Parallel—randomized controlled trial	Diet based on CO (39 ± 1% of total calories)	Diet based on safflower oil (39 ± 1% of total calories)	8 wk	Healthy	BW	-
Bahareh Nikooyeh et.al. 2023 [75]	Iran	I:32 C: 30	57.6 ± 1.9	29.7	Parallel—randomized controlled trial	Diet based on CO	1)Diet based on enriched CO 2)Diet based on sunflower oil	12wk	T2DM	BMI,WC	-

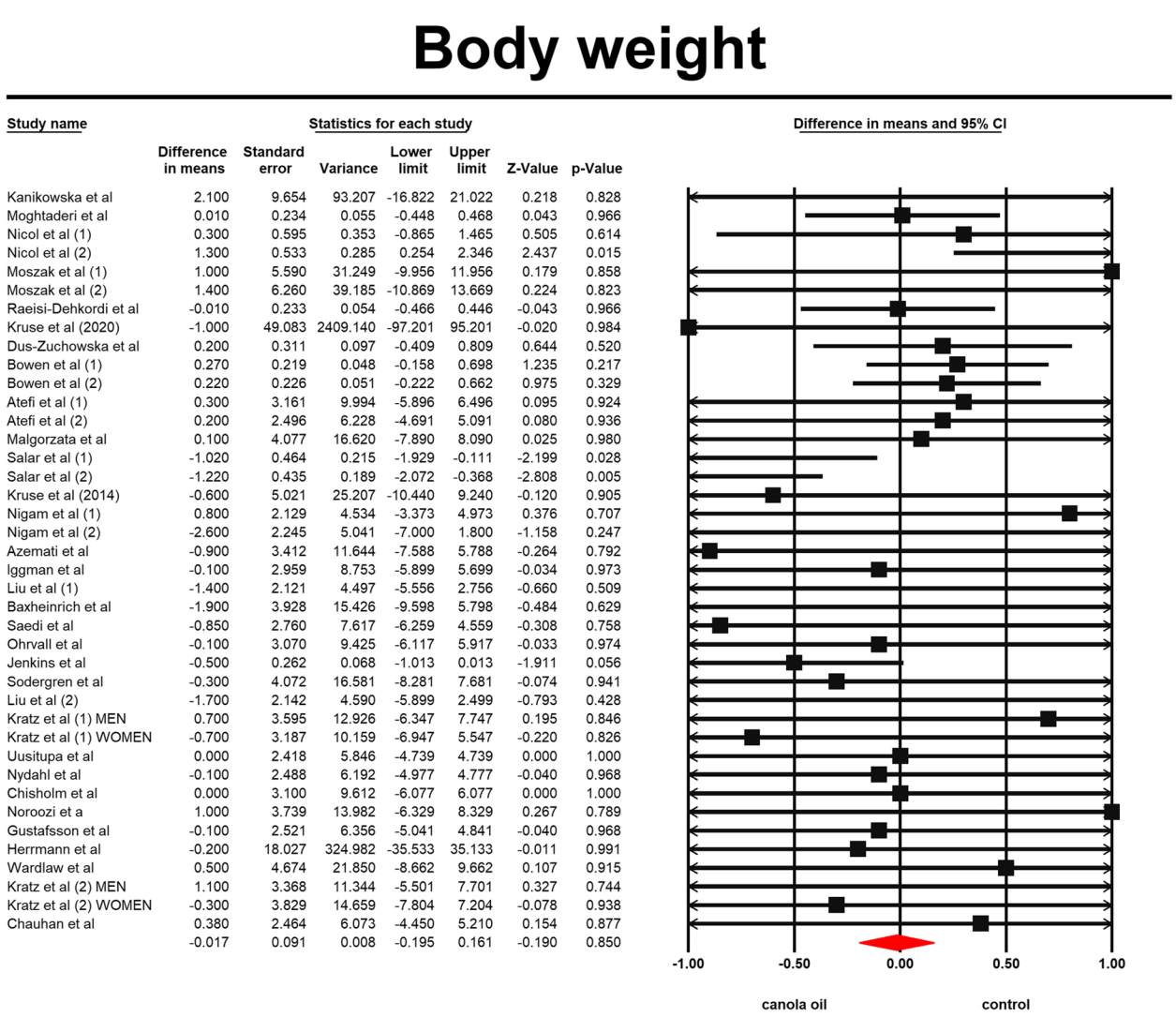

Fig. 2 The effect of CO consumption on BW

Results from subgroup analysis

Table 4 contains the subgroup analysis results. We classified the studies according to design, country, type of study population, age (year), type of intervention in the control group, duration (weeks), and canola oil dosage (g/d). The subgroup analysis showed that canola oil supplementation could significantly reduce BW in type 2 diabetes patients (WMD: −0.431 kg, 95% CI: −0.72, −0.13, *P* value: 0.005), parallel design studies (WMD: −0.4 kg, 95% CI: −0.75, −0.006, *P* value: 0.01), patients over 50 years old (WMD: −0.731 kg, 95% CI: −1.11, −0.34, *P* value < 0.001) and the use of canola oil with a dose of more than 30 g/d (WMD: −0.73 kg, 95% CI: −1.12, −0.34, *P* value < 0.001).

In addition, canola oil supplementation significantly increased WC only in parallel design studies (WMD: 0.65 cm, 955 CI: 0.07, 1.23, *P* value: 0.028), hyperlipidemia patients (WMD: 5.12 cm, 95% CI: 1.53, 8.7, *P* value: 0.005), no intervention of oil in the control group (WMD: 0.84 cm, 95% CI: 0.18, 1.51, *P* value: 0.013) and the use of canola oil with a dose of more than 30 g/d (WMD: 0.77 cm, 95% CI: 0.07, 1.47, *P* value: 0.03).

Moreover, the subgroup analysis related to the WHR variable showed that canola oil supplementation could significantly increase WHR only in cross-over design studies (WMD: 0.003 cm, 95% CI: 0.001, 0.005, P value: 0.004), Asian population(WMD: 0.003 cm, 95% CI: 0.001, 0.006, *P* value: 0.002), healthy population (WMD:

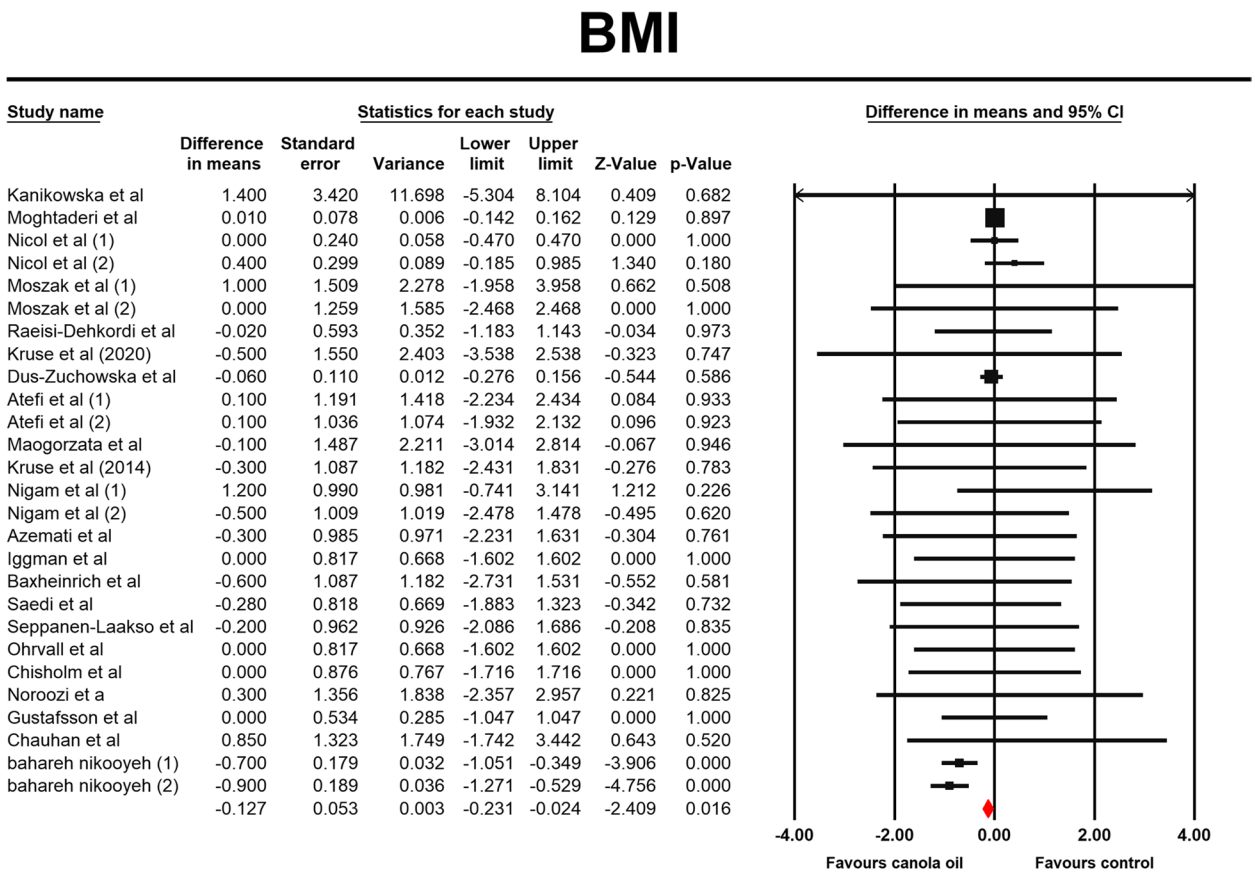

Fig. 3 The effect of CO consumption on BMI

0.003 cm, 95% CI: 0.000, 0.005, *P* value: 0.03), type 2 diabetes patients (WMD: 0.003 cm, 95% CI: 0.000, 0.006, *P* value: 0.04), postmenopausal patients (WMD: 0.26 cm, 95% CI: 0.04, 0.49, *P* value: 0.01), patients under 50 years of age (WMD: 0.003 cm, 95% CI: 0.001, 0.005, *P* value: 0.002) and studies with a duration of more than 8 weeks (WMD: 0.003 cm, 95% CI: 0.001, 0.006, *P* value: 0.002).

In addition, the subgroup analysis showed that canola oil supplementation could significantly reduce BMI only in parallel design (WMD: −0.41 kg/m^2, 95% CI: −0.98, −0.47, *P* value: < 0.001), T2DM patients (WMD: −0.73 kg/m^2, 95% CI: −0.6, −0.21, *P* value: < 0.001), patients over 50 years of age (WMD: −0.68 kg/m^2, 95% CI: −092, −0.45, *P* value: < 0.001) and intervention of sunflower oil in the control group (WMD: −0.4 kg/m^2, 95% CI: −066, −0.14, *P* value: 0.003).

No other significant effects of CO were seen in other anthropometric indices including: HC, VFM, FM, and LBM in subgroup analysis.

Publication bias

After applying the "trim and fill" method, some studies were added to account for potential missing data in the weight and body composition meta-analysis to adjust for publication bias. Table 5 summarizes the results of Begg's rank correlation, Egger's liner regression, "fail-safe N" tests, and correlated effect size.

Discussion

In the present study, we summarized and analyzed the results of RCTs investigating the effect of CO consumption on anthropometric measurements [15, 74, 76–81]. Based on our findings, CO supplementation could not significantly alter BW and WC but slightly increase WHR. In addition, no significant changes were seen in other anthropometric indicators including BMI, FM, HC, LBM, and VFM after supplementation with CO. The results of the current meta-analysis changed the previously published meta-analysis in 2018 [16]. We investigate nearly 650 more participants rather than the previous one [16]. In addition, the effect of CO

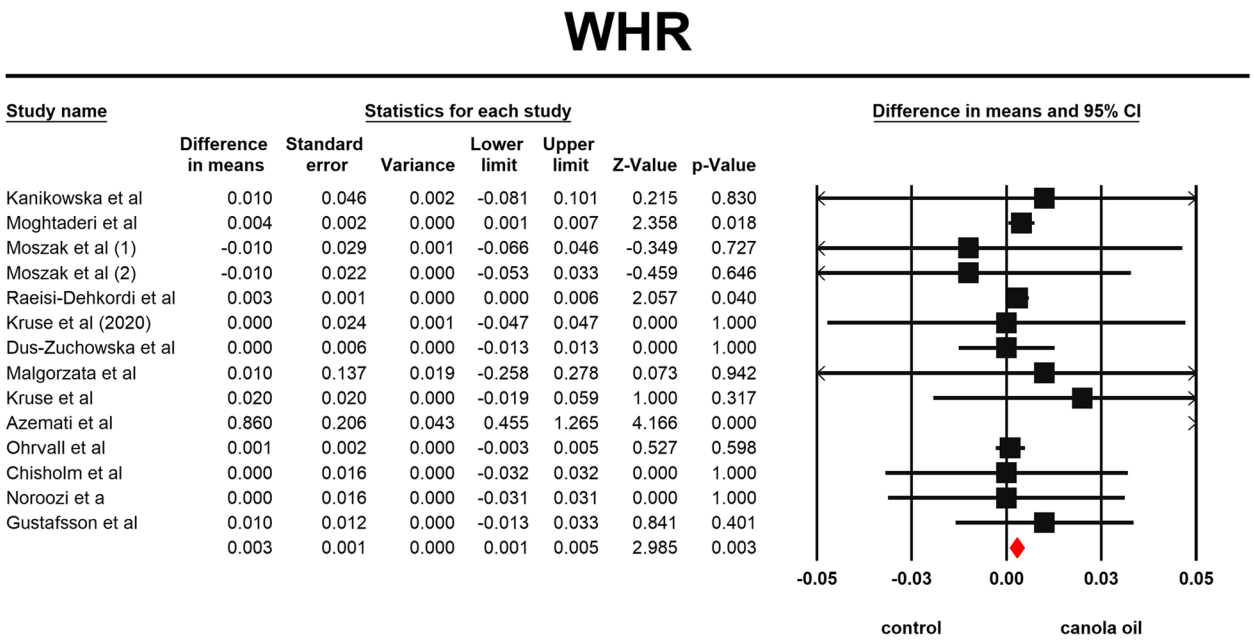

Fig. 4 The effect of CO consumption on WHR

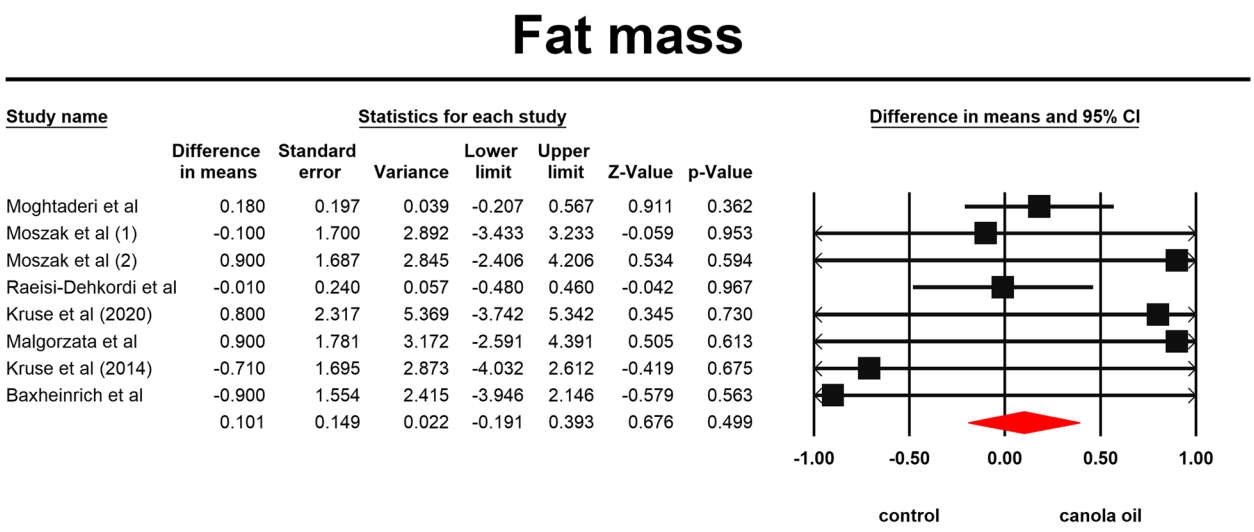

Fig. 5 The effect of CO consumption on Fat Mass

consumption on visceral fat mass was assayed for the first time in the present study.

Obesity is one of the most important health concerns worldwide [82]. Recently studies regarding the effects of nutritional supplementation for reducing or controlling obesity have been published [83–85]. In the present study, supplementation with CO did not significantly alter the BW. However, based on the result from the subgroup analysis, CO supplementation significantly decreased body weight in parallel design studies, diabetic patients, people more than 50 years old, and studies with consumption of more than 30 gr canola per day. Unlike

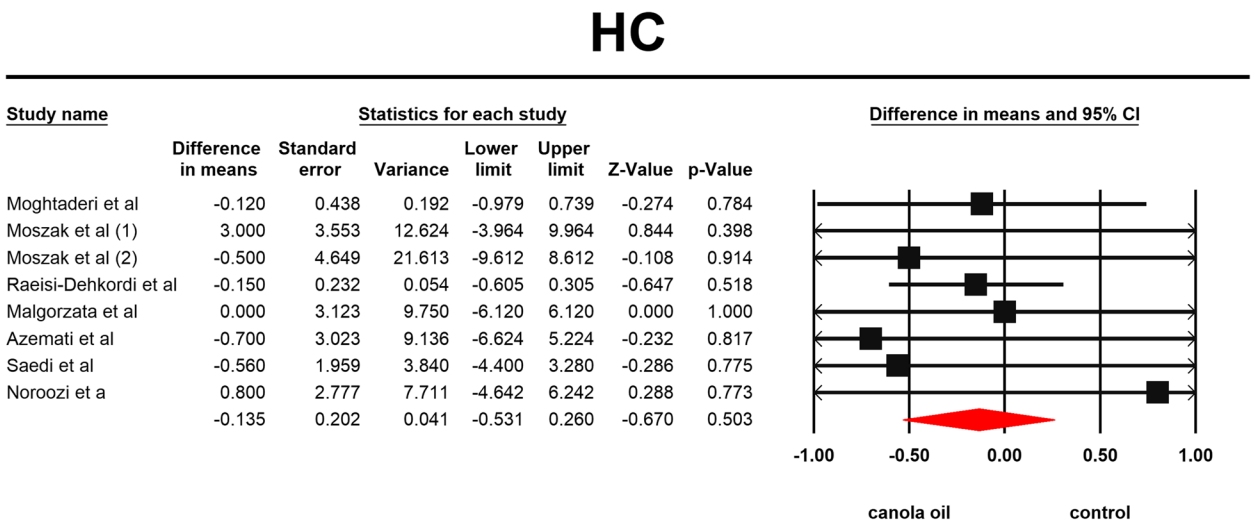

Fig. 6 The effect of CO consumption on HC

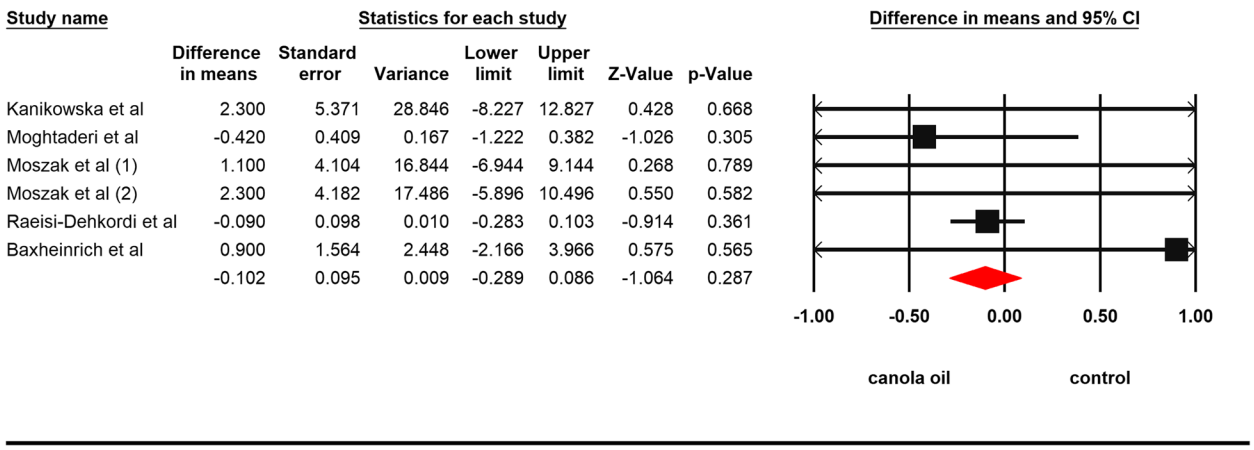

Fig. 7 The effect of CO consumption on LBM

our results, a previously published meta-analysis demonstrated that CO supplementation could decrease BW in all participants [16]. Based on our results, it seems there is a dose-dependent response to the consumption of CO. It seems that the weight loss effect of CO will appear in case of consumption of more than 30 g per day, in which we didn't see any significant effect from CO supplementation in people who consumed less than 30 g of CO per day. In addition, diabetic patients and older people (>50y) might take more advantage of supplementation with CO [86]. Based on evidence saturated fatty acids are more fattening compared to unsaturated fatty acids. The type of dietary fatty acids and the appropriate omega-3 to omega-6 ratio are also effective in the amount of fat deposition in the body [87]. It is noteworthy that CO is a rich source of essential unsaturated fatty acids such as omega-3 and −6 and also has a suitable ratio of omega-3 to omega-6 (1:2), which could explain its anti-obesity effects. In addition, special fatty acids such as MCTs (which are high in CO) could induce satiety more than long-chain fatty acids [88].

Our findings revealed no significant effect of CO on WC. However, subgroup analysis showed that CO supplementation significantly increased WC in studies with

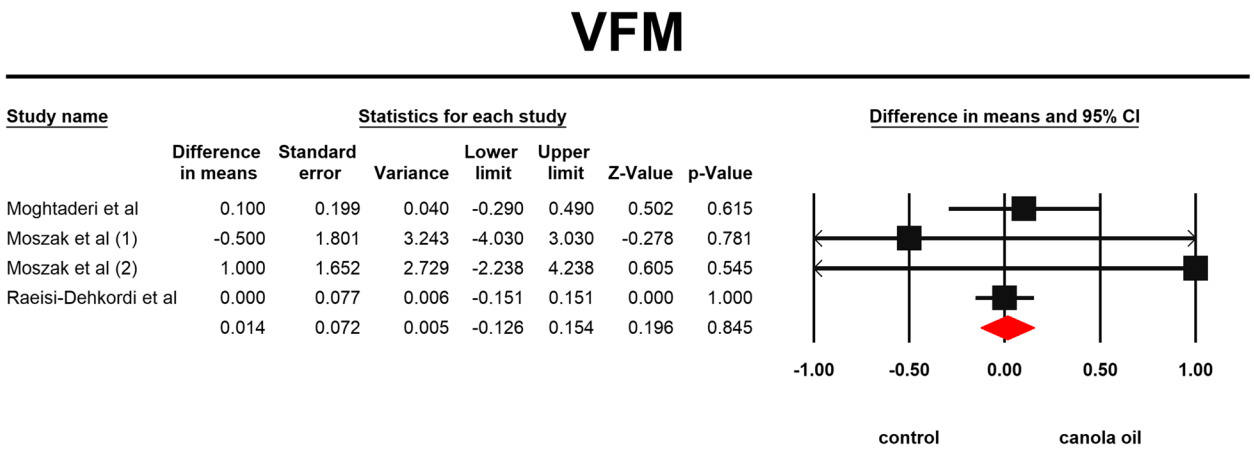

Fig. 8 The effect of CO consumption on VFM

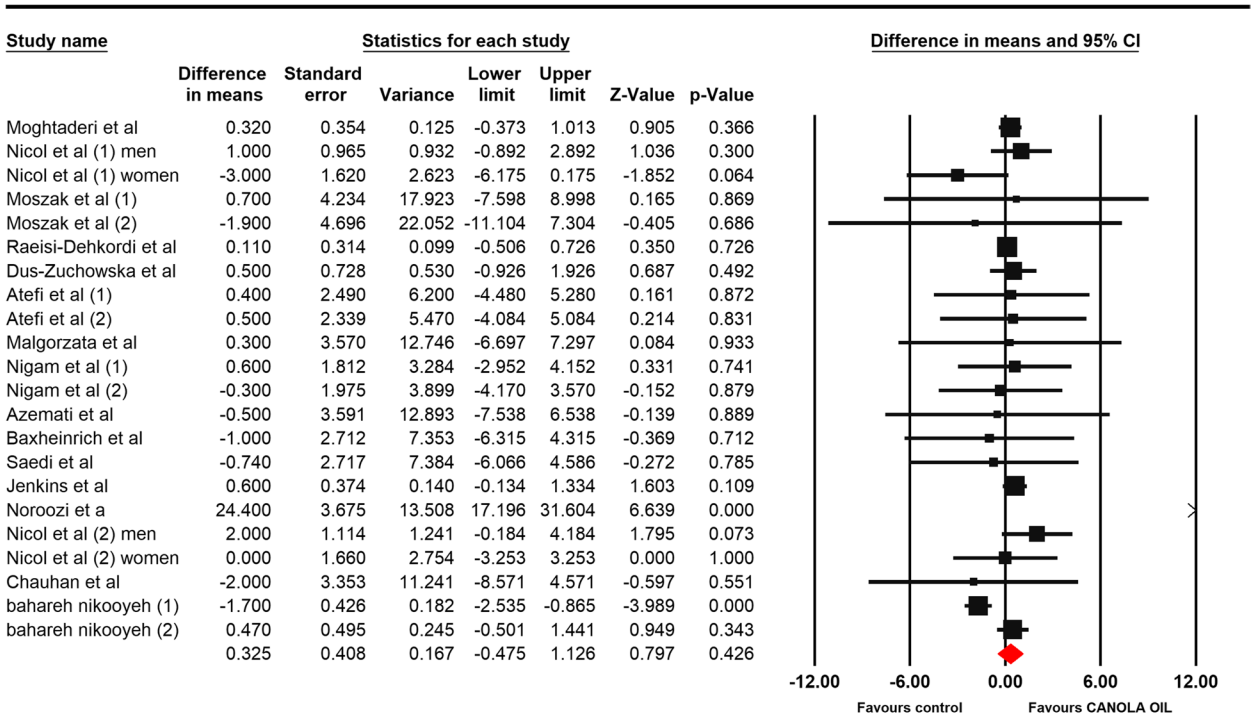

Fig. 9 The effect of CO consumption on WC

parallel design, hyperlipidemia patients, studies with no intervention of any oils in the control group, and intake of CO as the amount of more than 30 g/d. This finding followed the results from the previously published meta-analysis study [24]. Consistent with our result, CO oil had no significant effect on WC in people with dyslipidemia in another meta-analysis [89]. In addition, we found that supplementation with CO could slightly increase WHR. In the subgroup analysis, WHR also significantly increased after CO supplementation in studies

Table 4 Results of subgroup analysis of the included trials regarding the effects of canola oil on body weight and composition

	Number of comparison	WMD	CI 95%	*P* value	I^2 (%)	P-Heterogeneity	95% PI
Subgroup analyses for Body weight Outcome							
Study Design							
Parallel	27	−0.4	−0.75,−0.06	0.01	0	0.818	−0.75, - 0.04
Cross – over	13	0.127	−0.08,0.33	0.23	0	0.998	−0.11,0.35
Country							
Asian	12	−0.241	−0.52,0.04	0.09	5.96	0.387	−0.55, 0.04
Western	28	0.128	−0.1,0.3	0.27	0	0.988	−0.06, 0.3
Population							
Healthy	11	0.223	−0.16,0.61	0.26	0	0.882	−0.23,0.67
Obese	7	0.134	−0.46,0.72	0.65	0	0.969	−0.64, 0.9
T2DM	6	−0.431	−0.72,−0.13	0.005	40.21	0.137	−0.85, −0.005
Hyperlipidemia	7	−0.045	−2.15,2.06	0.967	0	1	−2.79, 2.71
NAFLD	3	−0.81	−3.83,2.21	0.6	0	0.547	−20.24, 18.64
Postmen opausa	2	−0.48	5.61,4.46	0.85	0	0.851	-
Metabolic syndrome	3	0.242	−0.06,0.55	0.122	0	0.851	−1.76, 2.24
Age (year)							
≤ 50	22	0.173	−0.02,0.37	0.09	0	0.988	−0.04, 0.38
> 50	17	−0.731	−1.11,−0.34	< 0.001	0	1	−1.15, −0.3
Control group							
Sunflower	8	−0.498	−1.18,0.18	0.15	0	0.854	−1.32, 0.34
Olive oil	8	0.161	−0.16,0.2	0.89	0	0.999	0.11, 0.2
With out oilintervention	11	−0.14	−0.59,0.3	0.53	0	0.49	−0.64, 0.36
Duration (week)							
8 >	25	0.21	−0.05,0.47	0.53	0	1	−0.06, 0.48
8 ≤	15	−0.19	−0.43,0.04	0.1	31.5	0.11	−0.44, 0.06
Dose (g/d)							
< 30	12	0.41	−0.05,0.88	0.08	0	0.91	−0.12, 0.94
≥ 30	12	−0.73	−1.12,−0.34	< 0.001	0	0.98	−1.17, −0.28
	Number of comparison	**WMD**	**CI 95%**	***P* value**	**I^2 (%)**	**P-Heterogeneity**	**95% PI**
Subgroup analyses for BMI Out come							
Study Design							
Parallel	19	0.1	−0.6, −0.21	< 0.001	0	0.99	−0.33, 0.53
Cross over	6	−0.01	−0.13,0.11	0.83	0	0.99	−0.43, 0.41
Country							
Asian	10	0.01	−0.13,0.16	0.85	0	0.98	−0.36, 0.38
Western	15	−0.009	−0.18,0.16	0.92	0	0.99	−0.36, 0.34
Population							
Healthy	5	0.03	−0.1,0.17	0.66	0	0.8	−0.47, 0.57
Obese	5	−0.05	−0.26,0.15	0.61	0	0.94	−0.6, 0.5
T2DM	3	0.02	−0.98, −0.47	< 0.001	0	0.99	−3.62, 3.66
Hyperlipidemia	6	0.002	−0.66,0.66	0.99	0	0.98	−1, 1.01
NAFLD	3	0.21	−1.04,1.47	0.73	0	0.42	−8.15, 8.57
Postmen opausa	2	−0.23	−1.84,1.37	0.77	0	0.91	-
Metabolic syndrome	1	−0.6	−2.73,1.53	0.58	0	1	-
Age (year)							
≤ 50	12	0.007	−0.1,0.12	0.9	0	0.96	−0.33, 0.34

Table 4 (continued)

	Number of comparison	WMD	CI 95%	*P* value	I^2 (%)	P-Heterogeneity	95% PI
> 50	12	−0.1	−0.92,−0.45	< 0.001	0	1	−0.6, 0.4
Control group							
Sunflower	5	−0.02	−0.66,−0.14	0.003	0	0.99	−0.51, 0.47
Olive oil	6	0.02	−0.86,0.9	0.96	0	0.84	−1.28, 1.32
With out oil intervention	8	0.25	−0.2,0.7	0.27	0	0.99	−0.4, 0.9
Duration (week)							
8 >	14	−0.04	−0.24,0.15	0.68	0	0.99	−0.41, 0.33
8 ≤	11	0.02	−0.11,0.16	0.71	0	0.98	−0.33, 0.37
Dose (g/d)							
< 30	11	0.009	−0.17,0.19	0.91	0	0.9	−0.37, 0.39
≥ 30	8	−0.17	−0.98,0.64	0.67	0	1	−1.23, 0.89
	Number of comparison	**WMD**	**CI 95%**	**P value**	**I^2 (%)**	**P-Heterogeneity**	**95% PI**
Subgroup analyses for Fat mass Out come							
Study Design							
Parallel	6	0.04	−1.35,1.44	0.94	9	0.94	−1.94, 2.02
Cross – over	2	0.1	−0.19,0.4	0.49	9	0.49	-
Country							
Asian	2	0.1	−0.19,0.4	0.49	0	0.49	-
Western	4	0.04	−1.35,1.44	0.94	0	0.94	−3.03, 3.11
Population							
Healthy	1	0.18	−0.2,0.56	0.96	0	1	-
Obese	3	0.03	−1.88,1.95	0.97	0	0.79	−12.41, 12.47
T2DM	1	−0.01	−0.48,0.46	0.96	-	1	-
Hyperlipidemia	-	-	-	-	0	-	-
NAFLD	-	0.8	−3.74,5.34	0.73	0	1	-
Postmen opausa	-	0.9	−2.59,4.39	0.61	0	1	-
Metabolic syndrome	1	−0.9	−3.94,2.14	0.56	0	1	-
Age (year)							
≤ 50	2	0.1	−0.19,0.4	0.49	0	0.54	-
> 50	6	0.04	−1.35,1.44	0.94	0	0.94	−1.94, 2.02
Control group							
Sunflower	-	-	-	-	-	-	-
Olive oil	3	−0.49	−2.5,1.51	0.62	0	0.82	−13.45, 12.47
With out oil intervention	1	0.9	−2.4,4.2	0.5	0	1	-
Duration (week)							
8 >	4	0.04	−1.67,1.76	0.95	0	0.86	−3.73, 3.81
8 ≤	4	0.1	−0.19,0.39	0.49	0	0.84	−0.53, 0.73
Dose (g/d)							
< 30	2	0.4	−1.94,2.75	0.73	0	0.67	-
≥ 30	4	−0.14	−1.89,1.59	0.86	0	0.87	−3.93, 3.65
	Number of comparison	**WMD**	**CI 95%**	**P value**	**I^2 (%)**	**P-Heterogeneity**	**95% PI**
Subgroup analyses for LBM Out come							
Study Design							
Parallel	4	1.15	−1.46,3.77	0.38	0	0.98	−4.6, 6.9

Table 4 (continued)

	Number of comparison	WMD	CI 95%	*P* value	I^2 (%)	P-Heterogeneity	95% PI
Cross – over	2	−0.1	−0.290.08	0.25	0	0.43	-
Country							
Asian	2	−0.1	−0.29,0.08	0.25	0	0.43	-
Western	4	0.15	−1.46,3.77	0.38	0	0.98	−7.79, 8.09
Population							
Healthy	1	−0.42	−1.22,0.38	0.3	0	1	-
Obese	3	1.82	−3.21,6.86	0.47	0	0.97	−30.85, 34.49
T2DM	1	−0.09	−0.28,0.1	0.36	0	1	-
Hyperlipidemia	-	-	-	-	-	-	-
NAFLD	-	-	-	-	-	-	-
Postmen opausa	-	-	-	-	-	-	-
Metabolic syndrome	1	0.9	−2.16,3.96	0.56	0	0.56	-
Age (year)							
≤ 50	3	−0.1	−0.29,0.08	0.26	0	0.66	−1.26, 1.06
> 50	3	1.07	−1.63,3.78	0.43	0	0.95	−16.49, 18.63
Control group							
Sunflower	-	-	-	-	-	-	-
Olive oil	1	0.9	−2.16,3.69	0.56	0	1	-
With out oil intervention	1	2.3	−5.86,10.49	0.58	0	1	-
Duration (week)							
8 >	3	1.82	−3.21,6.86	0.47	0	0.97	−30.85, 34.49
8 ≤	3	−0.1	−0.29,0.08	0.27	0	0.59	−1.26, 1.06
Dose (g/d)							
< 30	3	1.82	−3.21,6.86	0.47	0	0.97	−30.85, 34.49
≥ 30	1	0.9	−2.16,3.96	0.56	0	1	-
	Number of comparison	**WMD**	**CI 95%**	**P value**	**I^2 (%)**	**P-Heterogeneity**	**95% PI**
Subgroup analyses for WC Out come							
Study Design							
Parallel	17	0.65	−0.74,1.66	0.45	68.32	< 0.001	−0.63, 1.93
Cross – over	3	0.23	−0.2,0.66	0.3	0	0.84	−4.66, 5.12
Country							
Asian	10	0.28	−0.16,0.72	0.28	79.61	< 0.001	−0.61, 1.17
Western	10	0.55	−0.01,1.11	0.55	0	0.59	−0.43, 1.53
Population							
Healthy	5	0.38	−0.21,0.98	0.2	42.54	0.13	−1.02, 1.78
Obese	3	0.45	−0.93,1.84	0.52	0	0.87	−9.41, 10.31
T2DM	4	0.31	−0.15,0.78	0.18	0	0.79	−1.39, 2.01
Hyperlipidemia	3	5.12	1.53,8.7	0.005	94.54	< 0.001	−18.43, 28.67
NAFLD	2	0.18	−2.42,2.8	0.88	0	0.73	-
Postmen opausa	2	−0.09	−5.06,4.86	0.96	0	0.87	-
Metabolic syndrome	1	−1	−6.31,4.31	0.71	0	1	-
Age (year)							
≤ 50	10	0.34	−0.06,0.75	0.09	82.19	< 0.001	−0.53, 1.21
> 50	9	0.51	−0.17,1.2	0.14	0	0.99	−0.6, 1.62
Control group							
Sunflower	5	−0.06	−1.5,1.37	0.93	13.71	0.32	−2.59, 2.47

Table 4 (continued)

	Number of comparison	WMD	CI 95%	*P* value	I^2 (%)	P-Heterogeneity	95% PI
Olive oil	3	0.18	−2.34,2.71	0.88	0	0.88	−16.7, 17.06
With out oil intervention	7	0.84	0.18,1.51	0.013	86.43	< 0.001	−0.35, 2.03
Duration (week)							
8 >	7	1.04	−0.14,2.24	0.08	85.68	< 0.001	−0.73, 2.81
8 ≤	13	0.32	−0.04,0.68	0.08	0	0.7	−0.48, 1.12
Dose (g/d)							
< 30	10	0.45	−0.4,1.3	0.3	0	0.55	−0.78, 1.68
≥ 30	7	0.77	0.07,1.47	0.03	85.76	< 0.001	−0.45, 1.99
	Number of comparison	**WMD**	**CI 95%**	**P value**	**I^2 (%)**	**P-Heterogeneity**	**955 PI**
Subgroup analyses for WHR Out come							
Study Design							
Parallel	9	0.006	−0.008,0.02	0.41	57.41	0.01	−0.32, 0.34
Cross – over	5	0.003	0.001,0.005	0.004	0	0.8	−0.44, 0.45
Country							
Asian	4	0.003	0.001,0.006	0.002	82.82	0.001	−0.6, 0.61
Western	10	0.001	−0.002,0.005	0.51	0	0.99	−0.32, 0.32
Population							
Healthy	3	0.003	0.00,0.05	0.03	0	0.49	−1.79, 1.8
Obese	5	0.001	−0.01,0.01	0.9	0	0.85	−0.44, 0.45
T2DM	1	0.003	0.00,0.06	0.04	0	1	-
Hyperlipidemia	2	0.006	−0.01,0.02	0.5	0	0.61	-
NAFLD	1	0	−0.04,0.04	1	0	1	-
Post menopause	2	0.26	0.04,0.049	0.01	91.52	0.001	-
Metabolic syndrome	-	-	-	-	-	-	-
Age (year)							
≤ 50	7	0.003	0.001,0.005	0.002	0	0.98	−0.36, 0.36
> 50	7	0.001	−0.003,0.005	0.55	67.78	0.005	−0.36, 0.36
Control group							
Sunflower	2	0.01	−0.01,0.03	0.28	0.28	< 0.001	-
Olive oil	2	0.01	−0.01,0.04	0.44	0.44	0.52	-
With out oil intervention	4	0.001	−0.003,0.005	0.63	0.63	0.96	−0.6, 0.6
Duration (week)							
8 >	10	0.001	0.002,0.005	0.51	0	0.99	−0.32, 0.32
8 ≤	4	0.003	0.001,0.006	0.002	82.79	0.001	−0.6, 0.61
Dose (g/d)							
< 30	5	−0.001	−0.01,0.01	0.87	0	0.98	−0.45, 0.44
≥ 30	5	0.009	−0.01,0.03	0.43	77.47	0.001	−0.44, 0.46
	Number of comparison	**WMD**	**CI 95%**	**P value**	**I^2 (%)**	**P-Heterogeneity**	**95% PI**
Subgroup analyses for HC Out come							
Study Design							
Parallel	6	0.13	−2.16,2.42	0.91	0	0.96	−3.11, 3.37
Cross – over	2	−0.14	−0.54,0.25	0.48	0	0.95	-
Country							
Asian	5	−0.14	−0.54,0.25	0.47	0	0.99	−0.77, 0.49
Western	3	0.94	−3.16,5.04	0.65	0	0.77	−25.63, 27.51

Table 4 (continued)

	Number of comparison	WMD	CI 95%	*P* value	I^2 (%)	P-Heterogeneity	95% PI
Population							
Healthy	1	−0.12	−0.97,0.73	0.78	0	1	-
Obese	2	1.7	−3.82,7.24	0.54	0	0.55	-
T2DM	1	−0.15	−0.6,0.3	0.51	0	1	-
Hyperlipidemia	2	−0.1	−3.24,3.03	0.94	0	0.68	-
NAFLD	-	-	-	-	-	-	-
Post menopause	2	−0.36	4.61,3.89	0.86	0	0.87	-
Metabolic syndrome	-	-	-	-	0	-	-
Age (year)							
≤ 50	3	−0.13	−0.53,0.26	0.49	0	0.94	−2.65, 2.39
> 50	5	−0.01	−2.54,2.52	0.99	0	0.93	−4.11, 4.09
Control group							
Sunflower	2	−0.6	−3.82,2.62	0.71	0	0.96	-
Olive oil	-	-	-	-	-	-	-
With out oil intervention	-	−0.14	−3.11,2.81	0.92	0	0.92	-
Duration (week)							
8 >	4	0.89	−2.38,4.16	0.59	0	0.91	−6.28, 8.6
8 ≤	4	−0.15	−0.54,0.24	0.46	0	0.99	−1, 0.7
Dose (g/d)							
< 30	2	1.7	−3.82,7.24	0.54	0	0.55	-
≥ 30	3	0.07	−3.27,3.43	0.96	0	0.93	−21.71, 21.85
	Number of comparison	**WMD**	**CI 95%**	**P value**	**I^2 (%)**	**P-Heterogeneity**	**95% PI**
Subgroup analyses for VFM Out come							
Study Design							
Parallel	2	0.31	−2.07,2.7	0.79	0	0.53	-
Cross – over	2	0.01	−0.12,0.15	0.85	0	0.63	-
Country							
Asian	2	0.01	−0.12,0.15	0.85	0	0.63	-
Western	2	0.31	−2.07,2.7	0.79	0	0.53	-
Population							
Healthy	1	0.1	−2.07,0.49	0.61	0	1	-
Obese	2	0.31	−2.07,2.7	0.79	0	0.53	-
T2DM	1	0	−0.15,0.15	1	0	-	-
Hyperlipidemia	-	-	-	-	-	-	-
NAFLD	-	-	-	-	-	-	-
Postmen opausa	-	-	-	-	-	-	-
Metabolic syndrome	-	-	-	-	-	-	-
Age (year)							
≤ 50	2	2	−0.12,0.15	0.85	0	0.63	-
> 50	2	2	−2.07,2.7	0.79	0	0.53	-
Control group							
Sunflower	-	-	-	-	-	-	-
Olive oil	-	-	-	-	-	-	-
With out oil intervention	1	1	−2.23,4.23	0.54	0	1	-

Table 4 (continued)

	Number of comparison	WMD	CI 95%	*P* value	I^2 (%)	P-Heterogeneity	95% PI
Duration (week)							
8 >	2	0.31	−2.07,2.7	0.79	0	0.53	-
8 ≤	2	0.01	−0.12,0.15	0.85	0	0.63	-
Dose (g/d)							
< 30	2	0.31	−2.07,2.7	0.79	0	0.53	-
≥ 30	-	-	-	-	-	-	-

Table 5 Publication bias for anthropometric mesearments

	Corrected effect size		Begg's rank correlation test			Egger's liner regression test					Fail-safe N test
	WMD	95% CI	Kendall's Tau	z-value	*p*-value	Intercept	95% CI	t-value	df	*p*-value	n
BW	−0.09	−0.16,−0.03	−0.04	0.40	0.68	−0.07	−0.80,0.65	0.20	38	0.83	0
BMI	−0.15	−0.23,−0.06	−0.05	0.39	0.69	−0.26	−2.15,1.62	0.28	25	0.77	0
WHR	0.002	0.0009,0.0046	0.02	0.10	0.91	−0.19	−2.4,2.01	0.19	12	0.85	15
WC	0.98	0.12,1.84	−0.19	1.24	0.21	0.45	−0.76,1.66	0.77	20	0.44	0
VFM	0.02	−0.15,0.19	0	< 0.001	1.00	0.18	−3.89,4.25	0.19	2	0.86	0
LBM	−0.11	−0.26,0.02	0.13	0.37	0.70	1.60	0.01,3.18	2.8	4	0.04	0
HC	−0.02	−0.17,0.12	0.39	1.36	0.17	0.93	−0.31,2.18	1.83	6	0.11	0
FM	0.03	−0.11,0.19	−0.10	0.37	0.71	−0.13	−1.5,1.23	0.24	6	0.81	0

with cross-over design, Asian population, healthy population, type 2 diabetes patients, postmenopausal patients, patients under 50 years of age, and studies with a duration of more than 8 weeks. Although previous studies have shown that PUFA dietary source could alter fat distribution and improve metabolic risk factors [90], in some studies, for example, feeding a high-fat diet based on CO increased abdominal fat mass compared to the control group (receiving soybean oil and cornstarch) in rats [91]. In addition, another study showed that the consumption of oils containing omega-3 fatty acids could not significantly affect obesity-related risk factors [92]. Therefore the recommendation to consume CO should be taken with caution and attention. Maybe some other factors such as total dietary fat and the amount of CO consumption alter the effect. Because of the important effect of visceral fat on health issues, more RCTs are needed to investigate the accurate effect of CO on abdominal obesity.

This meta-analysis revealed that the CO supplementation did not significantly alter BMI, HC, VFM, FM, and LBM. Also, subgroup analysis showed no significant effect. It must be kept in mind that the amount of CO consumption is an important factor in achieving the desired results. For example, the consumption of 12.5 g of MCT (155 cal) in breakfast compared to intake up to > 20% of total daily energy (54 g of MCT daily or ~ 18 g per meal) did not show significant changes in body composition [93]. The health condition of participants also could affect the impact of CO consumption on body composition [93]. For example, the difference in BMI greatly affects the amount of oxidation and synthesis of fat in body tissues, especially the liver [94].

Our study has some strengths and limitations. We did a systematic review and meta-analysis on a large number of clinical trials in which the effects of CO consumption on various anthropometric measurements were investigated. In addition, the subgroup analysis was done based on various anthropometric variables to detect the accurate effect of CO in participants. We also did a subgroup analysis based on a large number of variables. To cover all relevant literature, a complete search was conducted across 4 databases (PubMed, ISI Web of Science, SCOPUS, Google Scholar) using PRISMA guidelines. In addition, the reference lists of the related reviews were searched. Standard methodologies were utilized to assess kappa statistics between the authors, heterogeneity, sensitivity analysis, and publication bias. There was perfect agreement in study selection between the reviewers. Also, the reviewers had substantial agreement regarding data extraction and quality assessment. In addition, the GRADE evidence profiles were applied to assess the total

quality of evidence related to the effect of canola oil on body composition. However, some limitations should be considered when our results interfere. The first limitation is the high between-study heterogeneity. Therefore, the interpretation of our findings should be done cautiously. We did a subgroup analysis to find the possible sources of heterogeneity. However, in some cases, these analyses were not able to resolve this problem. Second, included participants had different health conditions which further highlights the need for caution in the interpretation. We did a subgroup analysis to seek the precise effect of CO on anthropometric indicators in different conditions. Third, it must be kept in mind that some studies have evaluated the anthropometric index as a secondary outcome which could be different from studies that have investigated these indicators as a primary outcome.

It is suggested to conduct more RCTs with larger sample sizes and longer durations of intervention regarding the effect of canola oil on body composition in the future. Furthermore, it is suggested that more studies be conducted on the mechanisms regarding the effect of canola oil on body composition in the future.

Conclusion

Compared to other oil supplementation, CO could decrease BW, BMI and increase WHR, and WC in general or subgroup analysis. Further studies are needed to provide additional insight into how canola oil affects BW and composition in adults.

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Authors' contributions
A.M. conceived the study. A.M. and A.A. wrote the proposal. A.M. carried out the literature search. A.M. and F.B. carried out the literature screening. A.M. and F.B. carried out data extraction and independent reviewing. A.M. and F.B. conducted the quality evaluation of the included studies. A.M. conducted data analysis and interpretation. A.M. H.B. and M.M. wrote the manuscript. A.M. and H.B. conducted the critical revision of the manuscript. All authors read the manuscript and approved it.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
Not applicable.

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Competing interests
The authors declare no competing interests.

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