

Dietary Pulses, Satiety, and Food Intake: A Systematic Review and Meta-analysis of Acute Feeding Trials

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He is on the Clinical Practice Guidelines Expert Committee for Nutrition Therapy of both the CDA and EASD, as well as being on the ASN writing panel for a scientific statement on the metabolic and nutritional effects of fructose, sucrose and high fructose corn syrup. He is a member of the International Carbohydrate Quality Consortium (ICQC) and Board Member of the DNSG of the EASD. He serves an unpaid scientific advisor for the ILSI North America, Food, Nutrition, and Safety Program (FNSP). His wife is an employee of Unilever Canada. S.S.L., V.H.J., L.A.A.S., S.B.M. and L.A.L. have no declared conflicts of interest related to this article. Additional Supporting Information may be found in the online version of this article.

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Objective: To assess the effect of dietary pulses (beans, peas, chickpeas, lentils) on acute satiety and second meal intake, a systematic review and meta-analysis was conducted.

Methods: MEDLINE, EMBASE, CINAHL, and the Cochrane Registry (through May 6, 2013) were searched for acute controlled trials examining the effect of dietary pulses on postprandial satiety or second meal intake compared with isocaloric controls. Two independent reviewers extracted data and assessed methodological quality and risk of bias. Data were pooled by generic inverse variance random effects models and expressed as ratio of means (RoMs) for satiety and mean differences (MDs) for second meal food intake, with 95% confidence intervals (95% CIs). Heterogeneity was assessed (Q statistic) and quantified (I^2 statistic). Protocol registration: clinicaltrials.gov identifier, NCT01605422.

Results: Nine trials met the eligibility criteria. Dietary pulses produced a 31% greater satiety incremental area under the curve (IAUC) (RoM = 1.31, 95% CI: 1.09 to 1.58, $P = 0.004$; Phet = 0.96; $I^2 = 0\%$) without affecting second meal intake (MD = -19.94 , 95% CI: -75 - 35 , $P = 0.48$; Phet = 0.01; $I^2 = 63\%$). Our data are limited by the small sample sizes, narrow participant characteristics and significant unexplained heterogeneity among the available trials.

Conclusions: Pooled analyses show that dietary pulses contribute to acute satiety but not second meal intake.

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Introduction

Approximately 80% of weight loss interventions are ultimately unsuccessful, resulting in regain of weight lost during the trial (1). This may be due, in part, to hunger and food cravings that compromise longer-term adherence to energy-reduced eating plans (2). Thus a successful weight loss and maintenance intervention must manage appetite and reduce cravings, while maintaining a healthy diet.

Dietary pulses, defined by the FAO as non-oilseed legumes harvested solely for their dry grain (e.g., beans, peas, chickpeas, and lentils) (3), are high in fiber and protein and low in glycemic index—properties which have been shown to reduce appetite and acute food intake (4,5). Evidence from controlled feeding trials has shown that dietary pulses can lead to weight loss (6-8) and improve glycemic control (8,9), a metabolic benefit which itself has been linked to appetite reduction (10).

Mechanistically, there is a range of plausible factors supporting an effect on food intake regulation by dietary pulses. Proposed mechanisms include innate resistance to digestion of key components, bio-active compounds affecting digestion and satiety signals, and the low energy density of dietary pulses. In turn, these are related to the content and composition of constituents including carbohydrates (e.g., fiber, starches, and oligosaccharides), protein, and phytochemicals; the 2009 review article by McCrory et al. goes into more detail summarizing the relevant pathways (11).

Observational studies have also shown the intake of dietary pulses to be associated with reduced downstream chronic disease risks for cardiovascular disease (12) and certain cancers (13), as well as better overall diet quality (14). Owing to these benefits, dietary pulses have been recommended as part of a healthy diet by various international dietary guidelines including Canada's Food Guide (15) and the Dietary Guidelines for Americans (16). In light of evidence for beneficial effects on blood pressure (17), the American Heart Association (AHA) also recommends the consumption of dietary pulses

as part of Dietary Approaches for Stopping Hypertension (DASH) dietary patterns for cardiovascular risk reduction (18).

Despite evidence that dietary pulses may contribute to weight loss (6-8), it remains unclear whether this benefit is mediated by an acute effect of dietary pulses on food intake regulation (19-28). In particular, we were interested in the effects of whole dietary pulses, as distinct from specific components or isolates, which would only partially reflect the more practically relevant effects of the whole food. To summarize and quantify the data related to this question, we conducted a systematic review and meta-analysis of acute feeding trials investigating the effect of whole dietary pulses on satiety and second meal food intake in humans.

Methods

This systematic review and meta-analyses was planned and conducted following the Cochrane Handbook for Systematic Reviews of Interventions (29), and data were reported in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (30). The protocol was registered at clinicaltrials.gov (clinicaltrials.gov identifier, NCT01605422).

Literature search

We searched MEDLINE, EMBASE, CINAHL, and the Cochrane Registry from inception through May 6, 2013 for eligible trials. Database searches were supplemented by manual searches of the reference lists of included reports and previous reviews. No restrictions were placed on language. Supporting Information Table S1 shows our complete search strategy.

Study selection

We included randomized, acute single-bolus feeding trials with treatment and control arms matched for total energy ($\pm 10\%$ kcal) that provided an estimate of satiety, wherein satiety signifies the reduction of appetite after consumption (as opposed to satiation, which is defined as the fullness causing termination of a meal) (31), and/or second meal food intake (defined as the amount of food energy consumed at

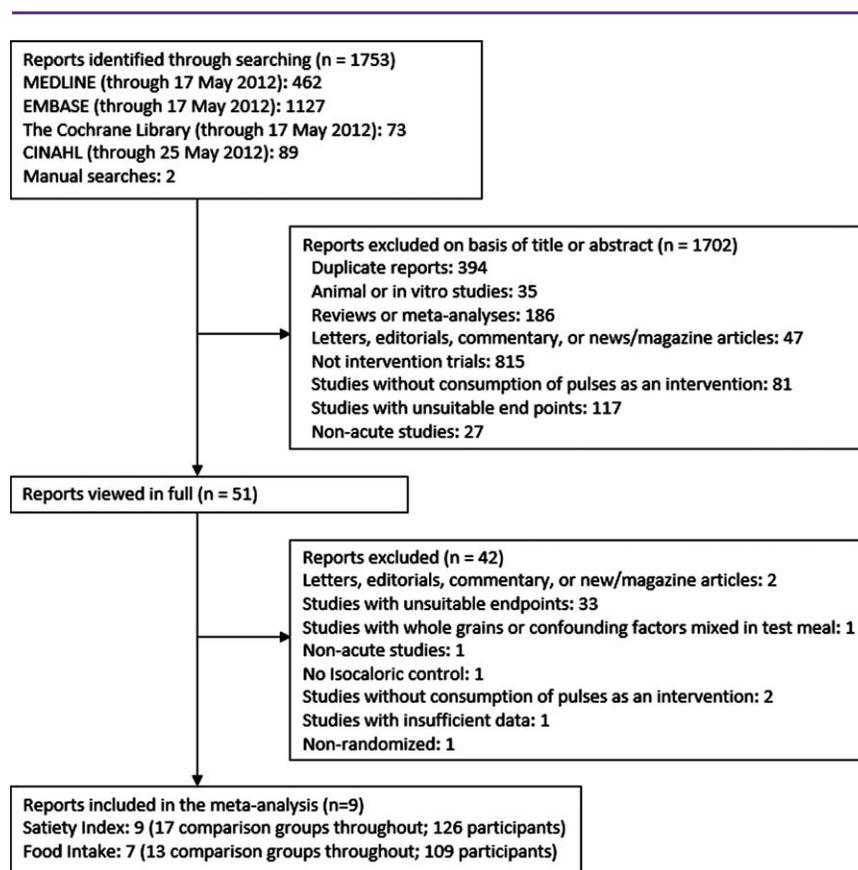


FIGURE 1 Summary of evidence search and selection.

the next meal) after acute ingestion of one or more whole dietary pulse varieties. Trials using processed dietary pulses were eligible if no parts of the dietary pulse were omitted; for example, trials using ground dietary pulse flours were included, but those using only dietary pulse protein were not. We excluded trials in which test meals contained other factors that could plausibly contribute to satiety effects, such as whole grains, which would complicate the isolation of a dietary pulse-specific effect; and trials using only dietary pulse extracts. Non-human trials were also excluded.

Data extraction

Two independent reviewers (SSL and VHJ) extracted relevant data. The following characteristics were extracted from each eligible trial: study setting, design, blinding, sample size, participant characteristics, dietary pulse form and type, second meal format, duration of test, and meal macronutrient distributions. The methodological quality and risk of bias of the eligible trials were also assessed independently, in duplicate (by SL and VHJ). Study quality was assessed using a modified Heyland methodological quality score (MQS) (32). This assessment tool assigns points for six different domains of study quality (randomization, blinding, analysis, sample selection and compatibility, follow-up, and quality of the intervention methods) for a total of 13 points. Trials with an MQS ≥ 8 were considered high quality. Risk of bias was assessed by the Cochrane risk of bias tool (29). This tool categorizes trials as high risk, low risk, or unclear risk of bias based on criteria pertaining to selection bias,

blinding, incomplete outcome data, and reporting bias. Any disagreements between reviewers were reconciled by consensus.

Statistical methods

Data were analyzed using Review Manager version 5.1.7 (The Nordic Cochrane Centre, The Cochrane Collaboration, Copenhagen, Denmark) for primary analyses and Stata version 12 (StataCorp, College Station, TX) for meta-regression and publication bias tests. We assessed two endpoints in our meta-analysis. The first was the satiety index (SI) as described by Holt et al., where $SI = (\text{satiety incremental area under the curve [IAUC] of test food}) / (\text{satiety IAUC of control})$ (20). When satiety IAUCs or SI were not directly provided, IAUCs were calculated from satiety graphs using previously described methods (33), and used to derive an SI (see Supporting Information Table S2 for details on derivation). In most trials satiety was assessed on a scale ranging from “extremely hungry” to “extremely full” or “extremely satisfied”. In trials where these data were unavailable, appetite incremental areas above the curve (IAACs) were used to derive a ratio analogous to SI. SI data were expressed as ratio of means (RoM) and 95% CIs and analyzed in the logarithm method described by Friedrich et al. (34). PlotDigitizer version 2.5.1 (Free Software Foundation, Boston, MA) was used to extract satiety scores from graphs where applicable.

The second endpoint we assessed was second meal food intake (FI; measured in kcal) at the first meal following ingestion of the dietary

TABLE 1 Characteristics of isocaloric single bolus feeding trials investigating the effect of dietary pulses on food intake regulation

Study-year (reference)	Subjects	Age (mean ± SD)	BMI (mean ± SD)	Setting	Design	Pulse form ^a	Pulse dose ^b
Hall et al., 2005 (19)	11 N (9M:2F)	31.6 ± 6.0 y	24.7 ± 2.7 kg m ⁻²	OP, Australia	C	Bread, M	7.7 g
Holt et al., 1995 (20)	11 N (6M:5F)	22.4 ± 2.8 y	24.3 ± 3.1 kg m ⁻²	OP, Australia	C	Whole, M	250 g ^b
Johnson et al., 2005 (21)	11 N (9M:2F)	32 ± 6.6 y	24.7 ± 2.7 kg m ⁻²	OP, Australia	C	Bread, M	17 g ^b
Keogh et al., 2011 (22)	20 N (10M:10F)	29.4 (20.1-44.8) y	28.1 (18.4-24.8) kg m ⁻²	OP, Australia	C	Bread, M	7.6 g ^b
Leathwood et al., 1988 (23)	6 N (3M:3F)	35-45 y	19.2-27.2 kg m ⁻²	OP, Switzerland	C	Whole, M	160 g
Lee et al., 2006 (24)	16 N (8M:8F)	58.6 ± 7.2 y	31.3 ± 4.5 kg m ⁻²	OP, Australia	C	Bread, M	38 g
Mollard et al., 2011 (25)	25 N (25M)	21.3 ± 2.5 y	21.6 ± 1.5 kg m ⁻²	OP, Canada	C	Whole, M	311 g
Winham et al., 2007 (26)	11 N (4M:7F)	40 ± 13 y	24.8 ± 4.3 kg m ⁻²	OP, USA	C	Spread, M	260 g
Wong et al., 2009 (27)	15 N (15M)	18-35 y	20-25 kg m ⁻²	OP, Canada	C	Whole, M	292.5 g

Comparator ^c	Pulse type	Energy ^d	Composition ^e	Duration ^f	MQS ^g	Funding sources	Conflict of interest
White bread	Lupin	308 kcal (adj 239)	67:19:14	175 min (b)	11	Agency	-
White bread	Lentil, Bean	239 kcal	70:09:21	120 min (b)	7	Agency, industry	-
White bread	Chickpea	313 kcal	65:27:08	175 min (b)	8	Industry	-
White bread	Lupin	313 kcal	61:26:13	120 min (b)	10	Agency, industry	-
Potato puree	Bean	298 kcal	44:30:26	270 min (b)	12	Industry	-
White bread	Lupin	395 kcal	61:16:23	180 min (a)	12	Agency	-
Mac and cheese	Chickpea, Lentil, Yellow Pea	600 kcal	69:14:16	260 min (a)	11	Industry	-
Placebo	Pinto Bean, Navy Bean, Black-eyed Pea	630 kcal	75:10:15	120 min (a)	9	Agency	-
White bread	Chickpea, Lentil, Navy Bean, Yellow Pea	300 kcal	75:06:19	120 min (a)	12	Agency, industry	-

BMI = body mass index; C = crossover; F = female; M = male; MQS = Heyland methodological quality score. N = normal (i.e. healthy patients).

^aDietary pulses were provided in flour form and incorporated into bread (Bread), in whole cooked form (Whole), or processed into the form of a spread (Spread); M indicates mixed meals (i.e. accompanied by other sources of energy, including sauces and drinks).

^bPulse dose represents the mean mass of pulse in intervention groups. For trials which did not provide enough data to calculate pulse dose, we used an approximation based on data provided. For Holt et al. (20), we assumed that the sauce weighs about 50 g for each pulse treatment, so that the mean pulse dose comes out to 250 g. For Johnson et al. (21), we used the values given from another study by the same group (19) to estimate the pulse dose that 24.3% flour replacement/104 g bread represents; by applying the same ratio as Hall et al. (19), 7.7 g/10% flour replacement/115 g bread, we found that 24.3% flour replacement/104 g bread would mean about 17 g chickpea flour in the test meal. For Keogh et al., the authors note that their bread was from Bodhi's Bakehouse, whose bread is about 10% lupin flour by mass, so 10% of 76 g is 7.6g (22).

^cComparator refers to the reference food used for the control meal. Placebo indicates no direct comparator, but extra orange juice was used compensate for caloric differences.

^dEnergy denotes the mean kcal of the test and control meals in each study (adj 239 indicates that the results were statistically adjusted to reflect a 239 kcal meal).

^eValues are for the energy from CHO:Fat:Protein as a mean of test and control meals.

^fDuration indicates length of time from either (a) after or (b) beginning of meal, over which IAUC was calculated.

^gStudy quality was assessed by the Heyland methodological quality score (MQS).

pulse meal. All FI data were expressed as mean differences (MD) with 95% confidence intervals (95% CIs); since all trials included were crossover trials, we estimated the between-treatment MD using

the methods described by Elbourne et al. (35). Missing standard deviations for difference in FI were imputed using published methods (29,35): we used the only trial reporting *P* value for a pairwise

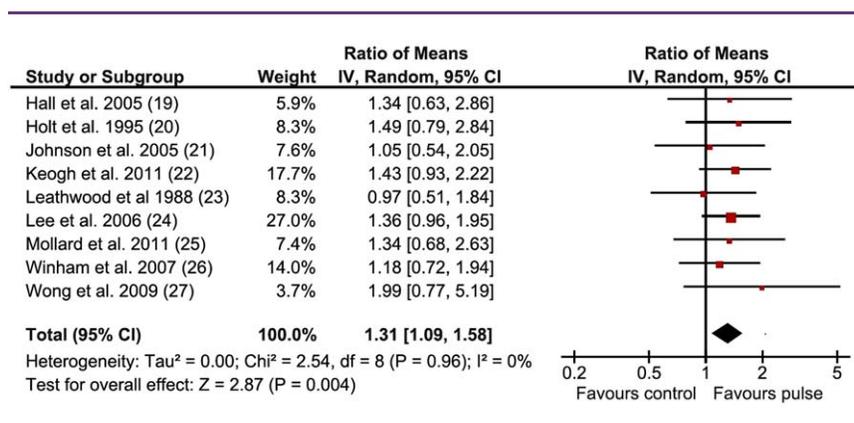


FIGURE 2 Forest plot of satiety index. The pooled effect estimate (diamond) is shown. Paired analyses were applied to all crossover trials (35). Data are expressed as RoMs with 95% CIs, using generic inverse-variance random-effects models. Inter-study heterogeneity was tested using the Cochran Q statistic (chi-square) at a significance level of $P < 0.10$. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

comparison between dietary pulse and control groups (19) to derive a between-groups correlation coefficient, which was used to impute the missing standard deviation of MD for the trial by Johnson et al. (21). For the trial by Holt et al. (20), we estimated standard deviations of FI values based on the standard deviations of the most methodologically similar trial (22), which we then used with the derived between-groups correlation coefficient to estimate a standard deviation of MD.

Both the pooled satiety index and pooled second meal food intake were derived using Dersimonian and Laird random-effects models. Multiple intervention arms within a single trial were pooled to obtain a single pairwise comparison (29).

The presence of interstudy heterogeneity was assessed using the Cochran Q statistic ($P < 0.10$ significant) and quantified using the I^2 statistic ($>50\%$ substantial) (29). Potential sources of clinical and/or methodological heterogeneity were investigated by sensitivity and subgroup analyses. In our sensitivity analysis we recalculated the pooled effect estimate after removal of each individual trial; additionally, for SI we also examined the effects of removing the trials using appetite IAACs instead of satiety IAUCs, and for FI we assessed the effects of using correlation coefficients of 0, 0.33, 0.66, and 0.99 in place of the imputed correlation coefficient to evaluate the impact of the imputed value on our pooled MD (29).

Our a priori subgroups were duration of satiety test (≤ 120 min or >120 min), dietary pulse type (beans, lentil, lupin, chickpea, or split peas), dietary pulse dose (approximate mass of one serving, <150 g or ≥ 150 g [<0.75 cup or ≥ 0.75 cup]) (14), study quality (Heyland MQS <8 or ≥ 8), disease status (healthy or other), study design (crossover or parallel), and difference in each protein and fiber content (<10 g or ≥ 10 g and <4 g or ≥ 4 g, respectively). Ad hoc analyses was also performed for mean BMI (<25 kg m^{-2} or ≥ 25 kg m^{-2}), absolute test meal protein content ($<20\%$ or $\geq 20\%$), and absolute test meal fiber content (<28 g/1000 kcal or ≥ 28 g/1000 kcal). The significance of between-subgroup differences was assessed using meta-regression with dummy variables, and continuous meta-regression analyses were used to assess effects of dietary pulse dose and differences in protein and fiber content on SI and FI.

Publication bias was assessed by inspection of funnel plots and by Egger and Begg tests.

Results

Search results

Figure 1 shows the trial selection process. Of the 1753 reports identified by our search, 1702 were excluded based on review of titles and abstracts. The remaining 51 reports were reviewed in full, and of these 42 were excluded, leaving a total of one trial from each of 9 reports providing data for acute feeding trials to be included in our analyses (19-27). All nine reports provided data for satiety or an equivalent measure, and seven reports provided data for FI (19-22,24,25,27).

Trial characteristics

Table 1 lists trial characteristics in detail. All trials were isocaloric, single bolus feeding trials; in total there were 126 participants, including 98 for which FI was reported. Subjects tended to be young to middle-aged (median age, 31.6 years [interquartile range {IQR}, 22.4-40 years]), with slightly more men than women enrolled (median male-female ratio, 6:5). Participants were healthy and generally of normal weight, with only a couple trials that had overweight or obese subjects (median BMI, 24.7 kg m^{-2} [IQR, 24.4-26.5 kg m^{-2}]). Trials tended to be small; the median number of participants was 11 (IQR, 11-18).

Dietary pulses were incorporated into test meals in a variety of ways: most commonly they were cooked whole (44%), or ground and used as flour in bread (44%). In one study they were served as a spread. These meals were most often tested against a white bread control (67%), but control meals also included potato purée, mac, and cheese, and a placebo spread. The dose varied substantially, ranging from 7.6-311 g (median, 160 g [IQR, 12.4-276.3 g]).

Overall, meals provided a median of 313 kcal energy (IQR, 299-498 kcal), and macronutrient distributions tended to have around 70%

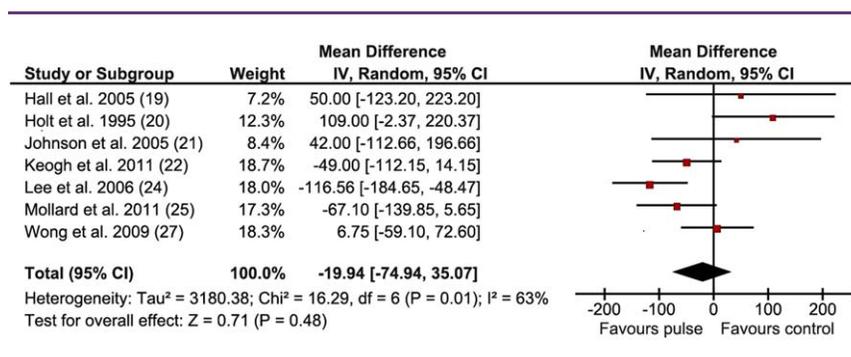


FIGURE 3 Forest plot of second meal food intake. The pooled effect estimate (diamond) is shown. Paired analyses were applied to all crossover trials (35). Data are expressed as mean differences with 95% CIs, using generic inverse-variance random-effects models. Interstudy heterogeneity was tested using the Cochran Q statistic (chi-square) at a significance level of $P < 0.10$. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

carbohydrate (ranging from 61 to 85% across all trials except one, which had 44% carbohydrate), with fat ranging from 6 to 30% and protein ranging from 8 to 26% of meal composition. Tests' duration was 120 min or more (median, 175 min [IQR, 120-220 min]). All trials used crossover designs in outpatient settings, and over half of the trials were based in Australia. Of the remaining trials, three were from North America, and one from Europe.

All trials were randomized, and 8 of 9 trials (89%) were considered high-quality (Heyland MQS ≥ 8) (see Supporting Information Table S3). Additionally, an assessment using the Cochrane Collaboration's tool for assessing risk of bias showed few indicators of high risk of bias (see Supporting Information Figure S1). Funding came from a variety of sources: 33% of trials were funded by agency, 33% by industry, and 33% by both. No conflicts of interest were declared.

Satiety

Figure 2 shows the effect of a dietary pulse meal on satiety, as compared to an isocaloric non-pulse meal. Overall, we found that dietary pulse meals increased satiety IAUCs by 31% as compared to a control (SI = 1.31, 95%CI: 1.09-1.58, $P = 0.004$; $P_{\text{het}} = 0.96$; $I^2 = 0\%$), an effect that remained throughout our sensitivity analyses; removal of no single trial affected heterogeneity estimates. Additionally, removal of the trials using average appetite in place of satiety (25) and (27) did not alter the significance of the overall effect. Inclusion of a trial that was excluded due to lack of randomization (28) also did not change the outcome.

Meta-regression identified no significant trial-level characteristics that modified the effect of dietary pulses on satiety (see Supporting Information Figure S2). Continuous meta-regression also found no significant relationship between dietary pulse dose, difference in protein, or difference in fiber and SI.

Second meal food intake

Dietary pulses meals did not significantly alter second meal food intake (MD = -20, 95% CI: -75-35, $P = 0.48$; $P_{\text{het}} = 0.01$; $I^2 = 63\%$); Figure 3 shows the effects of dietary pulse treatments on FI.

In sensitivity analyses, using different correlation coefficients did not significantly alter the outcome. However, exclusion of the trial

by Holt et al. (20) resulted in a marked increase in significance with nonsignificant heterogeneity (MD = -42; 95% CI: -89-5; $P = 0.08$; $I^2 = 46\%$; $P_{\text{het}} = 0.10$). Exclusion of other trials produced no significant changes.

Supporting Information Figure S3 shows subgroup analyses for FI. Our meta-regression identified no modification of the effect by any trial-level characteristic. Continuous meta-regression also found no significant relationship between dietary pulse dose, difference in protein, or difference in fiber and FI.

Publication bias

Supporting Information Figure S4 shows the funnel plots that were used to evaluate publication bias; there was no evidence of asymmetry or small-study effects for either SI or FI. Egger and Begg tests also did not indicate significant bias ($P > 0.05$).

Discussion

To our knowledge, ours is the first systematic review and meta-analysis to quantify the effect of dietary pulses on satiety or second meal food intake. Our analyses of 9 trials in 126 participants showed a 31% increase in satiety IAUCs following dietary pulse compared to control meals, without an effect on second meal food intake in generally healthy, younger participants.

Previous work has proposed that change in subjective satiety by 10% (AUC or mean) is likely to be a clinically meaningful effect (36). Our 31% increase in satiety was robust across dietary pulse types and forms, as well as a range of ages and BMI; aggregate analyses showed little to no heterogeneity, and the effects remained strongly significant through sensitivity analyses.

Although effects on satiety usually predict acute food intake (37), we found no effect of dietary pulses on second meal food intake. However, removal of the trial by Holt et al. (20) brought the P value down to 0.08, and eliminated significant heterogeneity. This effect of Holt et al (20) may be explained by the lack of any reported standard deviations from their FI data, with the consequence that our estimated standard deviations may not reflect the

actual standard deviations. It may also be explained by the parallel design that was used. Given that *ad libitum* food intakes vary greatly between individuals, this design combined with an imbalanced randomization may have resulted in an unrepresentative comparison.

Second meal setting is another factor that may have contributed to the non-significant effects on FI: two of our seven FI trials used varied buffets for the second meal (19,21)—a method that is considered not to be reflective of typical meals and liable to stimulate greater food intake in certain individuals (36,37). Predictably, the FI in these two trials had the highest variability (Hall et al. MD = 50, 95% CI: -123-223; Johnson et al. MD = 42, 95% CI: -113-197). Also worth considering is the timing of second meals; because protein, low-GI, and other delayed satiety effects take longer to appear, it is recommended that second test meals occur around 3-6 h after the preload so as to best reflect realistic mealtimes (38). Interestingly, only two trials had second meals over 3 h after the first meal, and these showed the greatest reductions in FI (24,25).

Regardless of effects on second meal food intake, an increase in satiety may translate into long-term weight loss. Several long-term randomized feeding trials have investigated the effect of dietary pulses on various metabolic outcomes, including weight (6-8). Although the subject remains controversial, trials have generally found greater weight loss in dietary pulse interventions compared to controls in long-term randomized feeding trials. Moreover, trials conducted using negative energy balance diets (for both treatment and control groups) (6,7) tend to show a greater difference in weight loss between treatment and control groups than those with neutral energy balance diets (8). Given that dietary pulses contribute to satiety, and satiety can in turn help with adherence to diets, it is plausible that the greater effects seen for dietary pulse interventions in negative energy balance trials are due to lessened cravings and/or hunger and thus greater adherence compared to the control groups. A study by Mollard et al. (39) supports this hypothesis; in their trial, they found that frequent consumption of dietary pulses over 8 weeks in an *ad libitum* diet resulted in reductions in food intake and waist circumference that were comparable to dietary advice to reduce energy intake. This suggests that regular dietary pulse consumption results in less calories consumed to reach a naturally comfortable state.

Our systematic review and meta-analysis has some important limitations. First, the external validity of our findings is limited by the small number of participants in individual trials (median = 11) and relatively homogeneous participant characteristics (no trials were done in children or adolescents and very few trials were conducted in older, overweight or obese participants). Second, we had fewer than ten trials for both SI and FI, and it has been previously suggested that subgroup analyses for meta-analyses incorporating fewer than ten trials have greater risk of spurious findings (40). Third, there was significant heterogeneity in our second meal food intake analysis which was largely explained away by the removal of Holt et al. (20). However, since the FI data from the Holt et al. trial was from a parallel design, with estimated standard deviations, it is possible that their data were not accurate representations of a comparison in FI between treatment groups. Fourth, there is the lack of standard methods for assessing both satiety and food intake; factors such as timing, wording of questions, scale, and foods offered varied across trials. In particular, satiety scores are difficult to make objec-

tive, being non-physiological measures. To overcome this issue, however, we used the satiety index, which controls for subjective differences between participants' perceptions and incorporates AUCs, which are regarded as being more robust measures of satiety than scores at individual time points (36). This index demonstrated a highly consistent signal for benefit (as shown by the lack of detectable inter-study heterogeneity with $I^2 = 0\%$) irrespective of protocol. Food intake measures were less robust, and methodological differences tended to be reflected in the outcomes and interstudy heterogeneity. Finally, because control meals did not match for relevant nutrients such as fiber or protein content, the practical implications of our findings are limited to comparisons between dietary pulses and generally high-glycemic index (GI) control foods such as white bread. Nevertheless, this remains a relevant comparison as dietary pulses are rarely used to replace foods high in fiber or other beneficial plant compounds (e.g., fruits, vegetables), and the substitution of dietary pulses for low-fibre high-GI foods is a legitimate and practical context in dietary planning. Protein matching, on the other hand, would provide additionally useful information in the context of dietary pulse substitution for meat and other animal protein.

In conclusion, consumption of dietary pulses may increase acute satiety in generally healthy participants. Although this increase in satiety does not manifest in a detectable decrease in second meal food intake, it may nevertheless explain some of the long-term weight loss benefits associated with dietary pulse consumption by supporting adherence to weight loss diets. There is, however, a need for further trials, given the low number of small trials, relatively homogeneous participant characteristics, and uncertainty in the effects on second meal food intake. Acute feeding trials with well-designed second meal settings, especially in overweight or obese groups, would help to isolate relevant clinical applications, and control meals matched for nutrients such as fiber and protein would be useful both to isolate the effects of dietary pulses and to direct future dietary recommendations. More high quality long-term trials would also help to strengthen the emerging evidence for the relationship between dietary pulses, satiety, and weight management. ○

Acknowledgments

JLS, CWCK, RJdS, and DJAJ conceived and designed the study. SSL conducted the systematic search. SSL and VHJ co-extracted data and assessed study quality and risk of bias. SSL performed data analyses with the assistance of RJdS, JLS, VHJ, AIC, VH, AM, and JB. Data were interpreted by SSL, JLS, RJdS, CWCK, VHJ, LC, SBM, LSAA, LAL, JB, and DJAJ. LC and SBM provided coordination and logistical support. SSL wrote the manuscript and prepared the tables and figures. All authors made significant revision of intellectual content and approved the final version of the manuscript. JLS, CWC, RJdS, and DJAJ are the guarantors for the manuscript.

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