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ORIGINAL RESEARCH

Effect of Different Types of Whole Dietary Pulses on Established Therapeutic Lipid Targets for Cardiovascular Risk Reduction: An Updated Systematic Review and Dose–Response Meta-Analysis of Randomized Controlled Trials

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BACKGROUND: Although dietary pulses are recognized by major clinical practice guidelines to reduce cholesterol and coronary heart disease risk, intake is low. There are no health claims for any pulse for cholesterol reduction, which could support uptake. We therefore conducted a systematic review and dose–response meta-analysis of randomized trials of the effect of different types of whole dietary pulses on lipid targets.

METHODS: MEDLINE, Embase, and the Cochrane Library were searched through March 2025 for trials ≥3 weeks. The primary outcome was low-density lipoprotein-cholesterol. Secondary outcomes were other lipid targets. Independent reviewers extracted data and assessed risk of bias. Certainty of evidence was assessed using Grading of Recommendations Assessment, Development, and Evaluation.

RESULTS: Thirty-eight trials (52 trial comparisons, n=2095) with a median of 6 weeks and dose of 130 g/d (0.5–0.67 cup/d) showed that whole dietary pulses decreased low-density lipoprotein-cholesterol (mean difference, −0.14 mmol/L [95% CI, −0.19 to −0.08]), non-high-density lipoprotein-cholesterol (−0.22 mmol/L [95% CI, −0.30 to −0.14]), apoB (apolipoprotein B) (−0.08 g/L [95% CI, −0.13 to −0.03]) and high-density lipoprotein-cholesterol (−0.03 mmol/L [95% CI, −0.05 to −0.01]) with no effects on other lipids. Analyses by pulse type showed similar results. A linear inverse relationship was shown for beans up to 1 cup/d for low-density lipoprotein-cholesterol (coefficient, −0.25 mmol/L/0.5 cup [95% CI, −0.48 to −0.02]) and non-high-density lipoprotein-cholesterol (−0.45 mmol/L/0.5 cup [95% CI, −0.71 to −0.18]). Grading of Recommendations Assessment, Development, and Evaluation was moderate-to-high for all outcomes, except apoB (very low).

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CONCLUSIONS: Whole dietary pulses likely result in small important-to-moderate reductions in lipid targets and trivial reductions in high-density lipoprotein-cholesterol. Similar effects were observed across pulse types with an inverse dose–response gradient for beans up to 1 cup/d. Future studies on chickpeas, dried peas, and lentils are warranted.

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Key Words: blood lipids ■ cardiovascular ■ clinical trials ■ dietary pulses ■ plant protein ■ protein ■ systematic review meta-analysis

CLINICAL PERSPECTIVE

What Is New?

- Whole dietary pulses reduce low-density lipoprotein cholesterol, non-high-density lipoprotein cholesterol, and apolipoprotein B with moderate certainty of evidence and with similar effects observed across pulse types in an updated systematic review and meta-analysis of randomized controlled trials examining differences across pulse types.
- Beans result in an inverse dose response up to 1 cup serving per day for low-density lipoprotein cholesterol and non-high-density lipoprotein cholesterol.

What Are the Clinical Implications?

- Our findings highlight the beneficial effects of dietary pulses on therapeutic lipid targets and support health claims, public health messages, and translation of clinical practice guidelines to encourage the consumption of dietary pulses to reduce cardiovascular risk.

Nonstandard Abbreviations and Acronyms

GRADE	Grading of Recommendations Assessment, Development, and Evaluation
MD	mean difference
MID	minimally important difference
ROB	risk of bias

Cardiovascular disease (CVD) continues to be a leading cause of death and a burden to health care systems in Canada and globally.[1,2] Low-density lipoprotein-cholesterol (LDL-C) is causal in the pathogenesis of atherosclerotic cardiovascular disease and thus, the primary risk factor for CVD.[3–5] However, individuals at high risk of CVD often do not achieve lipid targets, due to insufficient lowering with statins, statin-related side effects, poor medication adherence, and treatment inertia.[6,7] Major clinical practice guidelines for nutrition therapy[8–11] recommend dietary patterns with an emphasis on plant-based foods and plant-based protein sources for cardiovascular risk reduction. Plant-based protein sources include soy, dietary pulses, nuts, and seeds. Soy protein (25 g/d) and nuts (42 g/d or 1.5 oz/d) have health claims for cholesterol and coronary heart disease risk reduction,[12–15] whereas pulses do not. Food labeling can influence consumer's dietary choices and is a key strategy to support the consumption of nutrients, including those in alignment with dietary guidelines.[16] The global consumption of pulses is comparatively low, at 21 g per capita per day, without significant change over the past 3 decades, according to the Food and Agriculture Organization.[17] Intake of dietary pulses could benefit from a health claim if warranted.

Pulses are nonoil seeds in the legume family, such as dry beans, dry peas, chickpeas, and lentils. Traditional sources of pulse proteins, such as whole cooked pulses, have been associated with cardiovascular benefit.[18,19] There is some evidence that categories of dietary pulses may differ in effect on lipids.[20] A previous systematic review and meta-analysis of randomized controlled trials demonstrated a significant 0.17 mmol/L reduction in LDL-C with a median of 130 g, or ½ cup, of pulses per day compared with a non-pulse-containing control.[19] Since the publication of this meta-analysis over 10 years ago, there has been an increased focus on plant-based dietary patterns in dietary guidelines and thus an increased interest in the effect of plant-based foods, resulting in an emergence of randomized controlled trials investigating the potential cardiovascular benefits of various dietary pulses. To support the development of cardiovascular guidelines and health claims, we conducted a systematic review and meta-analysis of randomized controlled trials to investigate the effect of different pulses on established lipid targets for cardiovascular risk reduction and metabolic syndrome[8,21] with an assessment of the certainty of evidence using Grading of Recommendations, Assessment, Development, and Evaluation (GRADE).

METHODS

We followed the *Cochrane Handbook for Systematic Reviews of Interventions*[22] and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses 2020 (Table S1)[23] and registered the study protocol

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on PROSPERO (International Prospective Register of Systematic Reviews; CRD42023432826). There were no amendments after the initial registration of our protocol. The data used in this article are publicly available and the analytical code can be made available upon request. As this study is using publicly available data, it is exempt from ethical review.

Data Sources and Search Strategy

We conducted a systematic search in MEDLINE, Embase, and the Cochrane Central Register of Controlled Trials databases from inception through March 26, 2025. Our database search was supplemented with manual searches of references in the included trials. We did not have any language restrictions. Tables S2 and S3 present the search strategy.

Study Selection

We included randomized controlled trials in adults of all health backgrounds (including participants with severe CVD or end-stage renal disease, where available) with intervention periods ≥3 weeks investigating the effect of whole pulse consumption compared with a non-pulse-containing control on LDL-C and other lipids, including non-high-density lipoprotein-cholesterol (non-HDL-C), apoB (apolipoprotein B), HDL-C, and triglycerides. Whole pulses included intact whole pulses or pulse-based products that included all pulse components such as flours. Interventions including only peanuts and soybeans were excluded as they are in the broader family of legumes but not dietary pulses. Dry peas are typically consumed as split peas where the hull is removed during premarket processing and were considered to be whole pulses in the context of this study and how dry peas are traditionally consumed by humans. We required the follow-up period to be ≥3 weeks, a duration that aligns with the US Food and Drug Administration framework for the scientific evaluation of lipid-lowering health claims.[24] Randomized trials in pregnant women or children were excluded.

To determine eligibility, reports were first excluded based on a review of their titles and abstracts by 1 reviewer (S.B., S.Y., V.C., S.A.C., E.J.L., S.M.G., or Y.T.C.), followed by full text review of those reports that remained by at least 2 reviewers (S.B., S.Y., V.C., S.A.C., E.J.L., S.M.G., and Y.T.C.). Reviewer discrepancies were resolved by consensus or arbitration by the senior investigator (L.C.). In reports containing >1 eligible trial comparison, we included each available trial comparison separately.

Data Collection and Quality Assessment

Data were extracted using a standardized electronic form by at least 2 reviewers independently (S.Y., S.B., V.C., S.A.C., E.J.L., S.M.G., and Y.T.C.). Information included the pulse source (beans, lentils, chickpeas, dried peas, and legume intervention), the number of participants, the participant health status, sex, gender, and mean age, the study design, the energy balance (relative to the background diet), the energy level (intervention relative to the control), setting, comparator, the feeding control, processing (eg, canning, milling), the food form, the macronutrient profile of the diets, the saturated fat and fiber content of the intervention and control groups, the follow-up duration, cholesterol-lowering medication use, the funding source, and the outcome data. All lipid data that were available in publications that indicated lipid outcomes were measured; thus, no authors were contacted for missing outcome data. Graphically presented data were extracted from figures using Plot Digitizer.[25] Reviewer discrepancies in data extractions were resolved by consensus or arbitration by a senior investigator (L.C.).

Risk of Bias Assessment

Each included study was assessed for risk of bias (ROB) by 2 investigators using the Cochrane Risk of Bias V.2.0 tool.[26] The assessment was performed across 6 domains of bias (randomization process, deviations from intended interventions, missing outcome data, measurement of the outcome, selection of the reported result, and overall bias). Crossover studies were assessed for an additional domain of ROB arising from period or carryover effects. Each domain was assessed as "low" (a plausible bias unlikely to seriously alter the results), "high" (a plausible bias that seriously weakens confidence in results), or "some concern" (a plausible bias that raises some doubt about the results). An overall ROB was determined based on judgments from each domain. Reviewer discrepancies were resolved by consensus or arbitration by the senior investigator (L.C.).

Outcomes

The primary outcome was LDL-C, as the primary lipid target for CVD.[8] Secondary outcomes included other established lipid targets for CVD (non-HDL-C and apoB) and metabolic syndrome (HDL-C and triglycerides).[21] Mean differences (MDs) between the intervention and control arm and their SEs were extracted for each eligible trial comparison. If unavailable, they were derived from the available data using published formulas.[22] We used mean pairwise differences in change-from-baseline values over end values, when available. When median data were provided, they were converted to mean data with corresponding variances using methods developed by Luo et al.[27] and Wan et al.[28] When no variance data were available, the SD was borrowed from a trial with a similar size, number

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of participants, and nature of the intervention, including the food source and dose.[29] When an outcome was not reported, but the variables to calculate that variable were, the outcome was calculated using a standard formula. In studies that reported both the total cholesterol and HDL-C levels, we calculated non-HDL-C using the difference between the means and the inverse variance law for SDs.[30]

Statistical Analysis

STATA version 17 (StataCorp) was used for all analyses. As our primary research question was to assess the effect of different types of whole dietary pulses, we assessed the interaction (variation in the effect estimates) by pulse type using the *Cochrane Handbook* recommended standard Q test for subgroup differences using meta-regression (significance at $P<0.10$).[22] In the absence of interaction, we also assessed the influence of the pulse type. If 1 pulse type had >30% of the weight in a pooled analysis, we determined the pulse type to have a disproportionate influence on the analysis. In this scenario, there is an important influence because we cannot draw conclusions regarding the effect of whole dietary pulses.

We used the mean pairwise differences in the change from baseline (or end difference) between the pulse group and the non-pulse-containing control group as principal effect measurements (significance at $P<0.05$). We expressed results as the MDs with 95% CIs and used the generic inverse variance method with the DerSimonian and Laird random-effects model for data analyses.[31,22] We used a fixed effects model when the number of trials was ≤5.[32–34] Paired analyses were applied to all crossover trials using a within-individual correlation coefficient between the treatments of 0.5 to calculate the SEs.[35–37] When the arm in a trial with multiple intervention or control arms was used more than once, the corresponding sample size was divided by the number of times it was used for the calculation of the SE of the pooled effect in order to mitigate a unit-of-analysis error.[38]

Heterogeneity was assessed and quantified using the Cochran Q statistic and the I^2 statistic, where $I^2 \geq 50\%$ and $P_Q<0.10$ represent substantial heterogeneity.[39,22] Sources of heterogeneity were explored by sensitivity analyses, including individual trial influence, alteration of the pairwise comparison correlation coefficient, and subgroup analyses. The individual trial influence analysis involved systematically removing each trial comparison from the meta-analysis and recalculating the summary effect estimate. A trial whose removal explained the heterogeneity or changed the significance, direction, or magnitude of the effect by more than the minimally important difference (MID) for each outcome (prespecified as 0.1 mmol/L [5%] for LDL-C, non-HDL-C, HDL-C, triglycerides, and 0.04 g/L for apoB) was considered an influential trial.[12,40–42] To determine whether the overall results were robust to the use of different meta-analysis methods (random versus fixed effects) and to different correlation coefficients in crossover trials, we conducted sensitivity analyses using fixed effects (for outcomes with >5 trials) and using correlation coefficients of 0.25 and 0.75, respectively. When ≥10 trials were available, we explored sources of between-study heterogeneity using random effects meta-regression (significance at $P<0.05$)[43,44] with the following moderators one at a time: pulse type, the dose of dietary pulse, pulse protein energy, pulse fiber energy (g/1000 kcal), the pulse processing method (dried, canned, made into foods, baked good), the participant health status, sex, gender, and age, cholesterol-lowering medication use, the baseline outcome, the comparator, the duration of follow-up (≤12-weeks, >12-weeks), the study design (crossover, parallel), the energy balance of the intervention relative to the basal diet (positive, neutral, or negative), energy level (substitution, addition), the feeding control (metabolic, supplemented, dietary advice, or ad libitum), the difference in saturated fat and fiber content between the intervention and control, the type of MD (change from baseline, end differences), funding, and the ROB domains. We used meta-regression analyses to assess the significance of each subgroup categorically and continuously (where applicable). When ≥6 trials were available, we used generalized least squares trend estimation models and spline curve modeling (MKSPLINE procedure) to assess linear and nonlinear dose–response relationships. These models assume normally distributed errors and a smooth, continuous dose–response function across spline segments. Knots for the spline curve modeling were placed, following Harroll's principles,[45] at the 15th, 50th, and 95th percentiles of the observed distribution doses. This approach allows for a flexible nonlinear association while maintaining linearity in the tails. For studies contributing multiple dose comparisons, the models account for the correlation between dose levels within the same study, while treating estimates from different studies as independent. When ≥10 trials were available, we assessed for the presence of small-study effects (publication bias) by visual inspection of contour-enhanced funnel plots and formal testing with Egger's and Begg's tests (significance at $P<0.10$ due to the limited power of such tests).[46–48] If there was evidence of small-study effects, then we quantified the size of the potential publication bias or other causes of asymmetry by adjusting for the funnel plot asymmetry and assessing for small-study effects using the trim-and-fill method of Duval and Tweedie.[49] Although the Egger's and Begg's tests and the trim-and-fill methods have limited power and high noise when few trials are

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included, these formal testing approaches were supported by visual inspection of funnel plots and are recommended in the *Cochrane Handbook*.[22]

Subgroups analyses have not been corrected for multiple testing and should be interpreted as exploratory.

Certainty of Evidence

We assessed certainty of evidence using the GRADE approach and software (GRADEpro GDT, McMaster University, and Evidence Prime).[50] Two independent reviewers (S.B., S.Y.) conducted GRADE assessments with any discrepancies resolved by consensus or arbitration by the senior author (L.C.). The evidence was rated as having high, moderate, low, or very low certainty. Initially, the randomized controlled trials were rated as high certainty by default and then downgraded or upgraded based on prespecified criteria. Reasons for downgrading the evidence included ROB (assessed using the Cochrane ROB Tool), inconsistency (substantial unexplained interstudy heterogeneity defined as I^2 ≥50% and P_Q<0.10), indirectness (the presence of factors that limit the generalizability of the results), imprecision (the 95% CI for effect estimates overlap the MID for benefit or harm or lack of robustness from sensitivity analyses), and publication bias (significant evidence of small-study effects). Reason for upgrading the evidence included the presence of a significant dose–response gradient supporting the direction of the pooled effect estimate.[51–56] We assessed the importance of the magnitude of the pooled estimates using our prespecified MIDs and the effect size categories according to the GRADE guidance[57–59] as follows: a large effect (≥5× MID); moderate effect (≥2× MID); small important effect (≥1× MID); and trivial/unimportant effect (<1 MID).

RESULTS

Search Results

Figure 1 outlines our systematic search results and Table S4 presents the full list of studies retrieved from the search and reasons for exclusion at each stage of review. We identified 4934 reports from our systematic search, 4599 of which were excluded based on the title or abstract. Of the 335 reports reviewed in full, 38 trials met our eligibility criteria. The 38 trials provided data on 52 trial comparisons (49 trial comparisons on LDL-C, 51 on non-HDL-C, 52 on HDL-C; 49 on triglycerides, and 3 on apoB) involving 2095 participants.[60–97]

Trial Characteristics

Table and Table S5 describe the characteristics of the included trials. The trial size had a median of 23 to 28 participants (ranging from 5 or 6 to 150) for trials of LDL-C, non-HDL-C, HDL-C and triglycerides; a median of 25 participants (ranging from 20 to 114) for trials of apoB. Participants included adults with or without type 2 diabetes or hypercholesterolemia. There were approximately equal ratios of male and female adults. Participants had a median age of 52 years (ranging from 21 to 64 years) for LDL-C, non-HDL-C, HDL-C and triglycerides and a median age of 37 years (ranging from 26 to 48 years) for apoB. Most trials were conducted in outpatient settings (88–100%) with the majority performed in the United States (16) and Canada (13), followed by Iran (7), Australia (6), New Zealand (2), Mexico (2), United Kingdom (2), Pakistan (2), Singapore (1), and South Africa (1). Most of the trials followed a crossover study design (53%–67%), and the feeding control was mainly supplemented (56%–59%), followed by metabolic (22%–27%), dietary advice (14%–16%) and a mix of metabolic and dietary advice (2%) in trials of LDL-C, non-HDL-C, HDL-C, and triglycerides. All studies of apoB were supplemented. The median dose of whole dietary pulses was 130 g/d (ranging from 24 to 377 g/d) for LDL-C, non-HDL-C, HDL-C, and triglycerides and 90 g/day (90–253 g/d) for apoB. The types of pulses included in trials were predominantly beans (54%–57% in LDL-C, non-HDL-C, HDL-C, and triglycerides and 66% in apoB) and mixed sources (22%–27% in LDL-C, non-HDL-C, HDL-C, and triglycerides and 33% in apoB), with few studies in lentils (6%), chickpeas (2%), dried peas (4%), and a mix of legumes (soy and dietary pulses) (8%) in LDL-C, non-HDL-C, HDL-C, and triglycerides. For trials in LDL-C, non-HDL-C, HDL-C, and triglycerides, dietary pulses were provided as whole, canned pulses (47%–51%), followed by whole, canned, flavored pulses (14%), extracted, made into foods, and mixed processing (12%–18%) and few baked goods and whole dried (2%–6%). For trials of apoB, dietary pulses were provided as whole, baked into goods, extracted, made into foods, or mixed processing. The comparators in trials of LDL-C, non-HDL-C, HDL-C, and triglycerides were mainly usual diet (37%–39%) or carbohydrates (27%–29%), followed by animal protein and vegetables (8%–10%), another dietary pattern (Dietary Approaches to Stop Hypertension, high glycemic index, low carbohydrates) or other or mixed comparators (2%–6%). All trials of apoB compared the pulse intervention to usual diet. Most trials indicated that the delivery form and caloric contribution of the control matched that of the intervention (98% in LDL-C, non-HDL-C, HDL-C, and triglycerides; 66% in apoB). The median follow-up duration was 6 weeks (ranging from 3 to 52 weeks) for LDL-C, non-HDL-C, HDL-C, and triglycerides, and 6 weeks (ranging from 4 to 16 weeks) for apoB. Trials were funded by a mix of agency and industry sources (44%–47% in LDL-C, non-HDL-C, HDL-C, and triglycerides; 33% in apoB),

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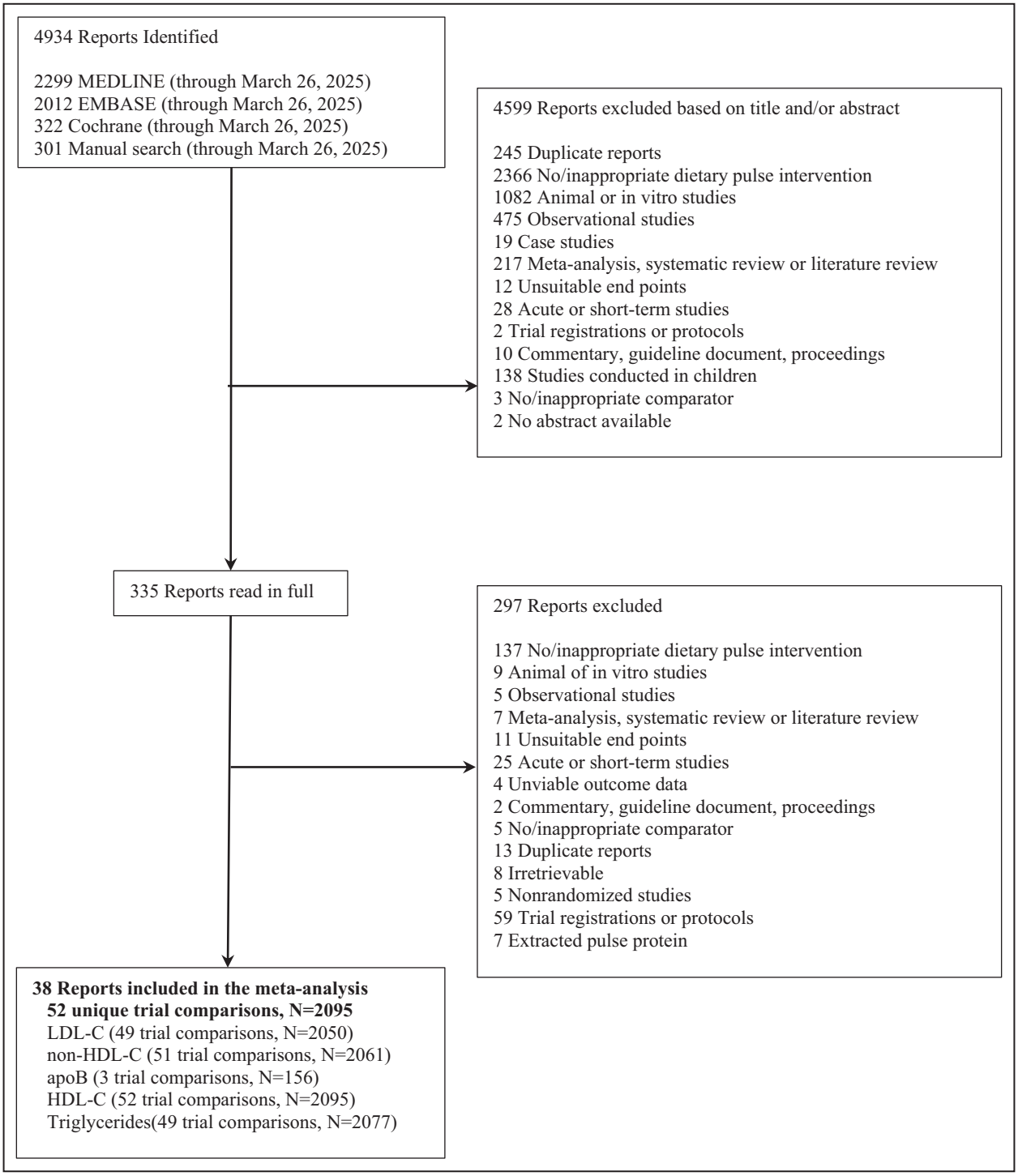


Figure 1. Flow of the literature on the effect of whole dietary pulses on blood lipids.
apoB indicates apolipoprotein B; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; and non-HDL-C, non-high-density lipoprotein cholesterol.

followed by agency (31%–33% across outcomes), and industry (14%–18% in LDL-C, non-HDL-C, HDL-C, and triglycerides; 33% in apoB), with few not reported (0%–6% across outcomes).

Risk of Bias

Figures S1 through S5 show the ROB assessments for individual trials using the Cochrane Risk of Bias Tool 2.0. Across outcomes, most trials were assessed as

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Table. Summary of the Characteristics of Included Trials Assessing the Effect of Whole Dietary Pulses on Blood Lipids*

Trial characteristics	LDL-C	Non-HDL-C	ApoB	HDL-C	Triglycerides
Trial comparisons, n	49	51	3	52	49
Study size, median (range)†	26 (6–150)	23 (5–150)	22 (20–114)	25 (5–150)	28 (6–150)
Age, y, median (range)	52 (21–64)	52 (21–64)	37 (26–48)	52 (21–64)	52 (22–64)
Health status, n	Absence of disease=9, diabetes/T2D/ prediabetes=11, adults with OW/OB=4, hypercholesterolemia=15, insulin resistance=4, pre-MetS=2, PCOS=1, mixed health status=1, other=2	Absence of disease=11, diabetes/T2D/ prediabetes=11, adults with OW/OB=4, hypercholesterolemia=15, insulin resistance=4, pre-MetS=2, PCOS=1, mixed health status=1, other=2	Adults with OW/OB=1, Hypercholesterolemia=1, pre-MetS=1	Absence of disease=11, diabetes/T2D/ prediabetes=11, adults with OW/OB=5, hypercholesterolemia=15, insulin resistance=4, pre-MetS=2, PCOS=1, mixed health status=1, other=2	Absence of disease=8, diabetes/T2D/ prediabetes=11, adults with OW/OB=5, hypercholesterolemia=15, insulin resistance=4, pre-MetS=2, PCOS=1, mixed health status=1, other=2
Male:female ratio, %‡	52:48	49:51	41:59	48:52	52:48
Country (No. of comparisons)	Australia=6, Canada=11, Iran=6, Mexico=2, New Zealand=2, Pakistan=2, Singapore=1, South Africa=1, United Kingdom=2, United States=16	Australia=6, Canada=13, Iran=6, Mexico=2, New Zealand=2, Pakistan=2, Singapore=1, South Africa=1, United Kingdom =2, United States=16	Canada=1, Mexico=1, South Africa=1	Australia=6, Canada=13, Iran=7, Mexico=2, New Zealand=2, Pakistan=2, Singapore=1, South Africa=1, United Kingdom =2, United States=16	Australia=6, Canada=10, Iran=7, Mexico=2, New Zealand=2, Pakistan=2, Singapore=1, South Africa=1, United Kingdom =2, United States=16
Study design, %, crossover: parallel	55:45	57:43	67:33	56:44	53:47
Feeding control, %, met:sup:dietary advice:metabolic, supplement	24:59:14:0	27:57:14:0	0:100:0:0	27:56:15:0	22:59:16:0
Lipid medication use ratio, %, yes:no:mixed:unclear	3:82:10:5	3:82:10:5	0:100:0:0	3:82:10: 5	3:82:10:5
Settings, %, inpatients:outpatients:inpatient, outpatient	87:13:0	87:13:0	0:100:0	88:12:0	87:13:0
Baseline body weight, kg, median (range)§	79.6 (63.3–89.9)	79.5 (63.3–89.9)	76.1 (72.5–76.6)	79.3 (61.5–89.9)	79.5 (63.3–89.9)
Baseline body mass index, kg/m^2, median (range)	28.9 (24.2–35.9)	28.9 (24.2–35.9)	26.6 (25.0–27.2)	28.9 (24.2–35.9)	28.9 (24.2–35.9)
Baseline outcome‖, median (range)	3.29 (2.04–6.44)	4.14 (2.79–8.15)	1.00 (0.89–1.37)	1.26 (0.78–1.72)	1.60 (0.86–2.64)
Follow-up duration, wk, median (range)	6 (3–52)	6 (3–52)	4 (4–16)	6 (3–52)	6 (3–52)
Pulse dose, g/d, median (range)	130 (24–377)	130 (24–377)	90 (32–253)	130 (234–377)	130 (24–377)
Pulse protein dose, g/d, median (range)	10.4 (1.8–29.2)	10.6 (1.8–29.2)	8.6 (8.6–24.5)	10.9 (1.8–29.2)	10.4 (1.8–29.2)
Pulse protein dose, %E, median (range)	1.9 (0.4–6.8)	2.0 (0.4–6.8)	1.7 (1.7–4.5)	2.0 (0.4–6.8)	2.0 (0.4–6.8)
Pulse fiber dose, g/d, median (range)	8.7 (1.5–27.0)	9.0 (1.5–27.0)	5.2 (5.2–26.3)	9.0 (1.5–27.0)	9.0 (1.5–27.0)
Pulse fiber dose, g/1000 kcal, median (range)	4.5 (0.8–13.8)	4.7 (0.8–13.8)	2.5 (2.5–12.2)	4.7 (0.8–13.8)	4.7 (0.8–13.8)
Pulse type, n	Dry beans=28, lentils=3, chickpeas=1, dry peas=2, legume intervention=4, mixed sources=11	Dry beans=28, lentils=3, chickpeas=1, dry peas=2, legume intervention=4, mixed sources=13	Dry beans=2, mixed sources=1	Dry beans=28, lentils=3, chickpeas=1, dry peas=2, legume intervention=4, mixed sources=14	Dry beans=28, lentils=3, chickpeas=1, dry peas=2, legume intervention=4, mixed sources=11
Pulse processing, n	Whole, dried=1, whole, canned=24, extracted and made into a food=7, whole, baked good=3, whole, canned, flavored=7, mixed processing=7	Whole, dried=1, whole, canned=24, extracted and made into a food=7, whole, baked good=3, whole, canned, flavored=7, mixed processing=9	Extracted and made into a food=1, whole, baked good=1, mixed processing=1	Whole, dried=1, whole, canned=25, extracted and made into a food=7, whole, baked good=3, whole, canned, flavored=7, mixed processing=9	Whole, dried=1, whole, canned=25, extracted and made into a food=7, whole, baked good=3, whole, canned, flavored=7, mixed processing=6
Comparator, no. of comparisons	Usual diet=18, carbohydrates=14, vegetables=4, high GI=1, DASH diet=2, animal protein=5, low-carbohydrate diet =3, other comparators=1, mixed comparators=1	Usual diet=20, carbohydrates=14, vegetables=4, high GI=1, DASH diet=2, animal protein=5, low-carbohydrate diet=3, other comparators=1, mixed comparators=1	Usual diet=3	Usual diet=20, carbohydrates=14, vegetables=4, high GI=1, DASH diet=2, animal protein=5, low-carbohydrate diet=3, energy restricted diet=1, other comparators=1, mixed comparators=1	Usual diet=18; carbohydrates=14; vegetables=4; high GI=1; DASH diet=2; animal protein=4; low-carbohydrate diet =3; energy restricted diet=1, other comparators=1; mixed comparators=1

(*Continued*)

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Table. Continued

Trial characteristics	LDL-C	Non-HDL-C	ApoB	HDL-C	Triglycerides
Energy balance, %, neutral:positive:negative¶	90:0:10	90:0:10	100:0:0	88:0:12	88:0:12
Energy control, %, substitution:addition:subtraction#	98:2:0	98:2:0	67:33:0	98:2:0	98:2:0
Funding sources, %,agency:industry:agency, industry:not reported**	33:14:47:6	31:18:45:6	33.3:33.3:33.3:0	33:17:44:6	33:14:47:6

ApoB indicates apolipoprotein B; DASH, Dietary Approaches to Stop Hypertension; GI, glycemic index; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MetS, metabolic syndrome; OW/OB, overweight/obesity; PCOS, polycystic ovary syndrome; and T2D, type 2 diabetes.

*All numbers with the exception of baseline values were rounded to the nearest whole number to improve readability.

†All sample sizes reflect participants included in the data analyzed.

‡Not all studies reported the number of women and men analyzed.

§Not all trials reported baseline values. Baseline values were not reported for baseline body weight (n=5).

‖units for LDL-C, non-HDL-C, HDL-C, and triglycerides are in mmol/L and for apoB, g/L.

¶Neutral energy balance refers to the maintenance of usual energy intake. Positive energy balance refers to a greater than normal energy intake. Negative energy balance refers to a deficit in normal energy intake.

#Energy control refers to the energy intake of the intervention group compared with the control group where substitution refers to energy matched between intervention and comparator, addition refers to excess energy between intervention and comparator, and subtraction refers to deficit in energy between intervention and comparator.

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having a low ROB in selection of the reported result (82%), measurement of the outcome (100%), missing outcome data (92%), deviation from the intended intervention (82%) and randomization (96%). Only 1 trial was assessed as having a high ROB in the deviations from intended interventions and randomization process domains (2%). Of crossover trials, most were rated as low ROB for bias arising from period and carryover effects (68%) with few trials assess as high ROB (28%). Most trials were judged overall as low (54%), some trials were assessed as some concerns (36%), and one trial was assessed as high (10%).

Primary Outcome

Figure 2 and Figure S6 show the effect of whole dietary pulses on LDL-C. Whole dietary pulse consumption resulted in a significant reduction in LDL-C (49 trials; MD, −0.14 mmol/L [95% CI, −0.19 to −0.08]; P_{MD}<0.001; substantial heterogeneity: I^2=57.32%; P_Q<0.001). There was no interaction by pulse type (P=0.976); however, beans represented >50% (57%) of the overall weight in the analysis, suggesting a potential influence of pulse type due to beans predominating the overall pooled effect estimate, thus necessitating the need to look at each pulse type separately. In analyses by pulse type, beans (28 trials; MD, −0.17 mmol/L [95% CI, −0.26 to −0.07]; P_{MD}<0.001; substantial heterogeneity: I^2=61.83%; P_Q<0.001), chickpeas (1 trial; MD, −0.18 mmol/L [95% CI, −0.35 to −0.01]; P_{MD}=0.043), legume interventions (4 trials; MD, −0.13 mmol/L [95% CI, −0.23 to −0.03]; P_{MD}=0.011; no heterogeneity: I^2=0.00%; P_Q=0.995), and mixed sources (11 trials; MD, −0.13 mmol/L [95% CI, −0.22 to −0.05]; P_{MD}=0.001; no substantial heterogeneity: I^2=41.26%; P_Q=0.074) resulted in significant reductions in LDL-C.

Secondary Outcomes

Figure 2 and Figures S7 through S10 present the effect of whole dietary pulses on the secondary outcomes including non-HDL-C, apoB, HDL-C, and triglycerides. Whole dietary pulse consumption resulted in a significant reduction in non-HDL-C (51 trials; MD, −0.22 mmol/L [95% CI, −0.30 to −0.14]; P_{MD}<0.001) with substantial heterogeneity (I^2=71.35%; P_Q<0.001), apoB (3 trials, MD, −0.08 g/L [95% CI, −0.13 to −0.03]; P_{MD}=0.003) with substantial heterogeneity (I^2=93.41%; P_Q<0.001), and HDL-C (52 trials; MD, −0.03mmol/L [95% CI, −0.05 to −0.01]; P_{MD}=0.005) with substantial heterogeneity (I^2=55.39%; P_Q<0.001). There was no effect on triglycerides (49 trials; MD, −0.02 mmol/L [95% CI, −0.05, 0.02]; P_{MD}=0.356) with no substantial heterogeneity (I^2=0.00%; P_Q=0.554). Interaction by pulse type was demonstrated in the analysis for apoB ($P_{interaction}$=0.006) but not for other outcomes. However, beans represented >50% (55%–57%) of the overall weight in the analyses across other outcomes, suggesting a potential influence of pulse type. In analyses by pulse type, beans (28 trials; MD, −0.24 mmol/L [95% CI, −0.36 to −0.11]; P_{MD}<0.001; substantial heterogeneity: I^2=76.29%; P_Q<0.001), legume interventions (4 trials; MD, −0.19 mmol/L [95% CI, −0.31 to −0.07]; P_{MD}=0.002; no heterogeneity: I^2=0.00%; P_Q=0.448), and mixed sources (13 trials; MD, −0.27 mmol/L [95% CI, −0.43 to −0.10]; P_{MD}=0.002; substantial heterogeneity: I^2=76.22%; P_Q<0.001) resulted in significant reductions in non-HDL-C and beans resulted in significant reductions in apoB (2 trials; MD, −0.13 g/L [95% CI, −0.19 to −0.07]; P_{MD}<0.001; with substantial heterogeneity: I^2=95.61%; P_Q<0.001) and HDL-C (28 trials; MD, −0.05 mmol/L [95% CI, −0.07 to −0.03]; P_{MD}<0.001; no heterogeneity: I^2=0.00%;

P_Q=0.709). Other pulse types did not significantly affect other secondary outcomes.

Sensitivity Analyses

Figures S11 through S36 present the individual trial influence analyses for the effect of whole dietary pulses. The removal of Jimenez-Cruz et al.[84] or Shams et al.[93] partially explained the substantial heterogeneity for the effect of whole dietary pulses (original: I^2=57%, P_Q<0.001; after study removed: I^2=45%, P_Q<0.001 and I^2=49%, P_Q<0.001) on LDL-C without affecting the magnitude or direction of the effect. The removal of Jimemez-Cruz et al. partially explained the substantial heterogeneity for the effect of beans on LDL-C (original: I^2=62%, P_Q<0.001; after study removed: I^2=43%, P_Q=0.010) without affecting the magnitude or direction of the effect. Although the removal of Chamberlin et al.[94] fully explained the substantial heterogeneity for the effect of lentils on LDL-C (original: I^2=69%, P_Q=0.040; after study removed: I^2=0%, P_Q=0.904), the direction of effect changed (original: MD, −0.09 mmol/L; after study removed: MD, 0.02). The removal of Hosseinpour-Niazi et al. (CC + TT carriers)[64] resulted in a loss of significance for the effect of legume interventions on LDL-C (original MD, −0.13 mmol/L [95% CI, −0.23 to −0.03], *P*=0.011; after study removed: MD, −0.13 mmol/L [95% CI, −0.27 to 0.01], *P*=0.050).

The removal of Chamberlain et al.[92] resulted in a gain of significance for the effect of lentils on non-HDL-C (original: MD, −0.13 mmol/L [95% CI, −0.35 to 0.09], *P*=0.234; after study removed: MD, −0.23 mmol/L [95% CI, −0.38 to −0.08], *P*=0.002).

Although the removal of Escobedo et al.[81] fully explained the substantial heterogeneity for the effect of dietary pulses on apoB (original: I^2=93%, P_Q<0.001; after study removed: I^2=0%, P_Q=0.873), the direction of effect changed (original: MD, −0.08 mmol/L; after study removed: MD, 0.02 mmol/L). The removal of Escobedo et al.[86] resulted in a loss of significance for the effect of beans on apoB (original MD, −0.13g/L [95% CI, −0.19 to −0.07], *P*<0.001; after study removed: MD, 0.03/L [95% CI, −0.06 to 0.12], *P*=0.506).

The removal of Kazemi et al.[68] or Mizelman et al.(female (F))[69] partly explained the substantial heterogeneity for the effect of whole dietary pulses on HDL-C (original: I^2=55%, P_Q<0.001; after study removed: I^2=47% P_Q<0.001). Although the removal of Chamberlin et al.[92] fully explained the substantial heterogeneity for the effect of lentils on HDL-C (original: I^2=86%, P_Q<0.001; after study removed: I^2=0%, P_Q=0.728), the direction of effect changed (original: MD, −0.05 mmol/L; after study removed: MD, 0.03 mmol/L). The removal of Hosseinpour-Niazi et al. (CC)[64] resulted in a gain of significance for the effect of legume interventions on HDL-C (original MD, −0.02 mmol/L [95% CI, −0.09 to 0.05], *P*=0.515; after study removed: MD, −0.06 mmol/L [95% CI, −0.11 to −0.00], *P*=0.021) and the removal of Wu et al.[96] changed the direction of effect (after study removed: MD, 0.01 mmol/L [95% CI, −0.02 to 0.04], *P*=0.357) for the effect of legume interventions on HDL-C.

The removal of Duane et al.[62] resulted in a gain of significance for the effect of mixed sources of dietary pulses on triglycerides (original MD, −0.05 mmol/L [95% CI, −0.12 to 0.02], *P*=0.196; after study removed: MD, −0.07 mmol/L [95% CI, −0.14 to −0.00], *P*=0.045).

Table S6 presents the sensitivity analyses for the use of fixed effect meta-analyses. The use of these different methodologies did not alter the direction, magnitude, or significance of the effect or evidence of substantial heterogeneity in any outcomes.

Table S7 presents sensitivity analyses for the use of different correlation coefficients (0.25 and 0.75) in paired analyses of crossover trials for each outcome. The use of these different correlation coefficients did not alter the direction, magnitude, or significance of the effect or evidence of substantial heterogeneity except for the use of a correlation coefficient of 0.25 in the analysis of the effect of whole dietary pulses on LDL-C where heterogeneity was not substantial (I^2=48.09%, P_Q<0.001).

Subgroup Analyses

Figures S37 through S72 present the categorical and continuous meta-regression analyses for the effect of whole dietary pulses, where there were ≥10 trial comparisons. There was significant effect modification for the effect of whole dietary pulses and beans on LDL-C and non-HDL-C by comparator, where the 1 trial that included a high glycemic index and the 3 trials of low carbohydrate comparators showed greater reductions, and by fiber difference where greater fiber differences resulted in greater reductions. There was also significant effect modification for the effect of beans on LDL-C and non-HDL-C by health status where the 3 trials in type 2 diabetes showed greater reductions, by feeding control where the 1 trial using dietary advice showed greater reductions, and by pulse processing where trials using whole, canned beans and whole, canned, flavored beans showed greater reductions. There was significant effect modification for the effect of mixed sources on non-HDL-C and triglycerides by type of mean differences where trials using change from baseline showed greater reductions.

Continuous subgroup analyses demonstrated effect modification by difference in fiber of the overall diet between groups for the effect of whole dietary pulses and beans on LDL-C and non-HDL-C and of whole dietary pulses on HDL-C where greater fiber in the intervention group showed greater reductions. Few other subgroups were significant, however were not consistent across outcomes.

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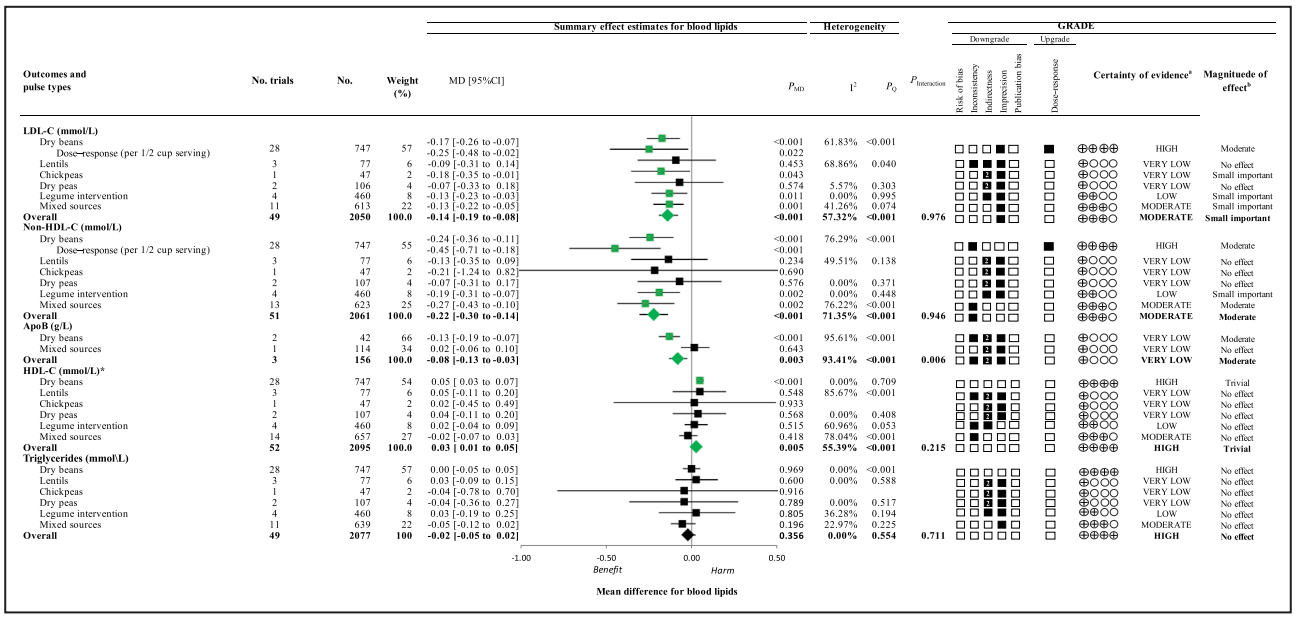


Figure 2. Summary plot of the effect of whole dietary pulses on blood lipids in randomized controlled trials.
Data are expressed as weighted mean differences with 95% CIs of the summary effect estimates using the generic inverse variance method modeled by random effect (≥5 trial comparisons) or fixed effect (<5 trial comparisons) meta-analyses. The between-study heterogeneity was assessed using the Cochran Q statistic, where P_Q<0.10 was considered statistically significant, and quantified by the I^2 statistic, where I^2≥50% was considered evidence of substantial heterogeneity. The effect estimates of whole dietary pulses from different sources are denoted as diamonds and labeled as "Overall." The interaction (variation in the effect estimates) between different sources of dietary pulses was assessed using the *Cochrane Handbook* recommended standard Q test for subgroup differences using meta-regression (significance at $P_{interaction}$<0.10). The effect estimates of individual dietary pulse protein types are denoted as squares. Any statistically significant reductions are highlighted in green. The GRADE of randomized controlled trials are rated as having a "high" certainty of evidence and can be downgraded by 5 domains and upgraded by 1 domain. The white squares represent no downgrades, filled black squares indicate a single downgrade or upgrade for each outcome, and the black square with a white "2" indicates a double downgrade for each outcome. [a]Because all included trials were randomized controlled trials, the certainty of the evidence was graded as high for all outcomes by default and then downgraded or upgraded based on prespecified criteria. Criteria for downgrades included ROB (downgraded if most trials were considered to be at high ROB); inconsistency (downgraded if there was substantial unexplained heterogeneity: I^2≥50%; P_Q<0.10); indirectness (downgraded if there were factors absent or present relating to the participants, interventions, or outcomes that limited the generalizability of the results); imprecision (downgraded if the 95% CIs crossed the MID for harm or benefit set at 0.1 mmol/L (5%) for LDL-C, non-HDL-C, HDL-C, and triglycerides and ±0.04 g/L for apoB,[12,40–42] or there was a concern with the robustness of the estimate resulting from sensitivity analyses); and publication bias (downgraded if there was evidence of publication bias based on the funnel plot asymmetry or significant Egger's or Begg's test (P<0.10) with the confirmation of evidence of small study effects by adjustment using the trim-and-fill analysis of Duval and Tweedie). The criteria for upgrades included a significant dose–response gradient that supports the direction of the pooled effect estimate. Please see Table S6 for details on the GRADE assessment. [b]For the interpretation of the magnitude, we used the MIDs (see [a]) to assess the importance of the magnitude of our point estimate using the effect size categories according to the GRADE guidance[57–59] as follows: a large effect (≥5× MID); moderate effect (≥2× MID), small important effect (≥1× MID), and trivial/unimportant effect (<1 MID). *Owing to the difference in the directionality of HDL-C compared with the other outcomes with regard to signal for benefit or harm, the sign for the MD was changed. apoB indicates apolipoprotein B; GRADE, Grading of Recommendations, Assessment, Development, and Evaluation; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MD, mean difference; MID, minimally important difference; non-HDL-C, non-high-density lipoprotein cholesterol; P_{MD}, P value of the mean difference; P_Q, P value of the heterogeneity; and ROB, risk of bias.

Subgroup analyses by ROB demonstrated significant effect modification for the effect of whole dietary pulses and mixed pulses on non-HDL-C by the domain deviations where trials with high ROB showed greater reductions; however, all categories showed significant reductions, for mixed pulses on non-HDL-C by the domain missing with trials with some concerns showing greater reductions, for whole dietary pulses on HDL-C by the domain randomization where trials with high ROB show increases, mixed pulses on HDL-C by the domain deviations where trials with high and some concerns show increases, and whole dietary pulses on triglycerides by the domain deviations where trials with high and some concerns show reductions.

Dose–Response Analyses

Figures S73 through S99 present linear and nonlinear dose–response analyses. No dose–response relationship was observed for the effect of whole dietary pulses

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or by pulse type in either linear or nonlinear analyses on LDL-C. A significant nonlinear dose response was observed for beans on LDL-C and non-HDL-C at 1 cup (P=0.040, P=0.030, respectively) thresholds. A linear analysis of the effect of beans on LDL-C and non-HDL-C up to 1-cup serving demonstrated an inverse linear dose response ($coef_{linear}$, −0.499 mmol/L per 1 cup serving [179.8 g] [95% CI, −0.095 to −0.048], P=0.030; $coef_{linear}$, −0.890 mmol/L per 1 cup serving [179.8 g] [95% CI, −1.416 to −0.364], P<0.001). There was a significant nonlinear dose response (P=0.033) and at the ½ cup threshold (P=0.027) for the effect of mixed pulses on LDL-C. There was a significant nonlinear dose response of whole dietary pulses on HDL-C (P<0.001) and at the ½ cup (P=0.004) and 1 cup (P=0.016) thresholds, where a linear analysis up to 1 cup demonstrated nonlinearity (P=0.001). There was no dose response for the effect of whole dietary pulses or by type on triglycerides.

Small-Study Effects

Figures S100 through S117 present the contour-enhanced funnel plots and assessments of small study effects across all outcomes where there were ≥10 trial comparisons. Significant publication bias was detected visually and statistically for the effect of whole dietary pulses on LDL-C (P=0.021, Egger's test), beans on LDL-C (P=0.034, Egger's test), whole dietary pulses on non-HDL-C (P=0.018, Egger's test, and P=0.087, Begg's test), beans on non-HDL-C (P=0.069, Egger's test), mixed sources on non-HDL-C (P=0.064, Egger's test, and P=0.044, Begg's test), and mixed sources on HDL-C (P=0.075, Egger's test). The trim-and-fill analyses for each of these did not demonstrate evidence of small-study effects as the inclusion of imputed studies did not change the significance, magnitude, or direction of the effect except for beans on LDL-C. Although there was evidence of small-study effect for the effect of beans on LDL-C, direction and magnitude did not change and there was no evidence of small-study effects for non-HDL-C with which LDL-C is highly correlated. There was no evidence of funnel plot asymmetry or publication bias for other analyses.

GRADE Assessment

Figure 2 and Table S8 present the GRADE assessments. The certainty of evidence for the effect of whole dietary pulses was moderate for LDL-C (small important effect) and non-HDL-C (moderate effect) owing to downgrades for imprecision and inconsistency, respectively; very low for apoB (moderate effect) owing to downgrades for inconsistency, indirectness, and imprecision; and high for HDL-C (trivial effect) and triglycerides (no effect). The certainty of evidence for the effect of beans on LDL-C and non-HDL-C was high (moderate effects) due to a downgrade for inconsistency and imprecision, respectively, and upgrade for linear dose response of beans up to 1 cup, was high for HDL-C (trivial effect) and triglycerides (no effect) and very low for apoB (moderate effects) owing to downgrades for inconsistency, indirectness, and imprecision. The certainty of evidence for the reductions observed for legume interventions and mixed sources on LDL-C (small important effects) and non-HDL-C (small important and moderate effects, respectively) were low and moderate, respectively, owing to downgrades for inconsistency, indirectness or imprecision and for the reductions observed for chickpeas on LDL-C (small important effect) was very low owing to a downgrade for imprecision and a double downgrade for indirectness as there was only 1 study, limiting generalizability. Each specific reasons for downgrades were explained in Table S6 footnotes 1 to 27. All other analyses were assessed as moderate to very low certainty.

DISCUSSION

This systematic review and meta-analysis included 52 trial comparisons providing data on 2095 middle-aged adults with or without hypercholesterolemia or type 2 diabetes, assessing the effect of different types of pulses predominantly from beans with a median dose of 130 g per day over a median follow-up of 6 weeks. We demonstrated that whole dietary pulse consumption resulted in a small important reduction in LDL-C (−0.14 mmol/L), a moderate reduction in non-HDL-C (−0.22 mmol/L) and apoB (−0.08 g/L), and a trivial reduction in HDL-C (−0.03 mmol/L), and no significant effect on triglycerides. Beans represented ~50% of the overall weight in analyses of each outcome, however there was no significant effect modification by pulse type except for the analysis of apoB. Analyses by pulse type showed beans, chickpeas, legume interventions and mixed sources decreased LDL-C and non-HDL-C (except for chickpeas) and only beans decreased apoB and HDL-C. Lentils and dried peas were not statistically significant but tended in the same direction as other pulse types. Dose response analyses demonstrated a linear dose response for beans at servings up to 1 cup/d for LDL-C (−0.499 mmol/L per cup) and non-HDL-C (−0.89 mmol/L per cup).

Findings in Relation to the Literature

This systematic review and meta-analysis provides an update on the previous analysis of dietary pulses on established lipid targets published over a decade ago[19] with a focus on examining the effect of different types of whole dietary pulses, including dose–response analyses and a GRADE assessment. With the addition of 28 trial comparisons, our meta-analysis with a median dose of 130 g whole pulses per day

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demonstrated similar reductions in LDL-C to the previous analysis (0.17 mmol/L [95% CI, −0.25 to −0.09]) with a median dose of 130 g of whole pulses per day. We also demonstrated a significant moderate reduction in non-HDL-C of −0.22 mmol/L (95% CI, −0.30 to −0.14); although their results were nonsignificant, they did tend to show a reduction (−0.09 mmol/L [95% CI, −0.19 to 0.00]). Both reductions correspond to 1 to 2× our MID for lipid targets (±0.1mmol/L[12,40–42]), which corresponds to ~1% to 2% risk reduction in CVD. The previous analysis did not observe significant interaction by pulse types; although we found >30% weight for 1 pulse type (dried beans), we demonstrated similar reductions across beans, chickpeas, and legume interventions and mixed sources, with tendencies for reductions by lentils and dried peas. Thus, all types of dietary pulses behave similarly. With 28 trial comparisons for dried beans, we also demonstrated a linear dose response of 0.25 mmol/L reduction in LDL-C and 0.45 mmol/L reduction in non-HDL-C per ½ cup of dried beans up to 1 cup with high certainty of evidence. Our results are also similar to our recent systematic review and meta-analysis of extracted pulse proteins of 14 trial comparisons with a median dose of 35 g pulse protein per day demonstrating that extracted pulse proteins decreased LDL-C by −0.23 mmol/L (95% CI, −0.36 to −0.10).[98] They also did not observe an interaction by pulse type nor a dose response, which may be due to the noted limited dose range of most studies (25–35 g protein) compared with ours (23–377 g pulses or 2–29 g protein). We found a reduction of −0.03 mmol/L (95% CI, −0.05 to −0.01) in HDL-C, which is driven primarily by the analysis by pulse types for beans. This reduction in HDL-C has not been assessed or replicated in previous systematic reviews and meta-analyses.[19,98] According to our established minimally important difference for HDL (±0.1mmol/L, which corresponds to a 1% risk reduction in CVD when taking statins[12,40–42]), this reduction in HDL is below a clinically meaningful change, especially in a nontherapeutic lipid target. When considering the reduction observed in our 2 therapeutic lipid targets (LDL-C and non-HDL-C), the reductions we observed are 4% and 6%, respectively, which is comparable to health claims for other foods in the United States and Canada.[12–15,99]

Although the present study did not demonstrate effect modification by sex, the previous analyses observed modification by sex, albeit in opposing directions. The meta-analysis of extracted pulse proteins found that studies with a greater proportion of women had greater reductions in LDL-C and non-HDL-C,[98] whereas the older meta-analysis of whole pulses found that trials with more men had a greater reduction in LDL-C and no effect modification on non-HDL-C.[19] The lack of effect modification by sex in the present study may be due to the larger number of studies (n=52 trial comparisons versus 14 and 26 in the previous meta-analyses, respectively) where data were not stratified by sex and with the majority representing equal ratio of men and women. This limited subgroup analyses by sex. This calls for future studies to stratify and report data by sex to better understand if there is effect modification by sex. The effect of sex could be the result of women receiving a higher proportion of their protein requirement through the intervention compared with men because the same absolute amount of extracted pulse protein was given to both women and men across all trials. Moreover, data on gender, including gender roles, would be useful as these can affect dietary adherence which has been observed in women, including a greater consumption of dietary pulses.[100] Future studies should also consider factors that may influence sex effects, including menopausal status for women and baseline levels, which modify interpretation of observations considering sex effects in CVD risk and disparities in nutritional behaviors across sexes[100] and the gender spectrum,[101,102] including preferences for the intake of dietary pulses.[100]

Whole pulses may lower cholesterol through their contributions of both protein and dietary fiber to the diet.[103] In the present study, there was effect modification by difference in fiber content of the intervention where studies with greater contribution of fiber to the diet resulted in greater reductions in LDL-C. Although we did not observe significant effect modification by pulse protein, those trials with greater pulse protein tended to result in greater reductions in LDL-C and non-HDL-C. Furthermore, in the previous meta-analysis on extracted pulse proteins, beans demonstrated a reduction of 0.24 mmol/L in LDL-C for a median dose of 35 g pulse protein, which represents ~350 g of beans. Given our linear dose response for beans up to 1 cup (~180 g), this reduction would predict a 0.10 mmol/L lower LDL-C for 11 g of bean protein or ~130 g/d beans, which was the median intake in the present analysis. Relative to the overall −0.25 mmol/L reduction in LDL-C per ½ cup beans in this study, protein may be a main driver of the reduction plus some additive effect of fiber and other nutrients like polyphenols. There is evidence from human studies on soy proteins, which are legumes like dietary pulses, that their bioactive peptides may sequester bile acids, reducing intestinal cholesterol absorption,[104,105] and may increase hepatic LDL-C receptor expression, resulting in an increased clearance of apoB-containing particles from circulation.[106] There is also evidence that the soluble and insoluble fibers within dietary pulses may disrupt enterohepatic circulation of cholesterol, preventing the reabsorption of bile acids by binding them

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in the intestinal lumen and promoting their excretion and increasing in hepatic production thereby reducing circulating cholesterol.[62,107–110]

Future Directions

The recommendations in national dietary guidelines[111] and clinical practice guidelines on nutrition therapy for dyslipidemia, CVD, and diabetes[8–11,112] are to consume more plant protein foods such as soy, pulses, nuts, and seeds. Although evidence in support of these recommendations include health claims for soy and nuts for cholesterol and coronary heart disease risk reduction,[12–14] there are no similar health claims for dietary pulses. The 0.14 mmol/L, or 4% reduction in LDL-C observed from the average 130 g/d of whole dietary pulses, is similar to those observed for the doses of a variety of foods carrying health claims for cholesterol or coronary heart disease risk reduction (eg, 25 g/d soy protein,[12] 45 g/d nuts,[13,14] 3 g/d of beta-glucan oat fiber,[5,40,113] 7 g/d psyllium,[40,113] and 2 g/d plant sterols[4,114]). With the global consumption of dietary pulses at only 21 g per capita per day, the present evidence could support a health claim, as well as updates to clinical practice guidelines and public health recommendations to drive population intake of dietary pulses for cardiovascular health and support health care providers in translating guidelines.

Strengths and Limitations

Strengths of our systematic review and meta-analysis include our comprehensive and reproducible search strategy, our inclusion of only randomized controlled trials to reduce evidence susceptible to bias, our comprehensive examination of possible sources of heterogeneity, examining the shape and strength of dose–response relationships, and application of the GRADE approach to assess the certainty of evidence.

Limitations of our study include that we downgraded for inconsistency for the effect of whole dietary pulses on non-HDL-C and apoB, as well as for some pulse types, due to unexplained heterogeneity. We downgraded for serious indirectness for apoB for the effect of whole dietary pulses due to inclusion of only 3 trials of 156 participants of generally younger adults (median 37 years) which limits generalizability and double downgraded for very serious indirectness for pulse types across outcomes with 3 trials or less because with 47 to 107 participants, a narrow range of age and health statuses limited generalizability. We downgraded for serious imprecision for the effect of whole dietary pulses on LDL-C and apoB and for various pulse types across outcomes because the 95% CIs of the pooled effect estimates crossed the prespecified MIDs, which means that results may not be clinically relevant.

Weighing the strengths and limitations, the certainty of evidence was moderate for the effect of whole dietary pulses on LDL-C and non-HDL-C, high on HDL-C and triglycerides, and very low for apoB. The certainty of evidence was high for the effect of beans on all outcomes except for apoB, which was very low and was moderate to very low for all other pulse types across outcomes.

CONCLUSIONS

Our systematic review and meta-analysis of available randomized controlled trials identified 52 trial comparisons providing data on 2095 middle-aged adults with or without hypercholesterolemia or type 2 diabetes, investigating the effect of different dietary pulses on therapeutic lipid targets. The synthesis of evidence provided a good indication that consuming pulses at a mean dose of 130 g/d results in a small important reduction in LDL-C (–0.14 mmol/L), as well as a good indication for a moderate reduction in non-HDL-C (–0.22 mmol/L) and apoB (–0.08 g/L) and a trivial reduction in HDL-C (–0.03 mmol/L), with no effect on triglycerides. Analyses by pulse type showed beans decreased LDL-C and non-HDL-C slightly, mixed pulse sources likely decreases LDL-C slightly, legume interventions may reduce LDL-C slightly, beans may reduce apoB and chickpeas may reduce LDL-C, but the evidence is very uncertain and only beans result in few differences in HDL-C. Lentils and dried peas showed no statistically significant effect but tended in the same direction as other pulse types. Dose–response analyses demonstrated a linear dose response for beans at servings up to 1 cup/d for LDL-C (–0.25 mmol/L per 0.5 cup) and non-HDL-C (–0.45 mmol/L per 0.5 cup). The main sources of uncertainty in the effect of whole dietary pulses were imprecision for LDL-C and inconsistency for non-HDL-C and inconsistency, imprecision, and indirectness for apoB due to few available trials. Imprecision was a main source of uncertainty across pulse types and indirectness for some types. To address these uncertainties, there remains a need for larger, high-quality randomized trials assessing a broader variety of pulse types, including lentils, chickpeas, and dry peas, as there were few studies identified. This evidence highlights the beneficial effects of dietary pulses on therapeutic lipid targets and support health claims, public health messages, and translation of clinical practice guidelines to encourage the consumption of dietary pulses to reduce cardiovascular risk.

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(Clinical Research Organization), and his two daughters, Wendy Jenkins and Amy Jenkins, have published a vegetarian book that promotes the use of the low glycemic index

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plant foods advocated here, *The Portfolio Diet for Cardiovascular Risk Reduction* (Academic Press/Elsevier 2020 ISBN:978-0-12-810510-8) and his sister, Caroline Brydson, received funding through a grant from the St. Michael's Hospital Foundation to develop a cookbook for one of his studies. John L. Sievenpiper has received research support from the Canadian Foundation for Innovation, Ontario Research Fund, Province of Ontario Ministry of Research and Innovation and Science, Canadian Institutes of Health Research, Diabetes Canada, American Society for Nutrition, The United Soybean Board (US Department of Agriculture soy "Checkoff" program), Protein Industries Canada (a Government of Canada Global Innovation Cluster), Almond Board of California, European Fruit Juice Association, the Glycemic Control and Cardiovascular Disease in Type 2 Diabetes Fund at the University of Toronto (a fund established by the Alberta Pulse Growers), the Plant Protein Fund at the University of Toronto (a fund which has received contributions from IFF among other donors), the Plant Milk Fund at the University of Toronto (a fund established by the Karuna Foundation through Vegan Grants), and the Nutrition Trialists Network Fund at the University of Toronto (a fund established by donations from the Calorie Control Council, Physicians Committee for Responsible Medicine, and Login5 Foundation). He has received food donations to support randomized controlled trials from the Almond Board of California, California Walnut Commission, Danone, Mantra Pharma, Terra Delyssa, House Foods, General Mills, WK Kellogg Company, Quaker, Procter & Gamble, Abbott, and Dairy Farmers of Canada. He has received travel support, speaker fees or honoraria from Nestlé, Abbott, General Mills, International Food Information Council, International Sweeteners Association, Calorie Control Council, International Stevia Council, Mantra Pharma Inc., Chinese Institute of Food Science and Technology, and Collaborative CME and Research Network. He has or has had ad hoc consulting arrangements with Almond Board of California, Perkins Coie LLP, Ingredion, and Brightseed. He is on the Clinical Practice Guidelines Expert Committees of Diabetes Canada, European Association for the Study of Diabetes, Canadian Cardiovascular Society, and Obesity Canada/Canadian Association of Bariatric Physicians and Surgeons. He serves as an unpaid member of the Board of Trustees of Institute for the Advancement of Food and Nutrition. He is Vice President – Clinical of the Canadian Nutrition Society, founding member of the International Carbohydrate Quality Consortium, Executive Board Member of the Diabetes and Nutrition Study Group of the European Association for the Study of Diabetes, and Director of the Toronto 3D Knowledge Synthesis and Clinical Trials foundation. His spouse is an employee of AB InBev. Laura Chiavaroli has received research support from the Canadian Institutes of Health Research, Protein Industries Canada (a Government of Canada Global Innovation Cluster), the United Soybean Board (US Department of Agriculture soy "Checkoff" program), and the Alberta Pulse Growers Association. She has received honoraria from the Arkansas Children's Hospital and Plant-Based Health Professionals UK and travel funding from the World Sugars Research Organization for an internal presentation in 2022. Shuting Yang, Songhee Back, Antonio Rossi, Victoria Chen, Erika J. Lin, Lukas Haintz, Yue-Tong Chen, Elmirah Ahmad, Jacqueline Gahagan, Vanessa Ha, Sonia Blanco Mejia, Russell J. de Souza, Joseph Beyene, Marcia M. English, Vladimir Vuksan, Robert G. Josse, and Lawrence A. Leiter report no relevant competing interests.

Supplemental Material

Tables S1–S8
Figures S1–S117

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