

# Effect of non-oil-seed pulses on glycaemic control: a systematic review and meta-analysis of randomised controlled experimental trials in people with and without diabetes

J. L. Sievenpiper · C. W. C. Kendall · A. Esfahani ·  
J. M. W. Wong · A. J. Carleton · H. Y. Jiang ·  
R. P. Bazinet · E. Vidgen · D. J. A. Jenkins

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## Abstract

**Aims/hypothesis** Dietary non-oil-seed pulses (chickpeas, beans, peas, lentils, etc.) are a good source of slowly digestible carbohydrate, fibre and vegetable protein and a valuable means of lowering the glycaemic-index (GI) of the diet. To assess the evidence that dietary pulses may benefit glycaemic control, we conducted a systematic review and meta-analysis of randomised controlled experimental trials investigating the effect of pulses, alone or as part of low-GI or high-fibre diets, on markers of glycaemic control in people with and without diabetes.

**Methods** We searched MEDLINE, EMBASE, CINAHL, and the Cochrane Library for relevant controlled trials of

≥7 days. Two independent reviewers (A. Esfahani and J. M. W. Wong) extracted information on study design, participants, treatments and outcomes. Data were pooled using the generic inverse variance method and expressed as standardised mean differences (SMD) with 95% CIs. Heterogeneity was assessed by  $\chi^2$  and quantified by  $I^2$ . Meta-regression models identified independent predictors of effects.

**Results** A total of 41 trials (39 reports) were included. Pulses alone (11 trials) lowered fasting blood glucose (FBG) (−0.82, 95% CI −1.36 to −0.27) and insulin (−0.49, 95% CI −0.93 to −0.04). Pulses in low-GI diets (19 trials) lowered glycosylated blood proteins (GP), measured as HbA<sub>1c</sub> or

A. Esfahani and J. M. W. Wong contributed equally to this study.

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J. L. Sievenpiper · C. W. C. Kendall · A. Esfahani ·  
J. M. W. Wong · A. J. Carleton · H. Y. Jiang · E. Vidgen ·  
D. J. A. Jenkins  
Risk Factor Modification Centre, St Michael's Hospital,  
Toronto, ON, Canada

D. J. A. Jenkins  
Division of Endocrinology, St Michael's Hospital,  
Toronto, ON, Canada

J. L. Sievenpiper · C. W. C. Kendall (✉) · A. Esfahani ·  
J. M. W. Wong · A. J. Carleton · H. Y. Jiang · R. P. Bazinet ·  
E. Vidgen · D. J. A. Jenkins  
Department of Nutritional Sciences, Faculty of Medicine,  
University of Toronto,  
The FitzGerald Building, Room 340, 150 College Street,  
Toronto, ON M5S 3E2, Canada  
e-mail: cyril.kendall@utoronto.ca

D. J. A. Jenkins  
Department of Medicine,  
Faculty of Medicine,  
University of Toronto,  
Toronto, ON, Canada

C. W. C. Kendall  
College of Pharmacy and Nutrition,  
University of Saskatchewan,  
Saskatoon, SK, Canada

H. Y. Jiang  
Michael G DeGroote School of Medicine,  
McMaster University,  
Hamilton, ON, Canada

fructosamine ( $-0.28$ , 95% CI  $-0.42$  to  $-0.14$ ). Finally, pulses in high-fibre diets (11 trials) lowered FBG ( $-0.32$ , 95% CI  $-0.49$  to  $-0.15$ ) and GP ( $-0.27$ , 95% CI  $-0.45$  to  $-0.09$ ). Inter-study heterogeneity was high and unexplained for most outcomes, with benefits modified or predicted by diabetes status, pulse type, dose, physical form, duration of follow-up, study quality, macronutrient profile of background diets, feeding control and design.

**Conclusions/interpretation** Pooled analyses demonstrated that pulses, alone or in low-GI or high-fibre diets, improve markers of longer term glycaemic control in humans, with the extent of the improvements subject to significant inter-study heterogeneity. There is a need for further large, well-designed trials.

**Keywords** Beans · Chickpeas · Diabetes · Glycaemia · Lentils · Peas · Pulses

### Abbreviations

%E	Per cent of energy
ADA	American Diabetes Association
GI	Glycaemic index
GP	Glycosylated blood proteins
FBG	Fasting blood glucose
FBI	Fasting blood insulin
HOMA-IR	HOMA of insulin resistance
IGT	Impaired glucose tolerance
MQS	Methodological quality score
SMD	Standardised mean difference

### Introduction

Diabetes has become one of the most important unmet prevention and treatment challenges. Despite the growing armamentarium of medications, which include five new classes since metformin was first approved in 1995 in the USA [1, 2], the combined prevalence of impaired glucose tolerance (IGT) and diabetes has surpassed 13% worldwide and continues to grow [3]. Although oral antihyperglycaemic agents have been shown to prevent the development of diabetes in high-risk individuals and to reduce the risk of microvascular complications in individuals with type 2 diabetes, they have failed to deliver the anticipated macrovascular benefits [4–6]. There are, however, a few exceptions [7, 8], such as the  $\alpha$ -glucosidase inhibitor acarbose, which has been shown to decrease incident diabetes in individuals with IGT [9] and cardiovascular events in individuals with type 2 diabetes [8].

Various dietary interventions share similarities with acarbose, which slows the rate of absorption such that dietary carbohydrate is converted into slowly absorbed or

‘lente’ carbohydrate, reducing postprandial glucose and insulin excursions and effectively lowering the glycaemic index (GI) of the diet. Dietary pulses (dried leguminous seeds, including chickpeas, beans, peas and lentils) are a good source of amylose starch, fibre, vegetable protein and antinutrients, such as phytates, phenols, lectins and enzyme inhibitors, some of which may act as natural inhibitors of both  $\alpha$ -amylase and  $\alpha$ -glucosidase [10]. In early studies, pulses were shown to result in exceptionally low glycaemic responses when fed to healthy volunteers [11], and in later experiments were demonstrated to possess a carbohydrate component that was more slowly digested than that of other foods such as cereals [12]. This property of slower absorption, which is common to  $\alpha$ -glucosidase inhibitors, makes pulses an important means of lowering the GI of the diet, a characteristic that has been exploited extensively in studies of low-GI diets [13–31], and would therefore be predicted to benefit glycaemic control and, by analogy with acarbose, CHD risk [8]. Based on limited clinical experimental evidence, the European (EASD) [32], Canadian (CDA) [33], and American (ADA) [34] diabetes associations recommend the consumption of dietary pulses as a means of optimising diabetes control through lowering the GI and increasing the dietary fibre content of the diet. To address the need for better clinical evidence to support nutrition recommendations for diabetes, we conducted a systematic review and meta-analysis of randomised clinical experimental trials investigating the effect of non-oil-seed dietary pulses, alone or as part of a low-GI or high-fibre diet, on markers of glycaemic control in people with and without diabetes.

### Methods

We followed the Cochrane Handbook for Systematic Reviews of Interventions Version 5.0.0, updated February 2008, for the planning and conduct of this meta-analysis [35]. The reporting followed the Quality of Reporting of Meta-analyses (QUOROM) guidelines [36].

**Study selection** We conducted a search of Medline (1950–), EMBASE (1980–), CINAHL (1982–) and the Cochrane Library (including The Cochrane Central Register of Controlled Trials [Clinical Trials; CENTRAL] database [1800–]) up to 22 May 2008, using the following search terms and Boolean operators: ‘beans OR bean OR lentils OR lentil OR chickpeas OR chickpea OR peas OR pea OR legume OR legumes OR leguminous OR lupin OR lupins OR vetch OR golden gram OR black gram OR green gram OR Bambara groundnut OR bambarra groundnut AND (glucose OR OGTT OR HbA<sub>1c</sub> OR A<sub>1c</sub> OR fructosamine OR insulin OR diabetes)’. The terms bean(s) (red and white

kidney, black, pinto, fava, and white [white, navy, haricot], black, brown, long, and mung, etc.) and pea(s) (yellow, green, split, black-eyed, cow, sugar, snap, snow, etc.) were used to capture specific pulses that have these words in their names. The search was restricted to human research studies. No limit was placed on language. Manual searches supplemented the database search strategy. We included randomised controlled experimental trials that investigated the medium- to long-term effect of supplementing the diet with non-oil-seed pulses in food form, alone or as part of a low-GI or a high-fibre dietary intervention, compared with a non-pulse control intervention on indices of glycaemic control in humans. Studies that were not randomised, had a follow-up period of <7 days, contained oil-seed pulses (peanuts or soy beans) or non-oil-seed pulses in extract or tablet/capsule form, lacked a suitable control intervention, or reported unbalanced or hyperenergetic comparisons, were excluded. We also excluded studies that contained multiple, concurrent interventions on the pulses' treatment arm, except studies in which low-GI or high-fibre dietary interventions included pulses. In cases where multiple publications with duplicate data existed for the same study, the article with the most information was included.

**Data extraction** Two investigators (J. M. W. Wong, A. Esfahani) independently extracted relevant data on study characteristics and outcomes using a standardised proforma. These data included information on study design (parallel, crossover, factorial, etc.); randomisation; blinding; sample size; participant characteristics (age, sex, BMI, diabetes status and presence of preexisting conditions [diabetes, metabolic syndrome, hypercholesterolaemia, coronary artery disease, CHD]); pulse type, form and dose; control diets; duration of follow-up and macronutrient profile of the background diet. Mean±SEM values for fasting (glucose, insulin, HbA<sub>1c</sub> and fructosamine concentrations and insulin sensitivity as assessed by HOMA of insulin resistance [HOMA-IR]) or postprandial (glucose and insulin concentrations and derived means, AUC and insulin sensitivity) variables were extracted. Studies that did not report SEM values had these values imputed from the SD, 95% CIs, *p* values, *T* and *F* statistics, using standard formulae [35]. If these data were unavailable, then SEM was calculated from an SD that was extrapolated by imputing the pooled SD from the other studies included in the meta-analysis, according to Furukawa et al. for unpaired data [37] and Elbourne et al. [38] for paired data. Studies that differed in the units used to report concentrations had their units converted using standard conversion factors. Where HOMA-IR was not reported, it was imputed using the formula of Matthews et al. [39]. Missing SEMs for HOMA-IR were imputed according to the standard formulae for functions of two variables [40]. The investigators also

assessed the quality of each study using the Heyland score [41], which assigns a score from 0 to 1 or 0 to 2 over nine categories of quality related to study design (randomisation, blinding, protocol analysis), sampling procedures (selection, compatibility, follow-up), and interventions (protocol description, co-interventions, crossovers) to give a total of up to 13 points (Electronic supplementary material [ESM] Table 1). Studies that reported 100% follow-up data were scored as intention-to-treat analyses. Designs with metabolic feeding control, where participants were provided with all foods on the study diets and all unused foods were returned for assessment of compliance, were also recorded as a measure of study quality. Disagreements were reconciled by consensus and, where necessary, by discussion with J. L. Sievenpiper or C. W. C. Kendall. Authors were not contacted to request additional information, except for one author who was contacted regarding the use of pulses in their study [23].

**Statistical analyses** Data were analysed using Review Manager (RevMan) 5.0.17 (Cochrane Library software, Oxford, UK). Three separate pooled analyses were conducted using the generic inverse variance method with inverse variance weighting for pulses alone, pulses in low-GI dietary interventions and pulses in high-fibre dietary interventions. Outcomes included glycosylated blood proteins (GP; HbA<sub>1c</sub> or fructosamine, with HbA<sub>1c</sub> preferred if the duration of the trial was longer than 12 weeks), fasting blood glucose (FBG) and insulin (FBI) concentrations and HOMA-IR, with change from baseline differences preferred to differences at study end between pulse and control interventions. Paired analyses were applied to all crossover trials, according to Elbourne et al. [38], necessitating the expression of data as standardised mean differences (SMD) with 95% CIs, where SMDs are interpreted as follows: <0.4, small effect size; 0.4–0.7, moderate effect size; and >0.7, large effect size. To address the possible introduction of a unit-of-analysis error by including trials with multiple intervention arms compared with a single control arm, we combined pulse-containing arms to create a single pair-wise comparison. Inter-study heterogeneity was tested by Cochrane's *Q* ( $\chi^2$  at a significance level of *p*<0.10) and quantified by *I*<sup>2</sup>, where *I*<sup>2</sup>≥50% is considered to be evidence of substantial heterogeneity and ≥75%, considerable heterogeneity [35]. Random effects models were applied for instances where heterogeneity was significant; fixed effects models were used otherwise. Sources of heterogeneity were investigated by sensitivity analyses, in which the effect of systematically removing each study was assessed. A priori subgroup analyses were also undertaken to investigate sources of heterogeneity, including the effect of participant category (normoglycaemia, type 1 diabetes, type 2 diabetes, any diabetes); pulse type (*Phaseolus vulgaris* [black, white, pinto, red and white kidney beans],

*Cicer arietinum* [chickpeas], *Vicia faba* [fava beans], *Vigna unguiculata* [black-eyed peas]), dosage ( $<$  and  $\geq 100$  g/day) [42] and physical form (whole, flour, fibre); duration of follow-up ( $\leq$  and  $>4$  weeks) and study quality according to the Heyland methodological quality score (MQS;  $<$  and  $\geq 8$ ) [41] on both the pooled effect estimates and their heterogeneity. Additional post hoc subgroup analyses were undertaken to investigate the effect of the macronutrient profile (carbohydrate  $<$  and  $\geq 55\%$  of energy [%E], fat  $\leq$  and  $>30\%$  E, protein  $<$  and  $\geq 20\%$  E) [32–34], and fibre content ( $<$  and  $\geq 40$  g/day) [32–34] of the background diet; GI ( $\leq$  and  $>70$  [bread scale]) of the test diet and difference in GI between the test and control diets ( $\Delta$  GI  $\leq$  and  $>15$ ) [43, 44]; level of feeding control (metabolic, non-metabolic), study design (parallel, crossover), and use of washout periods (yes, no) in crossover trials. Adjustment for multiple comparisons was made in the post hoc subgroup analyses using the Bonferroni procedure. Multivariate analyses were performed by meta-regression, where inverse variance-weighted multiple regression models were selected by All Possible Regression using the adjusted  $R^2$  criterion (Number Cruncher Statistical System software; NCS, Kaysville, UT, USA). Publication bias was investigated using Funnel plots.

**Role of the sponsor** The sponsors had no role in the design, conduct, or reporting of the present study.

## Results

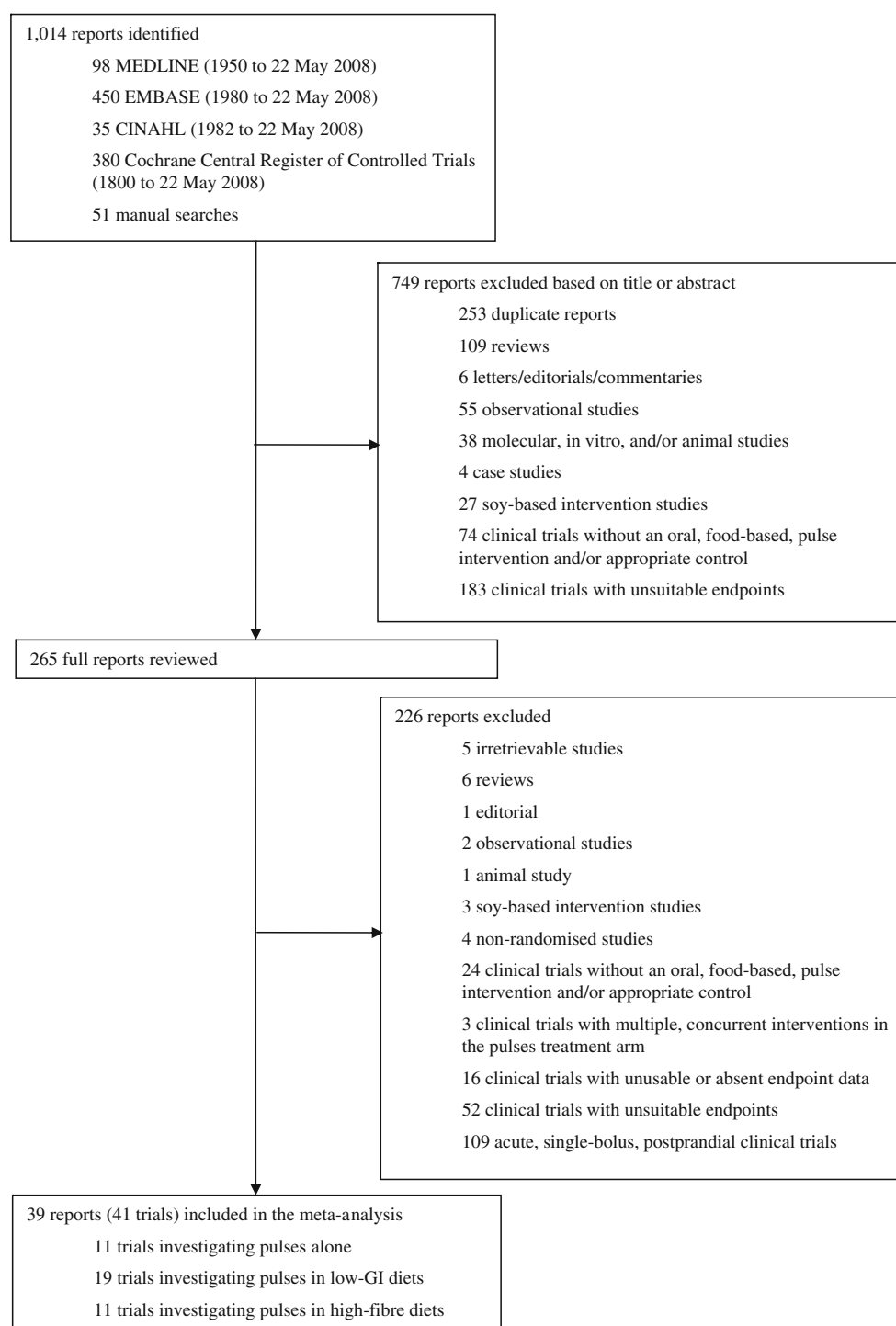
**Search results** Figure 1 shows the flow of the literature when the systematic search and selection strategies were applied. The initial search identified 1,014 reports, of which 749 were excluded based on the title or abstract, leaving 265 reports eligible for full review. Of these reports, 226 were excluded based on other criteria, leaving 41 trials (39 reports) that satisfied the inclusion criteria but did not meet the exclusion criteria, of which 11 trials (ten reports) investigated pulses alone, 19 trials (19 reports) investigated pulses in a low-GI dietary intervention, and 11 trials (ten reports) investigated pulses in a high-fibre dietary intervention.

**Trial characteristics** Tables 1, 2, and 3 show the characteristics of the 41 randomised controlled experimental trials included in each of the three meta-analyses: pulses alone [45–54], pulses in low-GI diets [13–31] and pulses in high-fibre diets [55–64]. The 11 trials (ten reports) investigating pulses alone (Table 1) were predominantly crossover in design (seven trials, five of which had a washout period), studied a total of 253 participants (type 2 diabetes,  $n=21$ ;

normoglycaemia and/or hypercholesterolaemia,  $n=232$ ) and had a mean sample size of 23. Pulse types included chickpeas, black-eyed peas and various beans (red and white kidney, black, pinto, fava and white [white, navy and haricot]), and the mean dosage was 152.1 g/day (range 15.5–465 g/day). The mean duration of study follow-up was 6.7 weeks (range 1–96 weeks). Background diets were largely high-carbohydrate, low-fat diets, consisting of 52%E carbohydrate (range 43–62%E), 18%E protein (range 15–20%E) and 29%E fat (range 20–37%E). Study quality assessed by the Heyland MQS ranged from 4–9, with only four of the 11 trials considered to be of high quality (MQS $\geq 8$ ). Only three trials were metabolically controlled.

The 19 trials investigating pulses in low-GI diets (Table 2) were predominantly crossover in design (12 trials, ten of which had a washout period), studied a total of 762 participants (type 2 diabetes,  $n=420$ ; type 1 diabetes,  $n=130$ ; normoglycaemia,  $n=212$ ) and had a mean sample size of 40. Pulse types included beans (white [white, navy and haricot], pinto, kidney, black, brown, long and mung), split peas, chickpeas, lentils and unspecified legumes; dosing information was not available. The mean GI of the pulse-containing low-GI diets was 66.9 (range 54.3–79). The mean duration of study follow-up was 14.3 weeks (range 2–52 weeks). Background diets were largely high-carbohydrate, low-fat diets, consisting of 50%E carbohydrate (range 36–60%E), 20%E protein (range 12–26%E) and 29%E fat (range 21–42%E). The mean fibre content of the pulse-containing low-GI diets was 33 g/day (range 12–72 g/day). Study quality assessed by the Heyland MQS ranged from 5 to 8, with ten of 19 trials considered to be of high quality (MQS $\geq 8$ ). Only four trials were metabolically controlled.

The 11 trials (ten reports) investigating pulses in high-fibre diets (Table 3) were predominantly crossover in design (nine trials, only three of which had a washout period) and studied a total of 641 participants (type 2 diabetes,  $n=140$ ; type 1 diabetes,  $n=89$ ; normoglycaemia,  $n=412$ ), with a mean sample size of 58. Pulse types included lima beans; green and yellow peas; green, white (white, navy, haricot), brown and pinto beans; chickpeas; lentils; unspecified legumes; and lupin bean kernel fibre; dosing information was not available. The mean fibre content of the pulse-containing high-fibre diets was 58 g/day (range 26.9–96.6 g/day). The mean duration of the follow-up period was 19.9 weeks (range 1.4–156 weeks). Background diets were largely high-carbohydrate, low-fat diets, consisting of a mean of 51.9%E carbohydrate (range 23–70%E), 19.6%E protein (range 13–62%E) and 29.8%E fat (range 10–55%E). Study quality assessed by the Heyland MQS ranged from 3 to 9, with six of 11 trials considered to be of high quality (MQS $\geq 8$ ). Only three trials were metabolically controlled.

**Fig. 1** Flow of the literature

**Pulses alone** Figure 2 shows forest plots for the effect of pulses alone on FBG, FBI, HOMA-IR and GP. Pooled analyses showed that FBG ( $-0.82$ , 95% CI  $-1.36$  to  $-0.27$ ) and FBI ( $-0.49$ , 95% CI  $-0.93$  to  $-0.04$ ) were significantly decreased by pulses alone, whereas HOMA-IR and GP were not. There was evidence of considerable ( $I^2 \geq 75\%$ ), significant ( $p < 0.10$ ) inter-study heterogeneity for all out-

comes. Systematic removal of each trial during sensitivity analyses did not alter the significance of the effect estimate or heterogeneity for any of the outcomes, with one exception. Removal of Fruhbeck et al. [49] from the analysis eliminated the evidence for significant inter-study heterogeneity for FBI ( $I^2 = 0\%$ ,  $p = 0.47$ ) without altering the significant benefit of pulses alone on this outcome.

**Table 1** Characteristics of randomised controlled experimental trials investigating pulses alone

Study	Sample size	Subject category <sup>a</sup>	Design <sup>b</sup>	Follow-up	Washout (Y/N) <sup>c</sup>	Quality (MQS) <sup>d</sup>	Metabolic (Y/N)	Interventions (pulse type) <sup>e</sup>	Pulse format	Dosage (g/day)	GI <sup>f</sup>	Fibre (g/day)	Soluble fibre (g/day)	CHO/protein/fat <sup>g</sup>
Anderson et al. 1984 [45]	20 (20M:0F)	N (HC)	P	21 d	–	7	Y	Pulses – Beans (W/P) Control – Oat bran	Whole	101	–	44	16	43:20:37 44:20:36
Wursch et al. 1988 [46]	6 (6M:0F)	N	C	7 d	Y	8	N	Pulses – Beans (WK) Control – Potato flakes	Flakes	114	–	≥23.6	–	–
Cobiac et al. 1990 [47]	20 (20M:0F)	N (HC)	C	4 wk	N	9	N	Pulses – Beans (W) Control – Spaghetti	Whole	377.1	–	22.5	11.9	46:18:32 47:17:32
Fleming et al. 1988 [48]	6 (6M:0F)	N	C	8 d	Y	9	Y	Pulses – Beans (RK) Control – Bran cereal/wheat bread	Whole	465	–	57	–	50:20:30 50:20:30
Frühbeck et al. 1997 [49]	40 (40M:0F)	N (HC)	P	30d	–	9	Y	Pulses – Beans (F) Pulses – Beans (F) Pulses – Beans (F) Control – Wheat flour	Flour Flour Flour (raw) –	90 90 90 –	– – – –	20 21 20 19	6 6 6 5	62:18:20 62:18:20 62:18:20 62:18:20
Jang et al. 2001 [50]	21 (21M:0F)	DM2 (HC, CAD)	P	16 wk	–	6	N	Pulses – Beans (B) Control – Refined rice	Flour	15.5	–	26	–	61:19:20 69:15:16
Nestel et al. 2004 [51]	19 (9M:10F)	N (HC)	C	6 wk	N	5	N	Pulses – Beans (B) Control – Refined rice	Flour	15.5	–	26	–	61:19:20 69:15:16
Winham et al. 2007 [52]	23 (10M:13F)	N (HC)	C	8 wk	Y	7	N	Pulses – Chickpeas Control – Wheat-based foods	Whole+flour	140	–	33	–	47:19:30 44:19:31
Winham et al. 2007 [53]	16 (7M:9F)	N	C	8 wk	Y	7	N	Pulses – Beans (W) Control – Carrots	Whole	126.5	–	25.5	–	50:17:32 50:17:34
Pittaway et al. 2007 [54]	27 (10M:17F)	N	C	5 wk	Y	4	N	Pulses – Beans (P) Pulses – Peas (BE) Control – Carrots Pulses – Chickpeas Control – Wheat-based foods	Whole Whole – Whole –	120 120 – 140 –	– – – – –	23 19 21 28.4 29.3	2 1 2 – –	51:15:32 53:16:31 50:16:33 44:17:34 43:18:34

<sup>a</sup> CAD denotes coronary artery disease; DM2, type 2 diabetes; HC, hypercholesterolaemia; N, normoglycaemia<sup>b</sup> Study designs were parallel (P) or crossover (C)<sup>c</sup> Washout applies to crossover studies only<sup>d</sup> Study quality was assessed by the Heyland MQS, where an MQS of ≥8 is considered high quality (range 0–13) [41]<sup>e</sup> B denotes black; BE, black-eyed; F, fava; P, pinto; RK, red kidney; W, white (white, navy, haricot); WK, white kidney<sup>f</sup> The GI of the pulses alone and control diets represent values on a bread scale. GI values that were reported on a glucose scale were converted to a bread scale by dividing them by 0.7 [43, 44]<sup>g</sup> Macronutrient profiles of the background diets on each intervention are given as %E from carbohydrate (CHO)/protein/fat

d, day; F, female; M, male; N, no; wk, week; Y, yes

**Pulses in low-GI diets** Figure 3 shows forest plots for the effect of pulses in low-GI diets on FBG, insulin, HOMA-IR and GP. Pooled analyses showed that the level of GP was significantly decreased ( $-0.28$ , 95% CI  $-0.42$  to  $-0.14$ ), whereas FBG, FBI and HOMA-IR were not. There was evidence of substantial ( $I^2 \geq 50\%$ ), significant ( $p < 0.10$ ) inter-study heterogeneity for all outcomes, with the exception of FBI ( $I^2 = 0\%$ ,  $p = 0.47$ ). Systematic removal of each trial during sensitivity analyses did not alter the significance of the effect estimate or heterogeneity for any of the outcomes, with one exception. Removal of Bouche et al. [23] eliminated the evidence for significant inter-study heterogeneity for HOMA-IR ( $I^2 = 20\%$ ,  $p = 0.29$ ) without altering the significant lack of effect of pulses in low-GI diets on this outcome.

**Pulses in high-fibre diets** Figure 4 shows forest plots for the effect of pulses in high-fibre diets on FBG, FBI, HOMA-IR and GP. Pooled analyses showed that FBG ( $-0.32$ , 95% CI  $-0.49$  to  $-0.15$ ) and GP ( $-0.27$ , 95% CI  $-0.45$  to  $-0.09$ ) were significantly decreased by pulses in high-fibre diets, whereas FBI and HOMA-IR were not. There was evidence of considerable ( $I^2 \geq 75\%$ ), significant ( $p < 0.10$ ) inter-study heterogeneity for FBG and GP only. Systematic removal of each trial during sensitivity analyses did not alter the significance of the effect estimate or heterogeneity for any of the outcomes.

**Univariate subgroup analyses** Univariate a priori and post hoc subgroup analyses explored the effect of sources of heterogeneity on the effect of pulses, alone or as part of low-GI or high-fibre dietary interventions, on FBG, FBI, HOMA-IR and GP (ESM Tables 2, 3, and 4). Inter-study heterogeneity for a benefit or lack of benefit of pulses was explained by only a few subgroup analyses, as defined by a loss of significance by Cochran's  $Q$  ( $\chi^2$ ) in one or more of the subgroup categories. Otherwise, effects were found to be dependent on various subgroup categories, with persistent substantial to high heterogeneity ( $p < 0.10$ ,  $I^2 \geq 50\%$ ) observed across the three pooled analyses. The most robust modifiers of benefit in a priori subgroup analyses across the three pooled analyses were type 2 diabetes (two of four outcomes in the analysis of pulses in low-GI diets, and two of two outcomes in the analysis of pulses in high-fibre diets), follow-up  $>4$  weeks (two of three outcomes in the analysis of pulses alone, and one of two outcomes in the analysis of pulses in high-fibre diets), and MQS  $<8$  (two of three outcomes in the analysis of pulses alone, and one of four outcomes in the analysis of pulses in low-GI diets). Specific to the pulses alone analysis, pulse species including *Phaseolus vulgaris* (black, white, pinto, red and white kidney beans), *Cicer arietinum* (chickpeas) and *Vicia faba* (fava beans) were also identified as significant

modifiers. In post hoc subgroup analyses, the most robust modifier of benefit across the three pooled analyses after adjustment by the Bonferroni procedure was metabolic feeding control (GP in the analysis of pulses in low-GI diets, and FBG and GP in the analysis of pulses in high-fibre diets). Otherwise, significant modifiers tended to be specific to each analysis. In the analysis of pulses in low-GI diets, the pulses-mediated decrease in GP was dependent on a GI of  $\leq 70$  on the low-GI diet and a difference in GI between interventions ( $\Delta$  GI) of  $>15$ . Similarly, in the high-fibre pooled analysis, the pulses-mediated decrease in FBG was dependent on a fibre intake of  $\geq 40$  g/day on the high-fibre diet intervention and the difference in fibre intake between interventions ( $\Delta$  fibre) of  $>25$  g/day. Macronutrient profile and study design variously modified effects in each analysis, with no clear patterns.

**Multivariate analyses** To explore the heterogeneity identified in univariate analyses, inverse variance weighted multiple regression models assessed the independent predictors of effect for outcomes in the full database combining all three pooled analyses (ESM Table 5). The models were significant ( $p < 0.05$ ) for FBG and FBI only, explaining 47% and 66% of the variation in these outcomes, respectively. Significant ( $p < 0.05$ ) independent predictors from strongest to weakest were identified as dietary carbohydrate (%E) ( $\beta = -0.034$ , partial  $R^2$  [adjusted for other variables] = 0.22), type 2 diabetes ( $\beta = -0.57$ , partial  $R^2 = 0.18$ ), pulses alone interventions ( $\beta = -0.71$ , partial  $R^2 = 0.18$ ), and dietary protein (%E) ( $\beta = 0.084$ , partial  $R^2 = 0.14$ ) for FBG; metabolic feeding control ( $\beta = 1.05$ , partial  $R^2 = 0.41$ ) and  $\Delta$  fibre ( $\beta = 0.056$ , partial  $R^2 = 0.39$ ) for FBI; and pulses alone interventions ( $\beta = 0.92$ , partial  $R^2 = 0.31$ ) and follow-up (days) ( $\beta = -0.0046$ , partial  $R^2 = 0.23$ ) for GP.

**Publication bias** Funnel plots for each outcome were inspected for the presence of publication bias in the full database combining all three analyses (ESM Fig. 1). There was limited evidence of funnel plot asymmetry for FBG and FBI with a tendency for publication of small trials reporting large effects and error estimates that favour pulses (three trials for FBG and one for FBI) but no small trials reporting large effects that favour control. No evidence of asymmetry was observed for HOMA-IR or GP.

## Discussion

The present three pooled analyses of 41 randomised controlled experimental trials, in a total of 1,674 participants, are supportive of diabetes association nutrition guidelines recommending the consumption of dietary pulses

**Table 2** Characteristics of randomised controlled experimental trials investigating pulses in low-GI diets

Study	Sample size	Subject category <sup>a</sup>	Design <sup>b</sup>	Follow-up	Washout (Y/N) <sup>c</sup>	Quality (MQS) <sup>d</sup>	Metabolic (Y/N)	Interventions (pulse type) <sup>e</sup>	Pulse format	Dosage (g/day)	GI <sup>f</sup>	Fibre (g/day)	Soluble fibre (g/day)	CHO/protein/fat <sup>g</sup>
Jenkins et al. 1987 [13]	6 (6M:0F)	N	C	2 wk	Y	8	Y	LGI – Lentils (R) Control – HGI diet	Whole	–	63	~71.5	–	59:20:21
Jenkins et al. 1988 [14]	8 (2M:6F)	DM2	C	2 wk	Y	8	N	LGI – Lentils Control – HGI diet	Whole	–	104	~57.75	–	62:19:20
Collier et al. 1988 [15]	14 (12M:2F)	DM1	C	6 wk	Y	8	N	LGI – Beans (U) Control – HGI diet	Whole	–	67.3	34	–	54:22:24
Brand et al. 1991 [16]	16 (10M:6F)	DM2	C	12 wk	Y	8	N	LGI – Beans (U) Control – HGI diet	Whole	–	90.5	28	–	53:21:26
Fontvielle et al. 1992 [17]	18 (12M:6F)	12 DM1, 6 DM2	C	5 wk	N	8	N	LGI – Beans (U), peas (U) Control – HGI diet	Whole	–	68.7	39	–	49:18:33
Wolever et al. 1992 [18]	6 (3M:3F)	DM2	C	6 wk	Y	7	Y	LGI – Beans (RK/U), chickpeas, lentils, peas (U) Control – HGI diet	Whole	–	81.5	23.5	–	47:16:37
Wolever et al. 1992 [19]	15 (7M:8F)	DM2	C	2 wk	Y	7	Y	LGI – Beans (RK/U), chickpeas, lentils, peas (U) Control – HGI diet	Whole	–	77	26	9.1	44:22:30
Frost et al. 1994 [20]	51 (36M:15F)	DM2	P	12 wk	–	6	N	LGI – Beans (U), pulse vegetables Control – HGI diet	Whole	–	90	26	6.5	46:19:31
Gilbertson et al. 2001 [21]	104 (52M:52F)	DM1	P	12 mo	–	6	N	LGI – Beans (W) Control – CHO exchange diet	Whole	–	54.3	26.9	13	46:18:36
Komindr et al. 2001 [22]	10 (0M:10F)	DM2	C	4 wk	N	8	Y	LGI – Beans (M/L) Control – HGI diet	Noodles/whole	–	91.4	26.7	12	45:19:36
Bouche et al. 2002 [23]	22 (22M:0F)	N	C	5 wk	Y	8	N	Control – Diabetic diet LGI – Legumes	Whole	–	58	34	10.2	57:20:23
Jimenez-Cruz et al. 2003 [24]	14 (6M:8F)	DM2	C	6 wk	Y	5	N	LGI – Beans (P) Control – HGI diet	Whole	–	86	33	9.8	57:20:23
										–	60	41	10.9	59:20:21
										–	87	37	9.5	59:20:21
										–	77	21	8	49:23:25
										–	82	14	6	44:22:32
										–	79	21	–	49:17:34
										–	80.7	21	–	49:17:34
										–	70	–	–	58:12:30
										–	106	–	–	58:12:30
										–	100	–	–	58:12:30
										–	58.6	31	–	39:20:38
										–	104.3	19	–	42:18:37
										–	62.9	34	–	60:21:23
										–	80	25	–	64:18:20

Jimenez-Cruz et al. 2004 [25]	8	DM2	C	3 wk	Y	6	N	LGI – Beans (P/U), peas (U)	Whole	–	85.7	53	–	51:23:26
Frost et al. 2004 [26]	55 (48M:7F)	N (CHD)	P	12 wk	–	8	N	Control – HGI diet LGI – Beans (U); legumes	Whole	–	102.9	30	–	54:18:27
Rizkalla et al. 2004 [27]	12 (12M:0F)	DM2	C	4 wk	Y	7	N	Control – HGI diet LGI – Beans (M/W), lentils; chickpeas	Whole	–	71	20	–	49:18:31
McMillan-Price et al. 2006 [28]	129 (31M:98F)	N	P	12 wk	–	7	N	Control – HGI diet LGI – Legumes (high- CHO diet)	Whole	–	81	15	–	47:18:32
								LGI – Legumes (reduced-CHO, high-Pro diet)	Whole	–	55.7	34	–	36:21:37
								Control – HGI diet (high CHO)	Whole	–	101.9	21	–	39:21:39
								Control – HGI diet (reduced-CHO, high-Pro)	Whole	–	64.3	–	–	56:19:22
								LGI – Legumes	Whole	–	62.9	–	–	40:26:29
Wang et al. 2007 [29]	72 (36M:36F)	DM2	P	5 mo	–	8	N	Control – Food exchange list control diet	Whole	–	100	–	–	60:18:19
Ma et al. 2008 [30]	40 (19M:21F)	DM2	P	1 yr	–	8	N	LGI – Legumes	Whole	–	84.3	–	–	42:28:27
Wolever et al. 2008 [31]	162 (74M:88F)	DM2	P	1 yr	–	7	N	Control – ADA diet LGI – Beans (W/RK/ B/P/Mx), chickpeas, peas (S), lentils (R)	Whole	–	76.6	12.2	–	38:20:42
								Control – HGI diet	Whole	–	80.4	12.1	–	39:20:43
								Control – Low-CHO diet	Whole	–	78.7	36.3	–	52:21:27
									Whole	–	90.3	21	–	47:20:31
									Whole	–	84.9	23	–	39:19:40

<sup>a</sup>DM1 denotes type 1 diabetes; DM2, type 2 diabetes; N, normoglycaemia

<sup>b</sup>Study designs were parallel (P) or crossover (C)

<sup>c</sup>Washout applies to crossover studies only

<sup>d</sup>Study quality was assessed by the Heyland MQS, where an MQS of  $\geq 8$  is considered high quality (range 0–13) [41]

<sup>e</sup>CHO denotes carbohydrate; HGI, high GI; L, long; LGI, low GI; M, mung; Mx, mixed; P, pinto; Pro, protein; R, red; RK, red kidney; S, split U, unspecified; W, white (white, navy, haricot)

<sup>f</sup>The GI of the low-GI and control diets represent values on a bread scale. GI values that were reported on a glucose scale were converted to a bread scale by dividing them by 0.7 [43, 44]

<sup>g</sup>Macronutrient profiles of the background diets on each intervention are given as %E from carbohydrate (CHO)/protein/fat mo, month; yr, year; wk, week

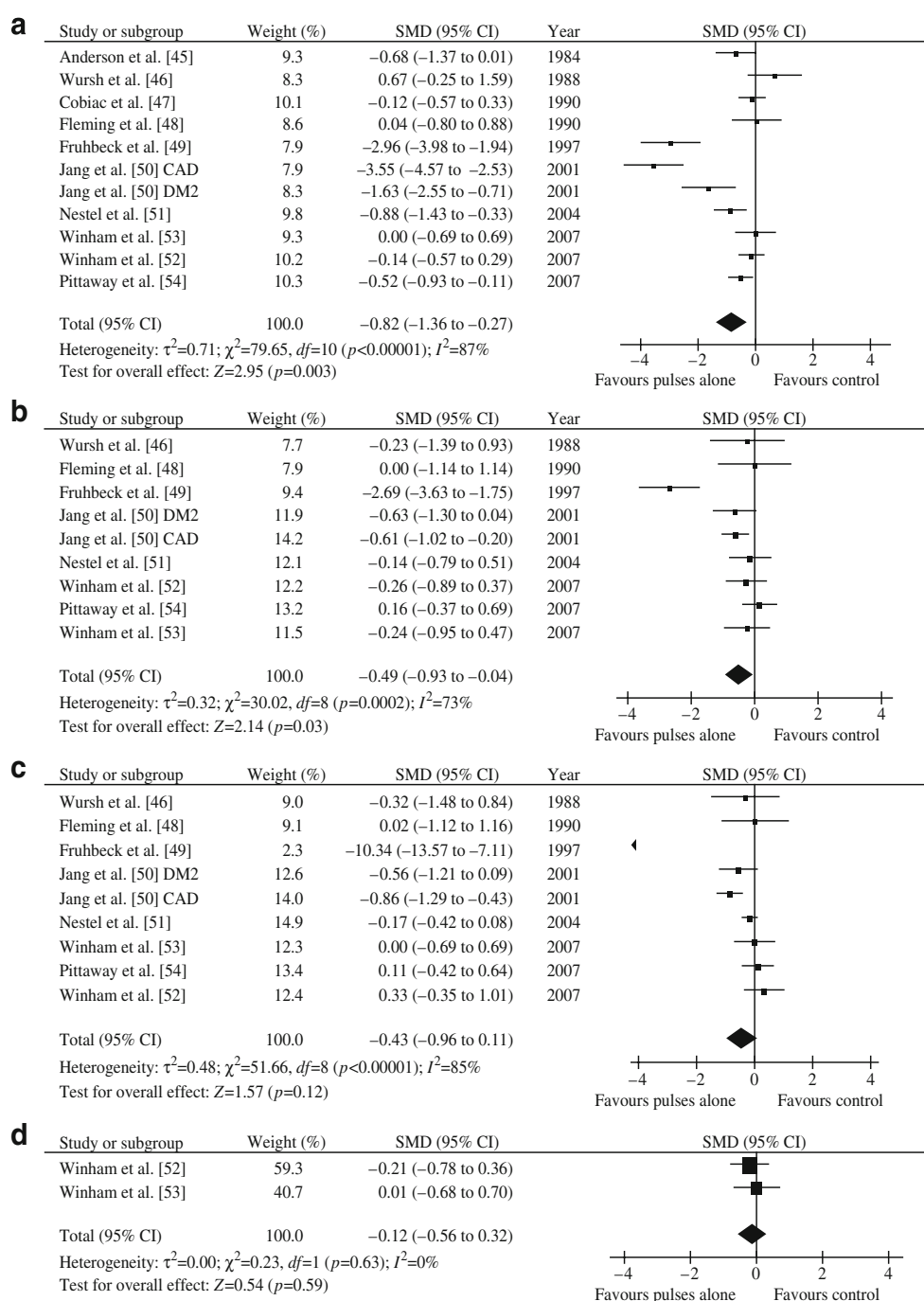
**Table 3** Characteristics of randomised controlled experimental trials investigating pulses in high-fibre diets

Study	Sample size	Subject category <sup>a</sup>	Design <sup>b</sup>	Follow-up	Washout (Y/N) <sup>c</sup>	Quality (MQS) <sup>d</sup>	Metabolic (Y/N)	Interventions (pulse type) <sup>e</sup>	Pulse format	Dosage (g/day)	GI <sup>f</sup>	Fibre (g/day)	Soluble fibre (g/day)	(CHO/protein:fat) <sup>g</sup>
Simpson et al. 1981 [55]	18 (10M:8F)	DM2	C	6 wk	N	9	N	HF – Beans (RK/W/Bu) Control – Low-fibre diet	Whole	–	–	96.6	–	61:21:18
	9 (4M:5F)	DM1	C	6 wk	N	9	N	HF – Beans (RK/W/Bu) Control – Low-fibre diet	Whole	–	–	17.6	–	40:21:39
Kimmonth et al. 1982 [56]	10 (6M:4F)	DM1	C	6 wk	N	8	N	HF – Beans (W/U), unrefined diet Control – Low-fibre diet	Whole	–	–	96.6	–	61:21:18
Riccardi et al. 1984 [57]	14	6 DM1, 8 DM2	C	10 d	N	8	Y	HF – Beans (Bu), legumes, high-CHO diet Control – Low-fibre, high-CHO diet	Whole	–	–	17.6	–	40:21:39
Karlstrom et al. 1987 [58]	15 (8M:7F)	DM2	C	3 wk	N	5	N	HF – Peas (G/Y), beans (G/B/W), chickpeas, lentils Control – Low-fibre diet	Whole	–	–	56.1	–	55:15:30
O'Dea et al. 1989 [59]	10 (10M:0F)	DM2	C	2 wk	Y	3	N	HF – Lentils, beans (U), high-CHO diet Control – Low-fibre, high-CHO diet	Whole	~200	–	45	–	65:24:10
								Control – Low-fibre, low-CHO, high-fat diet Control – Very low-fibre, low-CHO, low-fat diet	–	–	–	20	–	63:25:12
Anderson et al. 1991 [60]	10 (9M:1F)	DM1	C	4 wk	Y	6	Y	HF – Beans (RK/P/G), peas, high-CHO diet Control – Low-fibre, low-CHO diet	Whole	–	–	13	–	23:62:15
Singh et al. 1996 [61]	450	374N, 76 DM2	P	3 yr	–	7	N	HF – Peas, beans (RK), ADA diet Control – Low-fibre ADA diet	Whole	–	–	72.3	23.7	70:20:10
Chandalia et al. 2000 [62]	13 (12M:1F)	DM2	C	6 wk	Y	8	Y	HF – Beans (L), legumes Control – Low-fibre ADA diet	Whole	–	–	11	3.1	39:20:41
Giacco et al. 2000 [63]	54 (21M:33F)	DM1	P	24 wk	–	8	N	HF – Legumes Control – Low-fibre diet	Whole	~200	70	36.7	–	60:15:26
Hall et al. 2005 [64]	38 (38M:0F)	N	C	28 d	–	7	N	HF – Lupin kernel fibre Control – Low-fibre diet	Pulse fibre	–	90	15.4	–	57:15:29
										–	–	46	–	55:15:30
										–	–	24	8	55:15:30
										–	–	–	–	52:20:28
										–	–	–	–	55:17:28
										–	–	–	–	49:17:27
										–	–	24	–	52:17:26

<sup>a</sup> DM1 denotes type 1 diabetes; DM2, type 2 diabetes; N, normoglycaemia<sup>b</sup> Study designs were parallel (P) or crossover (C)<sup>c</sup> Washout applies to crossover studies only<sup>d</sup> Study quality was assessed by the Heyland MQS, where an MQS of  $\geq 8$  is considered high quality (range 0–13) [41]<sup>e</sup> Br denotes brown; Bu, butter; CHO, carbohydrate; G, green; L, lima; P, pinto; RK, red kidney; U, unspecified; W, white (white, navy, haricot); Y, yellow<sup>f</sup> The GI of the low-GI and control diets represent values on a bread scale. GI values that were reported on a glucose scale were converted to a bread scale by dividing them by 0.7 [43, 44]<sup>g</sup> Macronutrient profiles of the background diets on each intervention are given as %E from carbohydrate (CHO)/protein/fat

d, day; wk, week; y, year

**Fig. 2** Pooled effect of interventions involving pulses alone on the following markers of glycaemic control: FBG (a), FBI (b), HOMA-IR (c) and GP (d). SMDs (boxes) with 95% CIs (lines) for each study, study weights represented by the size of each box, and pooled effect estimates (diamonds) with 95% CI represented by the width of the diamonds were generated using the general inverse variance method with random (FBG, FBI, HOMA-IR) and fixed (GP) effects models. The article by Jang et al. 2001 [50] reported two separately controlled trials, one denoted as DM2 in patients with type 2 diabetes and coronary artery disease, and one denoted as CAD in patients with coronary artery disease alone

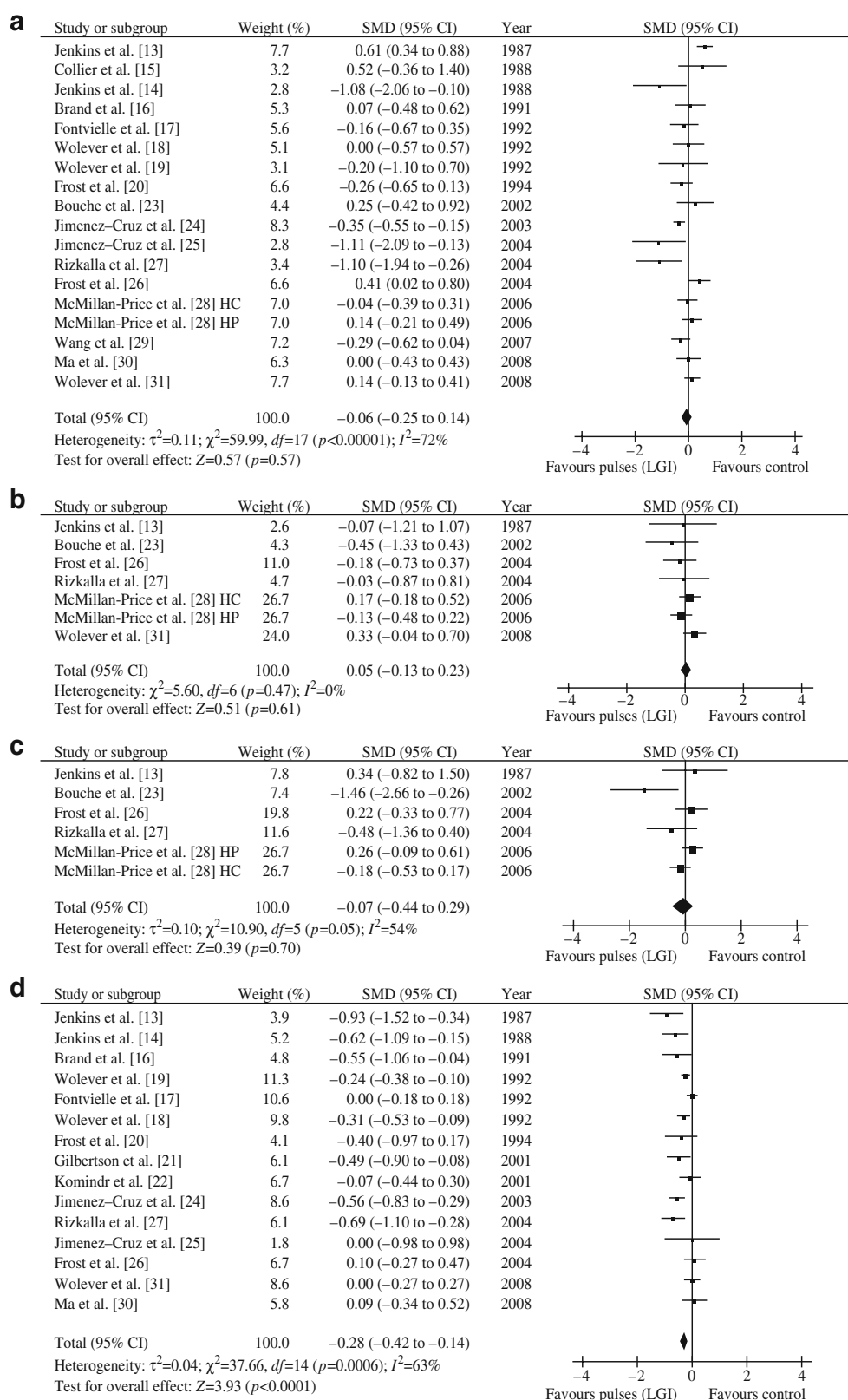


as a means of optimising diabetes control. Pulses given alone or as part of low-GI or high-fibre diets were found to improve the main markers of long-term glycaemic control, namely, GP ( $\text{HbA}_{1c}$  or fructosamine) by pulses in low-GI and high-fibre diets and FBG by pulses given alone or as part of high-fibre diets. The magnitude of the benefit conferred in terms of the normalisation of  $\text{HbA}_{1c}$  approached the 0.58–0.77% reduction reported for acarbose in two recent meta-analyses in type 2 diabetes [8, 65], which was found to be related to reduced cardiovascular events [8]. If we calculate the mean absolute reduction in  $\text{HbA}_{1c}$  in people with type 2 diabetes in

the present analysis (mean difference  $\text{HbA}_{1c} = \text{SMDGP} \times \text{pooled SD } \text{HbA}_{1c}$ ), then the SMD reductions in GP for pulses in low-GI and high-fibre diets correspond to an absolute reduction in  $\text{HbA}_{1c}$  of ~0.48%, a reduction that exceeds the clinically meaningful threshold ( $\geq 0.3\%$ ) proposed by the US Food and Drug Administration [66] and lies at the lower limit of efficacy expected for oral agents [1]. Observed benefits, however, were complicated by significant inter-study heterogeneity.

Possible modifiers of the primary effects were explored by planned and post hoc subgroup analyses, which

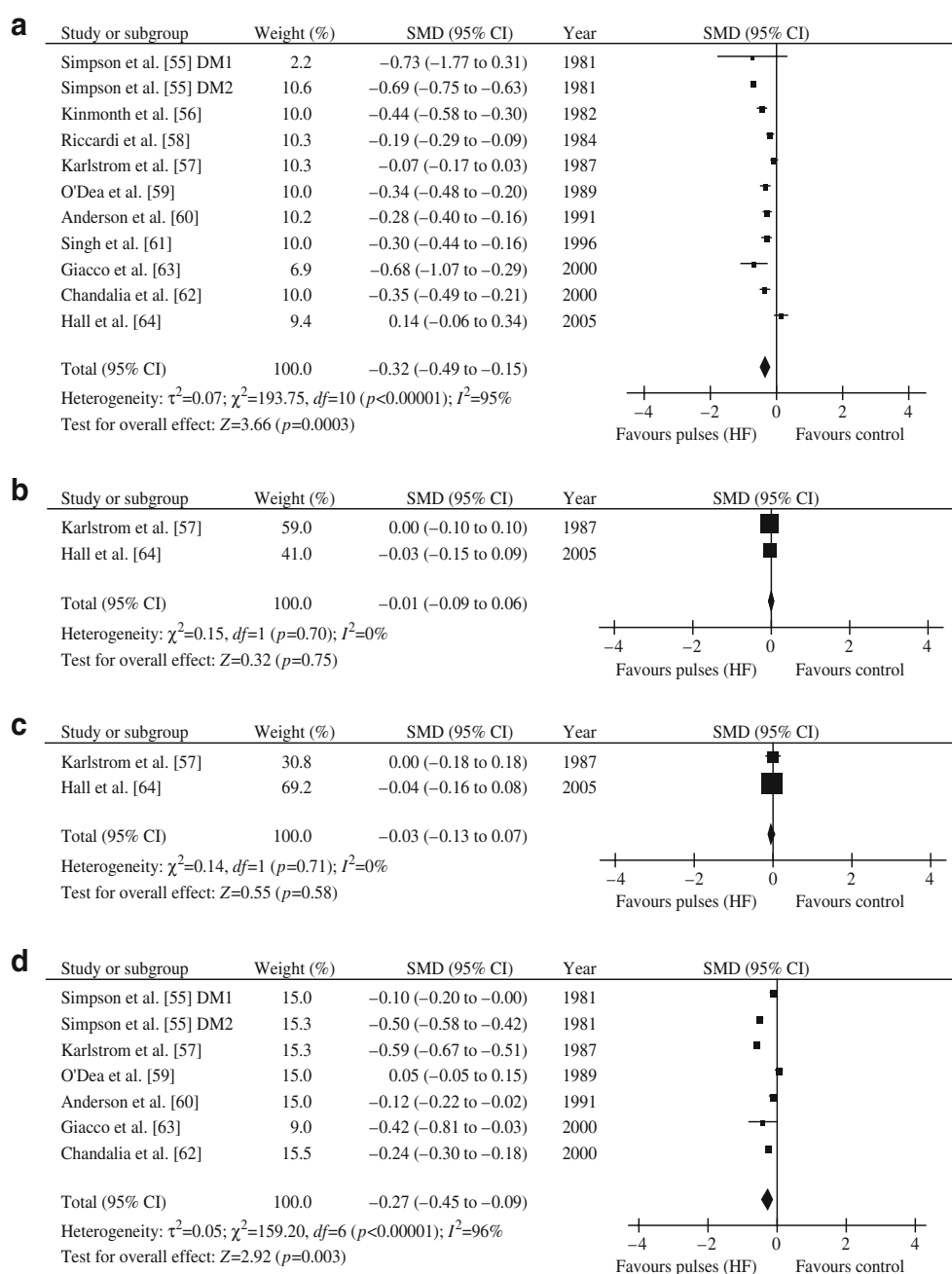
**Fig. 3** Pooled effect of pulses in low-GI (LGI) diets on the following markers of glycaemic control: FBG (**a**), FBI (**b**), HOMA-IR (**c**) and GP (**d**). SMDs (boxes) with 95% CIs (lines) for each study, study weights represented by the size of each box, and pooled effect estimates (diamonds) with 95% CI represented by the width of the diamonds were generated using the general inverse variance method with random (FBG, HOMA-IR, GP) and fixed (FBI) effects models. The article by McMillan-Price et al. 2006 [28] reported two separately controlled trials, one denoted as HC, a high-carbohydrate intervention, and one denoted as HP, a high protein intervention



identified several conditions under which benefits were gained, maintained or lost, despite the heterogeneity remaining largely unexplained. Diabetes status was found to be one modifier. A benefit of pulses for FBG and/or GP

was seen in either type 2 diabetes or any diabetes for all three pooled analyses. In multiple regression models, type 2 diabetes was identified as an independent predictor of FBG, explaining 26% of the variation in this outcome. In

**Fig. 4** Pooled effect of pulses in high-fibre (HF) diets on the following markers of glycaemic control: FBG (**a**), FBI (**b**), HOMA-IR (**c**) and GP (**d**). SMDs (boxes) with 95% CIs (lines) for each study, study weights represented by the size of each box, and pooled effect estimates (diamonds) with 95% CI represented by the width of the diamonds were generated using the general inverse variance method with random (FBG, GP) and fixed (FBI, HOMA-IR) effects models. The article by Simpson et al. 1981 [55] reported two separately controlled trials, one denoted as DM1 in patients with type 1 diabetes, and one denoted as DM2 in type 2 diabetes



contrast, the data in healthy normoglycaemic individuals were conflicting. There was a decreasing effect of pulses alone on FBG in the analysis of pulses alone, whereas there was an increasing effect of pulses in low-GI diets on FBG in healthy normoglycaemic individuals. Heterogeneity was reduced only for type 1 diabetes, remaining substantial to considerable across other categories of participants.

Glycaemic benefits also appeared to be modified by pulse type. The strongest evidence for benefit was for pulse interventions containing chickpeas (*Cicer arietinum*), which decreased FBG in the pulses alone analysis with very low heterogeneity. Potential benefit was also seen for

*Phaseolus vulgaris* (black, white, pinto, red and white kidney beans), but interpretation of these results was complicated by high heterogeneity, and for *Vicia faba* (fava beans), but the data were from only one study. Disentangling an effect of pulse type was made difficult by differences in physical form. In the analysis of pulses alone, the benefit of pulses was significant only for pulses as flakes/flour for FBI and HOMA-IR, and the effect estimates were much larger in favour of pulses as flakes/flour for FBG. In contrast, benefit was seen only for whole pulses in the analysis of pulses in high-fibre diets. Univariate and multivariate analyses of the effect of pulse type in low-GI diets and high-fibre diets were

not undertaken owing to confounding from deficiencies in reporting and the simultaneous use of multiple pulse types. More trials are needed to define optimal pulse types and physical form.

Another possible modifier of benefit was pulse dose. Benefits of pulses alone for FBG and FBI and HOMA-IR were unexpectedly seen only at doses less than the serving recommended by the American Heart Association (1/2 cup/day  $\approx$  100 g/day) [42]. Although the interactive effects of pulse dose could not be assessed in the analyses of pulses in low-GI and high-fibre diets, the level of dietary GI or dietary fibre achieved during the trials can be considered as proxies for the dose. If we take this approach, then only in the context of trials that achieved a true low-GI intervention (GI < 70) or large GI treatment differences ( $\Delta$  GI > 15) did pulses in low-GI diets decrease GP and show a tendency for lowering FBG. Similarly, only at fibre intakes at the EASD recommended level (> 40 g/day) [32], did pulses in high-fibre diets decrease FBG. The difference in fibre intake between the pulse and control interventions ( $\Delta$  fibre), however, was identified as a positive independent predictor of FBI, explaining 40% of the variation in this outcome in multiple regression models. These discrepancies in the data for dose and its proxies need to be reconciled by well-designed, long-term dose–response trials.

Follow-up was a further possible modifier. The benefit of pulses alone for FBG and FBI and of pulses in high-fibre diets for GP was only seen when the duration of follow-up was > 4 weeks. Follow-up was identified as a negative independent predictor of GP, explaining 23% of the variation in this outcome in multiple regression models. These data suggest that an adaptation period may be needed to realise the benefits of pulses. Trials with sufficiently long-term follow-up are needed to confirm the sustainability of benefits.

Other modifiers may have related to aspects of the macronutrient profile of background diets. Although macronutrient profile was not a reliable modifier of the effect of pulses in univariate subgroup analyses with both high and low carbohydrate (< and  $\geq$  55%E), fat ( $\leq$  and > 30%E) and protein (< and  $\geq$  20%E) diets variously modifying the effect of pulses in the pulses in low-GI and high-fibre diets analyses, both carbohydrate and protein were identified as independent predictors of FBG in multiple regression models. Dietary carbohydrate was inversely associated with FBG, explaining 22% of its variation, whereas protein was positively associated with FBG, explaining 14% of its variation.

Finally, aspects of study design and quality were identified as potential modifiers of benefit. Although a crossover or parallel design itself was not a reliable modifier of the effect of pulses, benefit was only seen for crossover trials with washout periods for FBG in the pulses

in high-fibre diets analysis and GP in the pulses in the low-GI diets analysis. Metabolic feeding control also appeared to be driving some of the effects. Benefit was only seen in trials with metabolic feeding control for GP in the pulses in low-GI diets analysis and both FBG and GP in the pulses in high-fibre diets analysis. It was also identified as an independent predictor of FBI in multiple regression models, explaining 42% of the variation in this outcome. Overall methodological quality of the trials appeared to exert a further influence. Only trials that did not meet the Heyland criteria for high quality (MQS < 8) [41] exerted a benefit in terms of FBG and GP, with high heterogeneity observed in all three pooled analyses. There was one exception for the effect on GP in the analysis of pulses in high-fibre diets, where the opposite was true. MQS, however, was not identified as an independent predictor for any outcome in multiple regression models. To address the effect of designs and quality on reproducibility, these data suggest the need for high-quality, ‘real world’ effectiveness (non-metabolic) trials with careful measures of compliance, side effects and acceptability, and designs to eliminate potential sequence and carryover effects.

Our data support the proposed main mechanism of action of pulses. Data from acute and chronic feeding studies suggest that pulses reduce both postprandial blood glucose and insulin excursions [46, 48, 50, 51], effectively lowering the GI of the diet through slowed absorption [11, 67, 68]. A direct benefit on measures of insulin sensitivity has also been reported for pulses alone using surrogate HOMA indices [50, 51] and has been implied by studies of pulses in low-GI diets using the euglycaemic–hyperinsulinaemic clamp [27]. The significant decrease in FBI in the presence of decreased FBG seen in the pooled analysis of pulses alone suggests an insulin-sensitising effect of pulses. However, this combination did not translate into a significant reduction in HOMA-IR, although the direction of the effect favoured pulses. These markers pertain only to the fasting state and are considered to be more closely related to hepatic insulin sensitivity than whole body insulin sensitivity [69]. It is more likely that postprandial measures of insulin sensitivity would show a benefit. This suggestion is supported by the larger effect sizes seen for insulin reduction in post-oral-glucose or mixed-meal profiles (120–360 min) following chronic feeding of pulses alone [46, 48, 50, 51]. These proposed mechanisms, however, need to be confirmed by euglycaemic–hyperinsulinaemic clamp studies.

Several caveats of our analysis need to be considered. First, our pooled analyses did not include long-term, postprandial glycaemic control outcomes. Although numerous studies showing acute, postprandial [44] and ‘second meal’ effects [68] can be found in the literature, there were only a handful of trials reporting long-term, postprandial data in each pooled analysis, and these data were derived from a

variety of diverse protocols, precluding meta-analyses. As postprandial glycaemic outcomes tend to be more sensitive markers of glycaemic control than are fasting outcomes [70], it can be argued that the effect estimates may have been larger, and thus more likely to be significant, with possibly less inter-study heterogeneity. Second, only published studies were included in the present analyses, making a publication bias possible. Funnel plots for the pooled analysis of pulses alone showed particular evidence of possible bias favouring the publication of small, positive trials with large effect and error estimates. Finally, heterogeneity remained unsatisfactorily explained by univariate subgroup analyses in the three pooled analyses, compromising the quality of the evidence. Some steps were taken to address this limitation. Exploratory multiple regression models were used to explain >45% of the variability in FBG and FBI and to identify significant independent predictors for GP, although we readily acknowledge the serious limitations in performing analyses with so few observations (trials). Random effects models were also used to incorporate the heterogeneity into the analyses, and systematic removal of each study during sensitivity analyses did not alter conclusions.

In conclusion, dietary non-oil-seed pulses may modestly improve medium to longer term glycaemic control through a possible insulin-sparing mechanism, when used alone or in combination with other dietary interventions to lower the GI or increase the dietary fibre of the diet. This benefit appears to be especially true when the pulse type is chickpeas, individuals have diabetes, diets are metabolically controlled, or the duration of follow-up is >4 weeks, conditions for which the best evidence exists. Although these factors do not offer robust explanations for the high level of heterogeneity, a benefit of pulses added to the diets of individuals with diabetes cannot be excluded. The heterogeneity in the pooled data highlight the need for large, well-designed, carefully conducted randomised controlled trials to resolve issues raised by the subgroup analyses of differential benefits related to diabetes status, pulse type, dose, physical form, follow-up, study quality, macronutrient profile of background diets, feeding control and design. A broader benefit of dietary pulses on metabolism must also be considered. Future analyses are planned to assess the effect of pulses on other areas of metabolic control, including lipids, body weight and blood pressure. The effect of pulses in other dietary interventions, such as Mediterranean or vegetarian diets, also needs to be studied.

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