Online Supplemental Material

Re: Chiavaroli et al. The Effect of Low-Glycemic Index/Load Dietary Patterns on Glycemic Control and Cardiometabolic Risk Factors in Diabetes: A Systematic Review and Meta-Analysis of Randomized Controlled Trials

Supplemental Methods

METHODS

We followed the Cochrane Handbook for Systematic Reviews of Interventions (version 6.1)(1) for the conduct and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines(2) (**Supplemental Table S1**) for the reporting of this systematic review and meta-analysis. The protocol was registered at ClinicalTrials.gov (NCT04045938).

Search strategy and selection criteria

Supplemental Tables S2-S3 shows the search strategy(2). Validated filters from the McMaster University Health Information Research Unit were applied to limit the database search to controlled studies only (3). We searched MEDLINE, EMBASE, and the Cochrane Central Register of Controlled Trials through May 13, 2021. These searches were supplemented with manual searches of the reference lists from included trials.

We included randomized controlled trials with a follow-up duration ≥3 weeks investigating the effect of low-GI or low-GL diets on measures of glycemic control, blood lipids, adiposity, blood pressure, or inflammation in those with type 1 or type 2 diabetes. We excluded studies that were multi-modal with co-interventions (i.e., trials which were designed in such a way that the effect of GI or GL could not be isolated), had non-energy matched control, were in pregnant or breastfeeding women, or did not report viable endpoint data. No restrictions were placed on language.

For the intervention to be eligible in low-GI trials, it had to explicitly self-identify as low-GI, include low-GI foods and had to have an expected difference in GI between the intervention and control groups. For the intervention to be eligible in low-GL trials, the GL intervention had to explicitly self-identify as low-GL and be described as being reduced in both carbohydrate quantity and glycemic index. We selected a follow-up duration of ≥3 weeks based on FDA minimum study duration for cholesterol reduction of ≥3-weeks(4) and the WHO minimum study duration for weight change of ≥2-weeks(5). We felt that this was sufficient for all outcomes. We even felt it was sufficient for HbA1c, which is usually assessed clinically at 3-months, since meaningful reductions have been observed even at 3 weeks based on an analysis in patients with type 1 and type 2 diabetes where in the first 35.2 days, the rate of fall of HbA1c was >0.1% per day during intensive therapy(6). For all reports which passed through title and abstract review, at least 2 investigators (LC and DL, AA or AC) independently reviewed the full text using the inclusion criteria. Reviewer discrepancies were resolved by consensus or arbitration by the senior author (JLS).

Data extraction

Two investigators (LC and DL, AA or AC) independently reviewed and extracted relevant data from each included report using a standardized form including sample size, participant characteristics, study setting, design, feeding control, intervention, control, GI and GL dose (glucose scale) during intervention and control, dietary macronutrient, energy balance, follow-up, funding source and outcome data. When

GL was not reported but GI and carbohydrate (g/d) were, we calculated GL from these values as GI*carbohydrate (g/d) /100. If carbohydrate was reported as %E, we calculated g/d using total calories when available, otherwise assumed a 2000kcal diet. Authors were contacted for missing data. In the absence of outcome data and inability to obtain the original data from authors, values were extracted from figures using Plot Digitizer(7) where available. Discrepancies were resolved through consensus.

Risk of bias assessment

Included trials were independently assessed by two investigators (LC and DL, AA or AC) for risk of bias using the Cochrane Risk of Bias Tool(1). Assessment was done across 5 domains of bias (sequence generation, allocation concealment, blinding, incomplete outcome data and selective reporting). Risk of bias was assessed as either low (proper methods taken to reduce bias), high (improper methods creating bias) or unclear (insufficient information provided) for each of the 5 domains of bias according to our criteria for judging risk of bias in the Cochrane Risk of bias assessment tool (see **Supplemental Table S4**). Reviewer discrepancies were resolved by consensus or arbitration by the senior author (JLS).

Outcomes

The prespecified primary outcome was difference in HbA1c. Our EASD clinical practice guidelines committee chose HbA1c as the primary outcome because the glycemic index was designed specifically to target glycemic control through a reduction in postprandial glycemia and HbA1c is the principal target of glycemic control in those with diabetes according to clinical practice guidelines globally. Secondary outcomes included other markers of glycemic control (fasting glucose, fasting insulin); blood lipids (LDL-C, non-HDL-C, apo B, HDL-C, triglycerides); adiposity (body weight, body mass index (BMI), waist circumference), blood pressure (systolic blood pressure (SBP) and diastolic blood pressure (DBP)), and inflammation (C-reactive protein (CRP)). Change in anti-hyperglycemic medications or insulin, adverse events and intervention acceptability were added as a post-hoc secondary outcomes that were assessed narratively.

Data analyses

All analyses were conducted using STATA software, version 16.1 (StataCorp, College Station, TX, USA). Separate pooled analyses of study trial comparisons were conducted for each outcome using the generic inverse variance method with DerSimonian and Laird random-effects meta-analyses(8). Mean differences (MDs) between the intervention and control arms and their respective variance terms were extracted and used as the basis for analysis for each trial. If not provided, they were derived from available data using published formulas (1). When median data was reported, they were converted to mean data with corresponding variances using established methods (9-11). When no variance data was available, the standard deviation (SD) was borrowed from a trial similar in size, participants and nature of intervention. MDs and standard errors (SEs) were computed using change-from-baseline differences in preference over end-differences. For trials with multiple follow up timepoints, our approach was to follow the primary analysis plan of the included trials. For example, if a trial reported an average of multiple timepoints as the primary timepoint of interest, we used this average as the endpoint to assess the outcome. Where the analysis plan was not specific about the primary timepoint of interest, we used

the longest timepoint from baseline reported. For crossover trials and for within arm changes in parallel trials,(12) a correlation coefficient of 0.5 was used in pairwise analysis to calculate SEs(12-14). To mitigate a unit-of-analysis error, when arms of trials with multiple intervention or control arms were used more than once, the corresponding sample size was divided accordingly(1). Non-HDL-C values that were not reported were derived by subtracting HDL-C from total cholesterol values with SEs derived from HDL-C and total cholesterol variance data using the inverse variance law (15). In trials where the change in BMI was not reported, but where body weight was reported, if baseline BMI was available, then these data were used to calculate the height that could then be used to calculate the end BMI and change in BMI. The change in BMI variance was imputed using published formula(1) and a correlation coefficient of 0.5(12-14).

Data were expressed as MDs with 95% confidence intervals (CIs). Heterogeneity was assessed using the Cochran Q statistic and quantified using the I2 statistic. Significance for heterogeneity was set at $P<0.10$ with an $I2>50\%$ considered to be evidence of substantial heterogeneity(1). Sources of heterogeneity were explored using sensitivity and subgroup analyses. Sensitivity analyses were performed in which each individual trial comparison was removed from the meta-analysis and the effect size recalculated to determine whether a single trial comparison exerted an undue influence. A trial comparison whose removal explained the heterogeneity, changed the significance of the effect or altered the effect size by ≥ one minimally important difference [MID] (Supplemental Table S5) was considered an influential comparison. Sensitivity analyses were also performed using correlation coefficients of 0.25 and 0.75 to determine whether the overall results were robust to the use of different correlation coefficients. Where ≥10 trial comparisons were available, a priori subgroup analyses were conducted using random-effects meta-regression where heterogeneity of effect estimates (effect modification) was explored using prespecified subgroups (diabetes type, study design, follow-up duration, comparator diet, baseline measurements, diabetes duration and domains of risk of bias)(16, 17). Additional post-hoc subgroup analyses were conducted by age, energy balance, feeding control, test GI/GL (absolute in-trial achieved GI or GL in the low-GI/GL diets), difference in GI/GL (test-control), and funding source. Further post-hoc categorical subgroup analyses were conducted by presence of a wash-out period for crossover trials and continuous subgroup analyses by test fibre (absolute in-trial achieved dietary fibre in the low-GI/GL diets) and difference in fibre (test-control). We assessed significant difference within each subgroup category or where possible as a continuous variable. Residual I2 was estimated to measure the remaining heterogeneity after accounting for any effect modification. We also conducted dose response analyses to assess linear dose response gradients and non-linear dose response thresholds for dietary GI and GL (by both absolute in-trial achieved GI/GL on the low-GI/GL diets and difference in GI/GL, test-control) if there were ≥6 trial comparisons (18). Linear dose response analyses were assessed by random-effects meta-regression. Non-linear dose-response associations were assessed with restricted cubic splines with three knots at Harrell's recommended percentiles (15%, 50%, 85%)(19). Departure from linearity was assessed using the Wald test and its significance conferred non-linear model as the best fit. When ≥10 trial comparisons were available, publication bias was investigated by inspection of contour enhanced funnel plots(20) and formal testing using the Egger's and Begg's tests (at $P<0.05$)(21, 22). If publication bias was suspected, we attempted to adjust for funnel plot asymmetry by imputing the missing study data using the Duval and Tweedie trim-and-fill method(23).

GRADE assessment

The GRADE approach was used to assess the overall certainty of the evidence and produce evidence profiles where evidence was graded as high, moderate, low, or very low certainty(11, 24, 25). Two investigators (LC and DL, AA, AC or JLS) independently performed GRADE assessments for each outcome. Randomized controlled trials receive an initial grade of high by default and are downgraded based on pre-specified criteria. For risk of bias (assessed by the Cochrane Risk of Bias Tool), we downgraded if about one third of the domains assessed were rated as having a high risk of bias, although we also could make a judgement to downgrade if any one domain was highly rated as having high risk of bias which could have influenced bias in the overall outcome. For inconsistency, we downgraded if there was serious inconsistency as evidence of substantial heterogeneity ($I2 \geq 50\%$, $P < 0.10$) that was unexplained by any a priori sensitivity or subgroup analyses. If there was evidence of substantial unexplained heterogeneity by these criteria, then we confirmed this assessment by supplementing the approach with visual inspection of forest plots for the 2 additional criteria specified in the GRADE handbook: the presence of wide variance of point estimates across studies and minimal to no overlap of CIs for some studies(25). For indirectness, we downgraded if we judged the presence of factors that limited the generalizability of the results. For imprecision, we downgraded if the 95% CI for the effect estimates overlapped the MIDs for benefit or harm. For publication bias, we downgraded if there was significant evidence of small-study effect which we defined as results from a trim and fill analysis which showed imputed trials resulted in a different conclusion compared to the original data. We conducted trim and fill analyses if we identified evidence of publication bias by inspection of funnel plots and significance by either the Egger's or Begg's tests (at $P<0.05$). We also assessed the potential for upgrading evidence as a result of the presence of a dose response. A linear dose response which supports the effect estimate could be judged as reason for an upgrade. We then used the MIDs to assess the importance of the magnitude of our point estimates using the effect size categories according to GRADE guidance(11, 25) as follows: large effect = ≥5xMID, moderate effect = ≥2xMID, small but important effect = ≥1xMID, and trivial/unimportant effect = < 1 MID. Please refer to **Supplemental Table S5** for MIDs for each cardiometabolic outcome.

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Supplemental Tables

Supplemental Table S1: PRISMA Checklist

Section/topic	#	Checklist item	Reported on page #
TITLE			
Title	1	Identify the report as a systematic review, meta-analysis, or both.	1
ABSTRACT			
Structured summary	2	Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number.	3
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	5
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	5
METHODS			
Protocol and registration	5	Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number.	5
Eligibility criteria	6	Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale.	5-6, Supplemental Method, Supplemental Table S3
Information sources	7	Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched.	5-6, Supplemental Table S2
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	5-6, Supplemental Tables S2-S3
Study selection	9	State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis).	5-6, Figure 1, Supplemental Method
Data collection process	10	Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators.	6-7
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made.	6-7, Supplemental Method
Risk of bias in individual studies	12	Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis.	6, Supplemental Method, Supplemental Table S4
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	6-7, Supplemental Method
Synthesis of results	14	Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I^2) for each meta-analysis.	6-7, Supplemental Method

Section/topic	#	Checklist item	Reported on page #
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	6, Supplemental Method, Supplemental Table S4
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	6-8, Supplemental Method
RESULTS			
Study selection	17	Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram.	8, Figure 1
Study characteristics	18	For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations.	8-9, Table 1, Supplemental Table S6
Risk of bias within studies	19	Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12).	9, Supplemental Figures S1-S2
Results of individual studies	20	For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot.	9-10, Figure 2, Supplemental Figures S3-16
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	9-10, Figure 2, Supplemental Figures S3-16
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	9, Supplemental Figures S1-S2, Supplemental Figures S32,34,36,38,40,42,44,46,48
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	10-12, Supplemental Figures S17-S74, Supplemental Tables S7-10
DISCUSSION			
Summary of evidence	24	Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers).	12-16
Limitations	25	Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias).	15-16
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	16-17
FUNDING			
Funding	27	Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review.	17-8

From: Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group (2009). Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. PLoS Med 6(7): e1000097. doi:10.1371/journal.pmed1000097

For more information, visit: **www.prisma-statement.org**.

Supplemental Table S2: Search strategy for randomized controlled trials assessing the effect of low-GI/GL dietary patterns on glycemic control and cardiometabolic outcomes in diabetes

Database	Search Period	Search Terms
MEDLINE	1946 to May 13, 2021	1. glycaemic index.mp. 2. glycemic index.mp. 3. glycaemic ind*.mp. 4. glycemic ind*.mp. 5. glycaemic load*.mp. 6. glycemic load*.mp. 7. glycemic index/ 8. or/1-7 9. clinical trial.mp. 10. clinical trial.pt. 11. random:.mp. 12. tu.xs. 13. or/9-12 14. 8 and 13 15. limit 14 to animals 16. 14 not 15
Embase	1946 to May 13, 2021	1. glycaemic index.mp. 2. glycaemic load*.mp. 3. glycemic ind*.mp. 4. glycemic index/ 5. glycemic load*.mp. 6. or/1-5 7. random:.mp. 8. clinical trial:.mp. 9. exp health care quality/ 10. or/7-9 11. 6 and 9 12. limit 11 to animals 13. 11 not 12 14. limit 13 to animal studies 15. 13 not 14
The Cochrane Library	1946 to May 13, 2021	1. glycemic index/ 2. glycaemic ind*.mp. 3. glycemic ind*.mp. 4. glycemic load*.mp. 5. glycaemic load*.mp. 6. or/1-5

GI, glycemic index; GL, glycemic load

Supplemental Table S3: PICO framework of the search strategy

PICO framework[a] defined in the present systematic review and meta-analysis			
Participants	**Interventions**	**Comparators**	**Outcomes**
Individuals of all ages with type-1 or type-2 diabetes mellitus excluding pregnant or breastfeeding women	Dietary patterns focused on low-Glycemic index foods or on a low-Glycemic load	Higher glycemic index or glycemic load diets	HbA1c Fasting glucose Fasting insulin LDL-C Non-HDL-C HDL-C Triglycerides Apo-B Body weight Body mass index (BMI) Waist circumference Systolic blood pressure Diastolic blood pressure C-reactive protein (CRP)

Apo-B, apolipoprotein B; BMI, body mass index; CRP, c-reactive protein; HbA1c, hemoglobin A1c; HDL-C, high-density lipoprotein-cholesterol; Non-HDL-C, non-high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; PICO, participants, interventions, comparators, outcomes

[a]Moher D, Shamseer L, Clarke M, Ghersi D, Liberati A, Petticrew M, Shekelle P, Stewart LA and PRISMA-P Group. Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015 statement. Systematic Reviews 2015; 4:1. https://doi.org/10.1186/2046-4053-4-1

Supplemental Table S4: Criteria for judging risk of bias using the Cochrane Risk of bias assessment tool

RANDOM SEQUENCE GENERATION Selection bias (biased allocation to interventions) due to inadequate generation of a randomised sequence.		TORONTO 3D additional considerations
Criteria for a judgement of 'Low risk' of bias.	The investigators describe a random component in the sequence generation process such as: Referring to a random number table; Using a computer random number generator; Coin tossing; Shuffling cards or envelopes; Throwing dice; Drawing of lots; Minimization*. *Minimization may be implemented without a random element, and this is considered to be equivalent to being random.	LOW: Randomized and described using unpredictable method HIGH: Non randomized or predictable method used UNCLEAR: 1. Randomized but not described so unable to judge
Criteria for the judgement of 'High risk' of bias.	The investigators describe a non-random component in the sequence generation process. Usually, the description would involve some systematic, non-random approach, for example: Sequence generated by odd or even date of birth; Sequence generated by some rule based on date (or day) of admission; Sequence generated by some rule based on hospital or clinic record number. Other non-random approaches happen much less frequently than the systematic approaches mentioned above and tend to be obvious. They usually involve judgement or some method of non-random categorization of participants, for example: Allocation by judgement of the clinician; Allocation by preference of the participant; Allocation based on the results of a laboratory test or a series of tests; Allocation by availability of the intervention.	
Criteria for the judgement of 'Unclear risk' of bias.	Insufficient information about the sequence generation process to permit judgement of 'Low risk' or 'High risk'.	

ALLOCATION CONCEALMENT Selection bias (biased allocation to interventions) due to inadequate concealment of allocations prior to assignment.		TORONTO 3D additional considerations
Criteria for a judgement of 'Low risk' of bias.	Participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: Central allocation (including telephone, web-based and pharmacy-controlled randomization); Sequentially numbered drug containers of identical appearance; Sequentially numbered, opaque, sealed envelopes.	LOW: If a 3rd party did the randomization and is unpredictable to personnel until revealed on day of randomization (*Note:* also includes block randomization with use of different block sizes) HIGH: Non-randomized If predictable by personnel (*Note:* also includes block randomization with a set block size) UNCLEAR: If randomized but unclear if predictable to personnel
Criteria for the judgement of 'High risk' of bias.	Participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: Using an open random allocation schedule (e.g. a list of random numbers); Assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or nonopaque or not sequentially numbered); Alternation or rotation; Date of birth; Case record number; Any other explicitly unconcealed procedure.	
Criteria for the judgement of 'Unclear risk' of bias.	Insufficient information to permit judgement of 'Low risk' or 'High risk'. This is usually the case if the method of concealment is not described or not described in sufficient detail to allow a definite judgement – for example if the use of assignment envelopes is described, but it remains unclear whether envelopes were sequentially numbered, opaque and sealed.	

BLINDING OF PARTICIPANTS AND PERSONNEL[¥] Performance bias due to knowledge of the allocated interventions by participants and personnel during the study.		TORONTO 3D additional considerations
Criteria for a judgement of 'Low risk' of bias.	Any one of the following: No blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding; Blinding of participants and key study personnel ensured, and unlikely that the blinding could have been broken.	LOW: double blinded* single blinded (any one of participants/personnel/outcome assessors) if study was metabolically controlled If the study was unblinded and you believe that would NOT bias the outcome effect (i.e., participants in both groups given advice on background diet, advice on physical activity, etc. to try to reduce other factors changing beyond the intervention of interest)** HIGH: If the study was unblinded and you believe that will bias the outcome effect (i.e., if the subjects randomized to a healthy diet emphasizing oats change other components of their lifestyle to be healthy, e.g. increased physical activity.)** If it is clearly stated that the statistician was not blinded and there was no stated a priori analysis plan UNCLEAR: If unblinded and you cannot judge because of the way the study was described **Note: this may not always necessarily mean the statistician/outcome assessors so check* ***Note: will be somewhat subjective and require deliberation with the team*
Criteria for the judgement of 'High risk' of bias.	Any one of the following: No blinding or incomplete blinding, and the outcome is likely to be influenced by lack of blinding; Blinding of key study participants and personnel attempted, but likely that the blinding could have been broken, and the outcome is likely to be influenced by lack of blinding.	
Criteria for the judgement of 'Unclear risk' of bias.	Any one of the following: Insufficient information to permit judgement of 'Low risk' or 'High risk'; The study did not address this outcome.	

[¥] We assess "Blinding of participants and personnel" and "Blinding of outcome assessment" as one domain

BLINDING OF OUTCOME ASSESSMENT [¥] Detection bias due to knowledge of the allocated interventions by outcome assessors.		TORONTO 3D additional considerations
Criteria for a judgement of 'Low risk' of bias.	Any one of the following: No blinding of outcome assessment, but the review authors judge that the outcome measurement is not likely to be influenced by lack of blinding; Blinding of outcome assessment ensured, and unlikely that the blinding could have been broken.	Please refer to notes in previous section "Blinding of participants and personnel"
Criteria for the judgement of 'High risk' of bias.	Any one of the following: No blinding of outcome assessment, and the outcome measurement is likely to be influenced by lack of blinding; Blinding of outcome assessment, but likely that the blinding could have been broken, and the outcome measurement is likely to be influenced by lack of blinding.	
Criteria for the judgement of 'Unclear risk' of bias.	Any one of the following: Insufficient information to permit judgement of 'Low risk' or 'High risk'; The study did not address this outcome.	

[¥] We assess "Blinding of participants and personnel" and "Blinding of outcome assessment" as one domain

INCOMPLETE OUTCOME DATA Attrition bias due to amount, nature or handling of incomplete outcome data.		TORONTO 3D additional considerations
Criteria for a judgement of 'Low risk' of bias.	Any one of the following: No missing outcome data; Reasons for missing outcome data unlikely to be related to true outcome (for survival data, censoring unlikely to be introducing bias); Missing outcome data balanced in numbers across intervention groups, with similar reasons for missing data across groups; For dichotomous outcome data, the proportion of missing outcomes compared with observed event risk not enough to have a clinically relevant impact on the intervention effect estimate; For continuous outcome data, plausible effect size (difference in means or standardized difference in means) among missing outcomes not enough to have a clinically relevant impact on observed effect size; Missing data have been imputed using appropriate methods.	<u>Case A: If # started = # analyzed*</u> LOW: If NO drop-outs/missing data** If missing data is <20% and missing data were imputed (ITT) with any method of imputation If missing data is >20% and ITT used, there are NO imbalances or baseline differences between groups and the method of imputation used is NOT last observation carried forward (LOCF). HIGH: If missing data is between 20% to 40% and ITT used, and there ARE imbalances or baseline differences between groups If missing data is >40% <u>Case B: If # analyzed is < than # started</u> Could go either way depending on how you answer the following questions: a. Was missing data similar b/w treatment groups (<20% difference between groups and reasons similar, e.g. adverse events vs. other)? b. Were those excluded similar to those who completed? c. Was % missing data ≤20%***? LOW: If YES to all 3 questions UNCLEAR: If in between
Criteria for the judgement of 'High risk' of bias.	Any one of the following: Reason for missing outcome data likely to be related to true outcome, with either imbalance in numbers or reasons for missing data across intervention groups; For dichotomous outcome data, the proportion of missing outcomes compared with observed event risk enough to induce clinically relevant bias in intervention effect estimate; For continuous outcome data, plausible effect size (difference in means or standardized difference in means) among missing outcomes enough to induce clinically relevant bias in observed effect size; 'As-treated' analysis done with substantial departure of the intervention received from that assigned at randomization; Potentially inappropriate application of simple imputation.	
Criteria for the judgement of 'Unclear risk' of bias.	Any one of the following:	

	Insufficient reporting of attrition/exclusions to permit judgement of 'Low risk' or 'High risk' (e.g. number randomized not stated, no reasons for missing data provided); The study did not address this outcome.	HIGH: If NO to all 3; OR if missing data is >40% **Note: do NOT assume if a paper reports "ITT" it means they properly performed ITT analyses – check #s* ***Note: in old studies may not be able to determine if there were any drop-outs (e.g. no info on flow of participants). In these cases, rate LOW if state "recruited" X people; rate UNCLEAR if state "studied" or "used" X people* ****Note: 20% chosen b/c beyond this there is a high risk of imbalance in prognostic factors*

SELECTIVE REPORTING Reporting bias due to selective outcome reporting.		TORONTO 3D additional considerations
Criteria for a judgement of 'Low risk' of bias.	Any of the following: The study protocol is available and all of the study's pre-specified (primary and secondary) outcomes that are of interest in the review have been reported in the pre-specified way; The study protocol is not available but it is clear that the published reports include all expected outcomes, including those that were pre-specified (convincing text of this nature may be uncommon).	LOW: If protocol number was provided, all primary/secondary outcomes were reported in study's paper (especially primary) If no protocol number, study states the primary/secondary outcomes and it was reported If no protocol number and "wishy-washy" language, study provides a power calculation for an outcome (which is assumed to be primary) and this outcome is reported HIGH: 1. If protocol number provided, primary and secondary do NOT match what was reported or misrepresented primary outcome
Criteria for the judgement of 'High risk' of bias.	Any one of the following: Not all of the study's pre-specified primary outcomes have been reported; One or more primary outcomes is reported using measurements, analysis methods or subsets of the data (e.g. subscales) that were not pre-specified; One or more reported primary outcomes were not pre-specified (unless clear justification for their reporting is provided, such as an unexpected adverse effect);	

	One or more outcomes of interest in the review are reported incompletely so that they cannot be entered in a meta-analysis; The study report fails to include results for a key outcome that would be expected to have been reported for such a study.	UNCLEAR If no protocol number and “wishy-washy” language, no power calculation
Criteria for the judgement of ‘Unclear risk’ of bias.	Insufficient information to permit judgement of ‘Low risk’ or ‘High risk’. It is likely that the majority of studies will fall into this category.	

Supplemental Table S5: Minimally important differences for each cardiometabolic outcome

Outcome	MID	Reference
HbA1c	0.3%	European Medicines Agency. Guideline on clinical investigation of medicinal products in 4 the treatment or prevention of diabetes mellitus. 29 January 2018. CPMP/EWP/1080/00 Rev. 1. https://www.ema.europa.eu/en/documents/scientific-guideline/draft-guideline-clinical-investigation-medicinal-products-treatment-prevention-diabetes-mellitus_en.pdf Threshold proposed by the EMA as clinically meaningful: "When predefining a non-inferiority margin, it should be considered that even apparently small reductions in HbA1C have been shown to be clinically relevant in terms of risk reduction of diabetic complications. While a margin of 0.3% (3 mmol/mol) is generally considered as acceptable, the choice of the margin should always be discussed in the clinical context."
Fasting glucose	0.5mmol/L	David M. Nathan, Judith Kuenen, Rikke Borg, Hui Zheng, David Schoenfeld, and Robert J. Heine, for the A1c-Derived Average Glucose (ADAG) Study Group. Diabetes Care 2008 https://professional.diabetes.org/diapro/glucose_calc A conservative estimate associated with HbA1c and accounting for day-to-day variation in fasting glucose.
Fasting insulin	5pmol/L	Proportional reduction to fasting glucose
LDL-C, non-HDL-C, triglycerides	0.1mmol/L	1. Cholesterol Treatment Trialists' (CTT) Collaboration. Baigent C, Blackwell L, Emberson J, et al. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170,000 participants in 26 randomised trials. Lancet 2010;376:1670-1681 2. Ference et al. Eur Heart J 2018;39, 2540–2545 3. Cannon et al. N Engl J Med 2015; 372:2387-2397 Reduction of 1 mmol/L in LDL-C results in 20% reduction in vascular events.
apoB	0.04g/L	Effect sizes are more like 35-40% of that of LDL-C. 3.5mmol/L LDL-C is considered equivalent to 1.2g/L apo B (threshold for treatment of those at intermediate FRS) = 34% and 2mmol/L LDL-C is considered equivalent to 0.8g/L apo B (treatment target) = 40%, which is the same as 5% of 0.8g/L (near the upper end of our target level for whom we would still seek reductions to get to target) = 0.04g/L
Body weight	0.5kg	Ge L, Sadeghirad B, Ball GDC, da Costa BR, Hitchcock CL, Svendrovski A, Kiflen R, Quadri K, Kwon HY, Karamouzian M, Adams-Webber T, Ahmed W, Damanhoury S, Zeraatkar D, Nikolakopoulou A, Tsuyuki RT, Tian J, Yang K, Guyatt GH, Johnston BC. Comparison of dietary macronutrient patterns of 14 popular named dietary programmes for weight and cardiovascular risk factor reduction in adults: systematic review and network meta-analysis of randomised trials. BMJ. 2020 Apr 1;369:m696. doi: 10.1136/bmj.m696.

BMI	0.2kg/m^2	Roughly equivalent to 0.5kg
Waist circumference	2cm	2cm=1 full pant size
SBP, DBP	2mmHg	Lancet. 2002 Dec 14;360(9349):1903-13. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R; Prospective Studies Collaboration. A 2 mm Hg lower usual SBP would involve about 10% lower stroke mortality and about 7% lower mortality from ischemic heart disease or other vascular causes in middle age.
CRP	0.50 mg/L (4.76nmol/L)	1. Reynolds Risk Score. Available at: http://www.reynoldsriskscore.org/Default.aspx [Accessed March 14, 2018]. 2. Ridker, P.M. et al., 2008. C-reactive protein and parental history improve global cardiovascular risk prediction: the Reynolds Risk Score for men. Circulation, 118(22), pp.2243–51, 4p following 2251. Available at: http://dx.doi.org/10.1161/CIRCULATIONAHA.108.814251. 3. Ridker, P.M. et al., 2007. Development and validation of improved algorithms for the assessment of global cardiovascular risk in women: the Reynolds Risk Score. JAMA: the journal of the American Medical Association, 297(6), pp.611–619. Available at: http://dx.doi.org/10.1001/jama.297.6.611. 0.5mg/L change in hs-CRP is equal to 1% change in 10-year CVD risk

Apo-B, apolipoprotein-B; BMI, body mass index; CRP, c-reactive protein; CVD, cardiovascular disease; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; LDL-C, low-density lipoprotein-cholesterol; HDL-C, high-density lipoprotein-cholesterol; Non-HDL-C, non-high-density lipoprotein-cholesterol; SBP, systolic blood pressure

Supplemental Table S6a: Trial characteristics - design aspects

Study, year	Intervention, Control	No. of participants (M, F)[a]	Sex (%F)	Mean Age, yr (SD)	Diabetes Duration, yr (SD or Range)	Baseline HbA1c, % (SD)	Setting	Design	Feeding Control[b]	F/U Duration, wks	Funding Sources[c]
Brand et al. 1991		16 T2DM (10 M, 6 W)	38	62 (9)	5 (1-22)	7.7 (2)	OP, Australia	C (3-wk w/o)	DA	12	A
	Intervention										
	Control										
Cai et al. 2017		130 T2DM (70M, 60F)	46	56.7 (3.5)	6.3 (1.2)		OP, China	P	Supp	24	NR
	Intervention	65 T2DM				8.04 (0.62)					
	Control	65 T2DM				8.02 (0.54)					
Collier et al. 1988		7 T1DM (6M, 1F)	14	12 (5)	3 (1)		OP, Canada	C (10-wk w/o)	DA	6	A
	Intervention					10.0 (2.1)					
	Control					9.9 (1.6)					
Elhayany et al. 2010			48				OP, Isreal	P	DA	52	A
	Intervention - LGI	63 T2DM (35M, 28F)		57.4 (6.1)	6.2 (9.9)	8.3 (1.0)					
	Intervention - LGL	61 T2DM (31M, 30F)		55.5 (6.5)	5.5 (3.8)	8.3 (1.0)					
	Control	55 T2DM (27M, 28F)		56.0 (6.1)	5.1 (2.6)	8.3 (0.8)					
Fabricatore et al. 2011			80		NR		OP, USA	P	DA	40	A
	Intervention	40 T2DM (8 M, 32 F)		52.8 (8.9)		6.6 (1.3)					
	Control	39 T2DM (8 M, 31 F)		52.5 (8.1)		7.0 (1.2)					
Fontvieille et al. 1988		8 T1DM (4 M, 4 F)	50	43.5 (9.9)	14.6 (6.8)	NR	OP, France	C (no w/o)	Supp	3	A, I
	Intervention										
	Control										
Fontvieille et al. 1992		6 T2DM (2M, 4F); 12 T1DM (10M, 2F)	33	47.2 (11.6)	7.8 (5.0) T2DM; 13.4 (5.1) T1DM	NR	OP, France	C (no w/o)	DA	5	A, I
	Intervention										
	Control										
Frost et al. 1994			29		NR	NR	OP, UK	P	DA	12	A
	Intervention	25 T2DM (16M, 9F)		54 (2)							
	Control	26 T2DM (20M, 6F)		56 (3)							
Giacco et al. 2000		54 T1DM (21 M, 33 W)	61	28.2 (9.5)	10.3 (6.3)		OP, Italy	P	DA	24	A
	Intervention	29 T1DM (12 M, 17 W)				8.8 (1.0)					
	Control	25 T1DM (9 M, 16 F)				9.1 (1.3)					
Gilbertson et al. 2001		104 T1DM (52 M, 52 W)	50				OP, Australia	P	DA	52	A
	Intervention	55 T1DM (27 M, 28 W)		10.7 (1.6)	3.4 (1.3-12.2)	8.6 (1.4)					
	Control	49 T1DM (25 M, 24 W)		10.2 (1.6)	4.0 (1.1-9.9)	8.3 (1.3)					
Gomes et al. 2017		20 T2DM (10 M, 10 W)	50	42.4 (5.1)		NR	OP, Brazil	P	Supp	4	NR
	Intervention	10 T2DM (5 M, 5 W)		44.3 (4.8)	4.8 (1.5)						
	Control	10 T2DM (5 M, 5 W)		41.1 (3.2)	4.9 (1.6)						

Supplement Table S6a: (Continued)

Study, year	Intervention, Control	No. of participants (M, F)[a]	Sex (%F)	Mean Age, yr (SD)	Diabetes Duration, yr (SD or Range)	Baseline HbA1c, % (SD)	Setting	Design	Feeding Control[b]	F/U Duration, wks	Funding Sources[c]
Heilbronn et al. 2002		45 T2DM (23 M, 22 W)	49		NR		OP, Australia	P	Supp	8	NR
	Intervention	24 T2DM (11 M, 13 W)		57.5 (9.6)		6.65 (1.37)					
	Control	21 T2DM (12 M, 9 W)		56.0 (9.4)		6.35 (1.60)					
Järvi et al. 1999		20 T2DM (15M, 5F)	25	66.5 (50-77)	0.5-17	7.2 (1.4)	OP, Sweeden	C (no w/o)	Met	3.4	A
	Intervention										
	Control										
Jenkins et al. 2008		210 T2DM (128 M, 82 W)	39				OP, Canada	P	DA	24	A, I
	Intervention	106 T2DM (65 M, 41 W)		60 (10)	8.3 (6.5)	7.1 (1.0)					
	Control	104 T2DM (63 M, 41 W)		61 (9)	7.2 (5.9)	7.1 (1.0)					
Jenkins et al. 2012		121 T2DM (61M: 60F)	50				OP, Canada	P	DA	12	A
	Intervention	60 T2DM		58 (10.1)	9.2 (6.2)	7.4 (0.8)					
	Control	61 T2DM		61 (7.8)	8.6 (6.2)	7.2 (0.8)					
Jenkins et al. 2014		141 T2DM (77M, 64F)	45				OP, Canada	P	Supp	12	I
	Intervention	70 T2DM (38M, 32F)		59 (10)	7.6 (6.9)	7.4 (0.6)					
	Control	71 T2DM (39M, 32F)		59 (10)	7.5 (5.4)	7.2 (0.6)					
Jimenez-Cruz et al. 2003		14 T2DM (6M, 8F)**	57	53 (9)	8 (7)		OP, Mexico	C (6-wk w/o)	DA	6	I
	Intervention					8.5 (1.0)					
	Control					8.6 (1.1)					
Jimenez-Cruz et al. 2004		8 T2DM	NR	51 (3)	7 (6)	NR	OP, Mexico	C (4-wk w/o)	DA	3	A
	Intervention										
	Control										
Komindr et al. 2001		10 T2DM (0 M, 10 W)	100	(32-60)	NR	13.84	OP, Thailand	C (no w/o)	Supp	4	I
	Intervention										
	Control										
Luscombe et al. 1999 HGI		21 T2DM (14 M, 7 W)	33	57.4 (13.3)	6.3 (10.55)	NR	OP, Australia	C (no w/o)	Supp	4	A, I
	Intervention										
	Control										
Luscombe et al. 1999 MUFA		21 T2DM (14 M, 7 W)	33	57.4 (13.3)	6.3 (10.55)	NR	OP, Australia	C (no w/o)	Supp	4	A, I
	Intervention										
	Control										
Ma et al. 2008		40 T2DM (19 M, 21 W)	53	53.5 (8.4)	9.32 (9.7)		OP, USA	P	DA	52	A
	Intervention	19 T2DM (8 M, 11 W)		51.0 (8.3)	12.65 (11.9)	8.74 (1.26)					
	Control	21 T2DM (11 M, 10 W)		56.31 (7.9)	6.62 (6.5)	8.10 (1.28)					
Pavithran et al. 2020		80 T2DM (52 M, 28 W)	35	53.2	<10		OP, India	P	DA	24	A
	Intervention	40 T2DM (25 M, 15 W)		54.4 (7.6)		8.44 (0.96)					
	Control	40 T2DM (27 M, 13 W)		51.9 (7.4)		8.27 (0.99)					

Supplement Table S6a: (Continued)

Study, year	Intervention, Control	No. of participants (M, F)[a]	Sex (%F)	Mean Age, yr (SD)	Diabetes Duration, yr (SD or Range)	Baseline HbA1c, % (SD)	Setting	Design	Feeding Control[b]	F/U Duration, wks	Funding Sources[c]
Rizkalla et al. 2004		12 T2DM (12 M, 0 W)	0	54 (6.9)	NR		OP, France	C (4-wk w/o)	DA	4	A, I
	Intervention					7.56 (1.25)					
	Control					7.45 (1.21)					
Visek et al. 2014		20 T2DM (12M, 8F)	40	62.7 (5.8)	7 (4.1)	7 (2.88)	OP, Czech Republic	C (12-wk w/o)	DA	12	A
	Intervention										
	Control										
Wolever et al. 1992		6 T2DM (3M, 3F)	50	63 (10)	NR	NR	OP, Canada	C (4- to 6-wk w/o)	Met	6	A, I
	Intervention										
	Control										
Wolever et al. 2008		103 T2DM	58		NR	NR	OP, Canada	P	Supp	52	A, I
	Intervention	55 T2DM (~19M, 36F)		60.6 (7.5)*							
	Control	48 T2DM (~24M, 24F)		60.4 (7.9)*							
Yusof et al. 2009		100 T2DM**	NR	NR	NR		OP, Malaysia	P	Supp	12	A
		51 T2DM				7.68 (1.13)					
	Intervention										
	Control	49 T2DM				7.51 (1.24)					

A, agency; ADA, American Diabetes Association; C, crossover; Carb, carbohydrate; DA, dietary advice; F, female; F/U, follow-up; GI, glycemic index; GL, glycemic load; HbA1c, hemoglobin A1c; HGI, high glycemic index; I, industry; LGI, low-glycemic index; M, male; Met, metabolic; MUFA, monounsaturated fatty acid; NR, not reported; OP, outpatient; P, parallel; SD, standard deviation; Supp, supplemental feeding control; T1DM, type-1 diabetes mellitus; T2DM, type-2 diabetes mellitus; UK, United Kingdom; USA, United States of America; w/o, wash-put period; wks, weeks; yr, year

a all sample sizes reflect participants included in the data analyzed

b Metabolic feeding control (Met) is the provision of all meals and foods consumed during the study under controlled conditions. Supplemental feeding control (Supp) is the provision of some meals and foods consumed during the study. Dietary advice (DA) is the provision of counseling on the appropriate intervention and control diets.

c Agency funding is that from government, university, or not-for-profit sources. Industry funding is that from trade organizations that obtain revenue from the sale of products.

* Calculated before dropout.

**Completer Analysis, as used in data analysis.

¶¶ median and interquartile range (IQR).

§ based on 6-month data (as reported in a companion study: Fraser A, et al. A modified Mediterranean diet is associated with the greatest reduction in alanine aminotransferase levels in obese type 2 diabetes patients: results of a quasi-randomised controlled trial. Diabetologia. 2008. 51:1616–1622).

Supplemental Table S6b: Trial characteristics - dietary aspects

Study, year	Intervention, Control	Intervention or Comparator Diet	GI Dose[a]	GI difference between groups	GL Dose[a]	GL difference between groups	Intervention description	Adherence assessment	Diet (% Carb:Fat:Protein)[b]	Energy Balance[c]
Brand et al. 1991				9.2		~ -20¥	Subjects were seen weekly in their homes by the same research dietitian who provided detailed dietary instruction, assessment, provided recipes, sample foods, support and encouragement to maintain compliance; diets were personalized	**dietitians** assessed adherence weekly at home visits, including body weight (aim of weight maintenance); assessment of compliance not reported		Neutral
	Intervention	LGI diet	54.7		~99.5¥					
	Control	HGI diet	63.9		~119¥				46:31:19 1622kcal	
Cai et al. 2017				NA		NA	professional **nutritionists** provided **personalized** recipes; patients in both groups received supplemented foods and recipe food form on the 1st and 15th of each month and returned the completed forms at each of these times over the 6-months	NR		Neutral
	Intervention	LGI/High fibre	NR		NR		soluble fibre (fruit and vegetable fiber meal) and LGI grains (buckwheat) provided		NR	
	Control	Standard DM diet/HGI, Lower Fiber	NR		NR				NR	
Collier et al. 1988				9.1		~ 1¥	at 3wk intervals, 7-day diet records were collected	intake of some carbohydrate foods are reported demonstrating a higher intake of LGI foods in the LGI diet and a higher intake of higher GI foods in the control; no other assessment of adherence		Neutral
	Intervention	LGI diet	48.8 (1.8)		~176¥		LGI diet was **personalized** based on baseline food records where high GI foods were replaced with LGI foods; cooking instructions and recipes provided and sample menus individually developed where necessary; exchange lists provided for LGI foods		48.0(5.3):33.5(5.0):16.1(1.9) 3003(1235.6)kcal	
	Control	HGI diet	57.9 (0.8)		~175¥		baseline diet served as instruction for control		47.4(4.2):37.0(7.4):15.6(2.1) 2555(711.7)kcal	
Elhayany et al. 2010				NA		NA	Patients were followed up by the same **dietitian** every 2wks for 1 year; meetings followed a structured protocol and patients received personalized meal plan consultation	Adherence was assessed by 24-h food recall questionnaire, validated FFQ and physical activity questionnaire at baseline, 3 months, and 6 months; assessment for compliance not reported		Neutral
	Intervention - LGI	LGI diet	NR		NR		Only low glycmic index carbohydrates		45(6.8):36(5.6):20(3.3)§ 1758kcal	
	Intervention - LGL	LGL/High MUFA	NR		NR		Only low glycmic index carbohydrates (35% CHO); 45% fat that is high in MUFA		42(7.5):41(6.6):19(3.4)§ 1734kcal	
	Control	Standard ADA diet	NR		NR		Mixed glycemic index carbohydrates		46(7.1):37(6.3)19(2.8)§ 1710kcal	
Fabricatore et al. 2011				7.3		32.7	Doctoral- or masters-level-trained clinical psychologists provided dietary prescription (caloric intake) was **personalized** based on weight (<113.4 kg or ≥113.4 kg). Participants were given a calorie-counting guide			Negative[£]
	Intervention	LGL diet	57.4		88.6		Participants were given instructions on the glycemic effects of food and taught guidelines for identifying low-, moderate-, and high-GL items. Participants were prescribed goals to consume ≤3 and ≤1 serving per day of moderate-GL and high-GL items, respectively. Received recipes, sampled foods, eating plan, and given 3 servings of moderate-GL foods, and <1 serving of high-GL foods per day over 2 weeks	Participants were asked to record moderate- and high-GL foods and caloric intake in daily self-monitoring logs (3-day food records, 2 weekdays and 1 weekend day); assessment for compliance not reported	41:40:20 1500kcal	
	Control	Low fat diet	64.7		121.3		Participants were given low-fat recipes, eating plan, and 2 weeks' worth of meals and snacks on average and 30g fat per day	Participants were asked to record caloric and fat gram intake in daily self-monitoring logs (3-day food records, 2 weekdays and 1 weekend day); assessment for compliance not reported	50:33:19 1500kcal	

Supplement Table S6b: (Continued)

Study, year	Intervention, Control	Intervention or Comparator Diet	GI Dose[a]	GI difference between groups	GL Dose[a]	GL difference between groups	Intervention description	Adherence assessment	Diet (% Carb:Fat:Protein)[b]	Energy Balance[c]
Fontvieille et al. 1988				13.6		~-30¥	Participants were prescribed a **personalized** diet to maintain caloric intake and nutrient proportions	Compliance to diet prescription was attested at two further diet inquiries taken at the end of each 3-week period		Neutral
	Intervention	LGI diet	46.5 (2.5)		~115¥		Low glycemic foods (rice, biscuits, pasta, apples)		46.1 (4.5): 35.0 (2.8): 17.4 (1.4) 2152(223.4)kcal	
	Control	HGI diet	60.1 (5.1)		~145¥		High glycemic index foods (bread, potato, bananas)		45.4 (4.5): 36.0 (2.8): 16.9 (1.7) 2118(271.5)kcal	
Fontvieille et al. 1992				26.1		~-56¥	Participants were prescribed a **personalized** diet to maintain caloric intake and nutrient proportions	Compliance to dietary prescription was assessed based on a food diary, the last 7-day records of each diet was reviewed by a **trained dietitian**		Neutral
	Intervention	LGI diet	38.1 (5.3)		~91¥		Low GI foods (rice, biscuits, pasta, apple, peas/beans, rye bread) were used as recommended by **dietitians**		45.8 (7.2): 36.2 (6.8): 18.0 (2.5) 1834(311)kcal	
	Control	HGI diet	64.2 (3.1)		~147¥		Higher GI foods (bread, potato, banana) were recommended		44.9 (7.3): 36.3 (6.0): 18.8 (1.6) 1787(268)kcal	
Frost et al. 1994				3.5		~ 5¥	Each diet was prescribed **personally** to fit the subject's normal lifestyle through verbal and written instruction	Dietary acheivement was assessed by two 3-day diet diaries (end of week 4 and end of week 12); assessment for compliance not reported		Neutral
	Intervention	LGI diet	54.7		~120¥		Standard British Dietetic Association advice and encouraged to use whole grain rye bread (pumpernickel bread), oats, barley and pasta, and to increase the consumption of beans, pulse vegetables, and fruit		49:25:23 1800kcal	
	Control	Standard British Dietetic Association Advice	58.2		~115¥		Standard British Dietetic Association Advice (50% carbohydrate and more dietary fibre and 35% from fat)		44:32:22 1800kcal	
Giacco et al. 2000				14		~ -21¥	Patients consumed a weight-maintaining diet following an intensive dietary education program (diet history, formulation of a personalized diet, two 1-h educational sessions with a **dietitian** who provided recipes, written suggestions for eating out, and food choices). Individual meetings were held on a monthly basis over 24 weeks	Compliance to diet was evaluated based on a 7-day food records for each monthly study visit; deviations from prescribed diet (unsatisfactory when the average consumption of carbohydrate during the treatment period was 45% of total energy for both diets and/or the consumption of fiber was 20 g/day for the LF diet or 30g/day for the HF diet) were underlined to reinforce dietary prescription	50:30:20‡	Neutral
	Intervention	LGI diet/High fibre	50‡		~125¥		Patients were advised to consume one serving of legumes, three servings of high-fibre fruit, and two servings of high fibre vegetables		55:28:20‡ 1756kcal	
	Control	Low fibre diet	64‡		~146¥		Patients were advised to limit legume consumption and consume low fibre fruit and vegetables		55:28:17‡ 1846kcal	
Gilbertson et al. 2001				1.2		~ -3¥	Subjects underwent a diet education session in an outpatient setting by the same clinical **dietitian**. A flipchart and literature were provided or used to explain the diets	Each subject was instructed to complete a 3-day food diary (2 weekdays and 1 weekend) at 1, 3, 6, and 12 months of the intervention period. Food diaries were analyzed by the same research dietitian. Phone calls were made 2 weeks before clinic visits to ensure compliance	49:34:17	Neutral
	Intervention	Low GI diet	55.3 (4.8)		~135¥					
	Control	Carbohydrate exchange diet	56.5 (4.0)		~138¥					

Supplement Table S6b: (Continued)

Study, year	Intervention, Control	Intervention or Comparator Diet	GI Dose[a]	GI difference between groups	GL Dose[a]	GL difference between groups	Intervention description	Adherence assessment	Diet (% Carb:Fat:Protein)[b]	Energy Balance[c]
Jimenez-Cruz et al. 2003				12		53	Participants were given detailed instructions and a pamphlet on lower- or higher-GI foods depending on randomization	Participants completed unweighed dietary intake diaries for 1 day during the weeks 1, 4, and 6 of the two study periods		Neutral
	Intervention	LGI diet	44 (3)		86 (20)		Lower-GI foods (oranges, beans/legumes, yogurt, pasta, and corn tortillas)	Compliance was high; only four participants dropped out of the study during this diet	60:23:21 1421kcal	
	Control	HGI diet	56 (5)		139 (27)		Higher-GI foods (corn flakes, white bread, potatoes, ripe bananas)		64:20:18 1560kcal	
Jimenez-Cruz et al. 2004				8.5		32.7	Diets were **personalized** to meet participants' food habits with 3-day cycle menu plans	Subjects completed unweighed dietary intake diaries for 3 days during the first and third week of the 2 study periods		Neutral
	Intervention	LGI diet	42.6 (0.21)		108.63 (0.28)				51 (3): 26 (2): 23 (4) 1938(71)kcal	
	Control	HGI diet	51.12 (0.28)		141.29 (0.28)				54 (1): 27 (3): 18 (2) 1998(61)kcal	
Komindr et al. 2001				~16.3‡		~33.2‡	Dietary habit interviews and daily dietary records were collected for 6 weeks prior to the study and during the first 3 days of the baseline period for creation of personalized weight-maintaining diabetic study diets. Subjects were taught to prepare their own test diets from a 4-day rotating menu. Every 2 wks, pre-weighed carbohydrates and recipes were given from the metabolic kitchen	NR	55:32:13‡ 1474kcal‡	Neutral
	Intervention	LGI diet	~56.4‡		~114.2‡		Mungbean noodles (35% daily kcal intake)			
	Control	HGI diet	~72.7‡		~147.4‡		White rice (40% daily kcal intake)			
Luscombe et al. 1999 HGI				20		~ -47¥	Subjects were seen fortnightly by the same **research dietitian** who provided dietary instruction and assessment. Subjects were given personalized specific study foods, dietary guidelines, and menus	Patient compliance was assessed from 2-day weighed food records and 24h diet recall, completed fortnightly throughout each dietary phase		Neutral
	Intervention	LGI diet	43		~104¥		Wholegrain bread, low-GI cereal, and low-GI fruits and vegetables		51:23:22 1905kcal	
	Control	HGI diet	63		~151¥		Wholemeal bread, high-GI cereal, and high-GI fruits and vegetables		51:21:23 1809kcal	
Luscombe et al. 1999 MUFA				16		~ -21¥	Subjects were seen fortnightly by the same **research dietitian** who provided dietary instruction and assessment. Subjects were given personalized specific study foods, dietary guidelines, and menus	Patient compliance was assessed from 2-day weighed food records and 24h diet recall, completed fortnightly throughout each dietary phase		Neutral
	Intervention	LGI diet	43		~104¥		Wholegrain bread, low-GI cereal, and low-GI fruits and vegetables		51:23:22 1905kcal	
	Control	HGI/MUFA diet	59		~125¥		Canola oil, canola margarine, and almonds		42:35:21 2023kcal	
Ma et al. 2008				2.6		20	Dietary sessions were provided to participants by **two registered dietitians**	A 7-day dietary recall on the week prior to study visits was recorded and used for dietary assessment		Neutral
	Intervention	LGI diet	54.41 (4.52)		85.04 (42.55)		Participants were educated on how to choose low-GI foods and integration of GI foods was **personalized** based on lifestyle and taste preferences		38:42:20 1674kcal	
	Control	Standard ADA diet	57.06 (4.56)		105.07 (43.30)		The ADA diet includes carbohydrate counting. Total daily carbohydrate intake was **personalized** to participant's estimated caloric needs		38:43:20 1779kcal	

Supplement Table S6b: (Continued)

Study, year	Intervention, Control	Intervention or Comparator Diet	GI Dose[a]	GI difference between groups	GL Dose[a]	GL difference between groups	Intervention description	Adherence assessment	Diet (% Carb:Fat:Protein)[b]	Energy Balance[c]
Pavithran et al. 2020				NA		NA		Compliance was evaluated with 24h dietary recall at weeks 3, 11, 12, 18, 23, and 24 by **dietitians**. An FFQ was also collected.		Neutral
	Intervention	LGI diet	~43		~100		Subjects were advised to consume a diet plan with low GI recipes using traditional foods of Kerala cuisine, reinforced by a **dietitian**		62(5):24(4):16(2) 1511 (138)kcal	
	Control	HGI - usual diet	NR		NR		Subjects were adivsed and given instructions to consume a regular diet		66(5):21(5):16(3) 1450(157)kcal	
Rizkalla et al. 2004				32.3		~-77¥	Patients were given a list of recommended daily intake of commonly used foods and a substitution list to exchange within food groups. Subjects received individual counseling by a **dietitian** regarding food intake. Dietary intake was **personalized** according to usual dietary intake	To assess compliance, patients were asked to record food intake the last 7 days of each dietary period, which were then analyzed by a computer program		Neutral
	Intervention	LGI diet	39.0 (3.46)		~78¥		Carbohydrate items with a GI <45 on the glucose scale was recommended (pumpernickel, pasta, lentils, haricot beans, chickpeas, mung beans)		42:37:21 2222kcal	
	Control	HGI diet	71.3 (4.50)		~155¥		High GI foods >60 were recommended (wholemeal bread, baguettes, potatoes, and white rice)		42:37:20 2291kcal	
Visek et al. 2014				18		~-31¥	Subjects were instructed by a **dietitian** and obtained a recommended diet plan with instructions to keep a daily record of meal composition and ingredient weight.	Food records were reviewed by a **dietitian** on a biweekly basis and **personally** adjusted the diets; no other assessment of adherence		Neutral
	Intervention	LGI diet	49 (2)¶¶		~78¥		Subjects were given a list of meals and cookbook for low GI foods (<55), including pasta, legumes, wholemeal products, and advised to avoid higher GI foods such as potatoes and white bread		~37.2:36:18 1676kcal	
	Control	Standard DM Diet	67 (9)¶¶		~109¥				~36.2:40:17.3 1745kcal	
Wolever et al. 1992				28		~-56¥	For the first and last two wks of each period, subjects were provided with preweighed portions of all starchy foods, cheese, and tinned sauces in their diets. In the middle 2wk, subjects followed a detailed menu similar to that during metabolic periods	NR		Negative
	Intervention	LGI diet	58		~114¥				57:23:20 1388kcal	
	Control	HGI diet	86		~170¥				57:23:20 1388kcal	
Wolever et al. 2008				8.1		2	Subjects chose to receive 16-21 key foods (starchy carbohydrates with a GI between 24-29) of their choice from a list of foods during dietary interventions, and received a list of key foods to consume during their intervention period. Subjects received **personalized** advice from a **dietitian** at each study visit (2 and 4 wk after randomization, then every 4 wk)	Subjects recorded daily intake of key foods at 1, 3, 6, and 9 months after randomization; no other assessment of adherence		Neutral
	Intervention	LGI diet	55.1 (3.0)		133 (14.8)		Key foods included olive and canola oils or spreads, nuts, and other foods low in SFAs and high in MUFAs, to replace carbohydrate foods		51.9 (6.7):26.5 (5.9):20.6(3.0) 1800(371)kcal	
	Control	HGI diet	63.2 (2.8)		135 (20.8)		Adivce focused on following a healthy low-fat diet and avoiding low-GI foods		46.5 (6.2):30.8 (4.8):20.4 (2.8) 1890(333)kcal	
Yusof et al. 2009				7		23	**Personalized** dietary advice was given by the **same dietitian**	Diet was assesed with a 3-day food diaries at baseline and weeks 4 and 12, which were reviewed with subjects. Adherence to dietary instruction was assessed by a **dietitian**		Neutral
	Intervention	LGI diet	57(6)		108(32)		Subjects were instructed to eat at least one low-GI food from a list provided. Key foods and sample menus were provided to subjects		52(4):30(4):18(3) 1512(325)kcal	
	Control	HGI diet	64(5)		131(30)		Subjects were instructed to eat a set number of carbohydrate exchanges for each meal and advised to limit the use of refined sugars without referring to the GI concept. An exchange list and sample menu were provided to subjects		54(4):28(5):17(3) 1526(328)kcal	

A, agency; ADA, American Diabetes Association; C, crossover; Carb, carbohydrate; DA, dietary advice; F, female; F/U, follow-up; GI, glycemic index; GL, glycemic load; HbA1c, hemoglobin A1c; HGI, high-glycemic index; I, industry; LGI, low-glycemic index; M, male; Met, metabolic; MUFA, monounsaturated fatty acid; NR, not reported; OP, outpatient; P, parallel; SD, standard deviation; Supp, supplemental feeding control; T1DM, type-1 diabetes mellitus; T2DM, type-2 diabetes mellitus; UK, United Kingdom; USA, United States of America; wks, weeks; yr, year

a The majority of GI/GL values are based on reported in-trial achieved intakes based on food records, with the exception of 2 trials (denoted by ǂ) which reported prescribed GI values (Heilbronn et al. 2002, however they provided key foods which represented 60% of the total energy intake; and Giacco et al. 2000, however they reported 83% adherence in the intervention group). GI units are on the glucose scale. For Brand et al. 1991 and Giacco et al. 2000, although not explicitly noted in the papers, reported values were assumed to be on the bread scale so were converted (*0.71) to the glucose scale. All other studies which reported on the bread scale were also converted to the glucose scale for consistency. For 15 trials, GL values were not reported, however it was possible to calculate the GL using the GI and carbohydrate data provided (denoted by ¥).
b based on in-trial achieved macronutrient intakes, unless otherwise indicated (ǂ)
c Negative energy balance refers to a deficit in normal energy intake and/or intake below energy requirements. Neutral energy balance refers to the maintenance of usual energy intake and/or meeting energy requirements.

* Calculated before dropout
**Completer analysis, as used in data analysis
¶¶ median and interquartile range (IQR)
ǂ reported values based on prescribed intervention/control (not in-trial achieved intakes)
¥ GL values were calculated based on the GI and carbohydrate data reported
§ based on 6-month data (Fraser et al. Diabetologia. 2008;51:1616–1622)
£ Although it was not explicitly written it was a weight loss program, it was implied as such due to calorie counting and calorie levels of 1200-1500kcal for obese participants.

Supplemental Table S7: Medication and insulin changes

Trial	Effect on medication/insulin use
CHILDREN	
Collier et al. 1988	Only 1 subject substantially changed his insulin dose, a decrease in 13 units on the test diet and an increase of 13 units on control. Two other subjects made small alterations of <4 units. The overall mean change of insulin dose was not significantly different for the control (increase 3.4 (1.8) U/d) or the low-GI periods (decrease 2.3 (1.9) U/d)
Gilbertson et al. 2001	No significant differences in insulin dose at 12 months nor were there changes to insulin dose over the 12 months. Baseline control/carbohydrate exchange diet = 0.9 (0.3) U/kg; end control = 1.0 (0.3) U/kg; baseline low-GI = 1.0 (0.3) U/kg; end low-GI = 1.1 (0.3) U/kg
ADULTS	
Brand et al. 1991	NR
Cai et al. 2017	NR
Elhayany et al. 2010 - LGI	NR
Elhayany et al. 2010 - LGL	NR
Fabricatore et al. 2011	NR
Fontvieille et al. 1988	Significant decrease in daily insulin needs was observed when following the low-GI diet compared to the high GI diet (P<0.05). On the low-GI diet, daily insulin needs decreased for 6/8 patients.
Fontvieille et al. 1992	No significant difference between diets observed for insulin or drug requirements. End values for insulin High GI = 42 (16) U/d; low-GI = 41 (15) U/d.
Frost et al. 1994	N/A (not on medication)
Giacco et al. 2000	No significant difference in change in insulin dose was observed between the diets.
Gomes et al. 2017	NR
Heilbronn et al. 2002	N/A (not on medication)
Järvi et al. 1999	NR
Jenkins et al. 2008	In ITT analysis, antihyperglycemic medication dosages increased similarly in both groups (3 in low-GI diet and 3 in high cereal fibre diet), but reductions were more frequent in low-GI group (13 in low-GI vs. 4 in high cereal fibre, P=0.06).
Jenkins et al. 2012	Oral antihyperglycemic medication dosages increased in 2 participants of the high wheat fibre group, and decreased in 3 participants (1 from high wheat fiber, 2 from low-GI diet). Changes in medication were not different between groups (P=0.85).

Jenkins et al. 2014	Oral antihyperglycemic medication dosages increased in 1 and reduced in 5 participants on the test diet. Decreased in 4 participants on the control diet. No significant treatment differences. Serum lipid-lowering medications were decreased in 1 from test and 3 from control diet, no significant treatment difference in medication use.
Jimenez-Cruz et al. 2003	NR
Jimenez-Cruz et al. 2004	NR
Komindr et al. 2001	NR
Luscombe et al. 1999 - HGI	NR
Luscombe et al. 1999 - MUFA	NR
Ma et al. 2008	In ADA (control) group, two subjects decreased medication use and four added medication/increase dose at 6 months; between 6-12 months, 4 participants added or increased medication dose. In the low-GI group, 3 subjects decreased medication use and one increased at 6 months. Between 6-12 months, one participant decreased and two added medication or dose. Low-GI group had lower likelihood of switching to a new drug or increasing diabetes medication dosage (Odds ratio = 0.26, P=0.01)
Pavithran et al. 2020	NR
Rizkalla et al. 2004	NR
Visek et al. 2014	NR
Wolever et al. 1992	NR
Wolever et al. 2008	N/A (not on medication)
Yusof et al. 2009	One subject in GI group started insulin therapy.

ADA, American diabetes association diet; GI, glycemic index; ITT, intention-to-treat; LGI, low-glycemic index; LGL, low-glycemic load; MUFA, monounsaturated fatty acids; N/A, not application; NR, not reported

Supplemental Table S8: Acceptability Results*

Study	Assessment of Diet Acceptability
Gilbertson et al. 2001	The 53 children (and their parents) that had experienced both types of dietary approaches expressed an overall preference for the low-GI diet compared with the carbohydrate diet (P<0.01 and P<0.001 for the children and parents, respectively). The same subgroup of parents believed that the low-GI diet led to better control of blood glucose levels compared with the carbohydrate diet (P<0.001). The low-GI diet was the dietary regime that most parents and children selected to continue after completion of the study (P<0.001 and P<0.001 for the children and parents, respectively).
Jenkins et al. 2014	Participants ranked their level of satiety on a scale of 24 (starved/feeling weak) to +4 (painfully full) and palatability of study breads and diets at each visit on a scale of 1–10 (1 = strongly dislike, 10 = like very much). The test bread was rated more palatable than the control bread, as was the overall test diet compared with the control diet (P=0.002 and P=0.002, respectively).
Jimenez-Cruz et al. 2004	Individual questioning of subjects established that both diets were found acceptable and the diet plans were found easy to follow.
Luscombe et al. 1999 - HGI Luscombe et al. 1999 - MUFA	Questionnaires completed midway and at the end of each dietary intervention revealed all diets were well accepted and there were no significant differences in the ratings of the three diets in overall score of acceptability, taste, satiety or variety.
Ma et al. 2008	Participants completed a questionnaire at the end of the study to assess the acceptability of the study. Both groups of participants liked the diet they were prescribed (100% in the GI versus 88% in the ADA group; P=0.49). Additionally, all participants in the low-GI group reported the intervention was helpful versus 77% in the ADA group (P=0.11). Thirty-five percent of ADA group versus 23% of low-GI group reported that it was difficult for them to maintain the new diet (P=0.69). All participants in the low-GI group and 71% of those in the ADA group reported enjoying eating unfamiliar foods (P=0.05). There were no diet-related adverse events reported in either group during the study.

Rizkalla et al. 2004	The 12 subjects followed the two dietary periods of 4 weeks each without any difficulty. According to self-report, subjects' lifestyle was unchanged throughout the entire study.

*Seven of the 29 trials reported some assessment of acceptability. Note that one trial by Giacco et al. 2000 reported on gastrointestinal side effects of which 56% participants treated with the high-fibre/low-GI diet, recorded some minor gastrointestinal side effects (flatulence, meteorism, and diarrhea) in comparison with 40% of the those treated with the low-fibre/higher GI diet ($P>0.05$). However, none of these episodes induced patients to discontinue.

ADA, American diabetes association diet; GI, glycemic index; HGI, high-glycemic index; MUFA, monounsaturated fatty acids

Supplemental Table S9: Sensitivity analyses of the use of correlation coefficients of 0.25 and 0.75

	MD (95% CI), P-value I^2, P-value		
	Correlation Coefficient used in the Primary Analysis	**Correlation Coefficient used in Sensitivity Analyses**	
Outcome	**0.5**	**0.25**	**0.75**
Glycemic control			
HbA1c, %	-0.31 [-0.43, -0.19], P<0.001 I^2=75%, P_{het}<0.001	-0.31 [-0.43, -0.20], P<0.001 I^2=72%, P_{het}<0.001	-0.29[-0.42, -0.17], P<0.001 I^2=79%, P_{het}<0.001
Fasting glucose, mmol/L	-0.36 [-0.49, -0.23], P<0.001 I^2=54%, P_{het}<0.001	-0.34 [-0.46, -0.21], P<0.001 I^2=45%, P_{het}=0.007	-0.41 [-0.54, -0.27], P<0.001 I^2=67%, P_{het}<0.001
Fasting insulin, pmol/L	-2.66 [-8.82, 3.50], P=0.397 I^2=38%, P_{het}=0.091	-4.32 [-9.87, 1.23], P=0.127 I^2=23%, P_{het}=0.221	-0.59 [-7.04, 5.86], P=0.858 I^2=59%, P_{het}=0.005
Blood lipids			
LDL-C, mmol/L	-0.17 [-0.25, -0.08], P<0.001 I^2=70%, P_{het}<0.001	-0.18 [-0.27, -0.09], P<0.001 I^2=67%, P_{het}<0.001	-0.15 [-0.24, -0.07], P<0.001 I^2=76%, P_{het}<0.001
Non-HDL-C, mmol/L	-0.20 [-0.33, -0.07], P=0.002 I^2=70%, P_{het}<0.001	-0.17 [-0.29, -0.06], P=0.004 I^2=47%, $P_{het=}$0.006	-0.23 [-0.38, -0.08], P=0.003 I^2=89%, P_{het}<0.001
HDL-C, mmol/L	0.01 [-0.01, 0.04], P=0.351 I^2=57%, P_{het}<0.001	0.01 [-0.02, 0.04], P=0.495 I^2=46%, P_{het}=0.005	0.01 [-0.02, 0.04], P=0.514 I^2=74%, P_{het}<0.001
Triglycerides, mmol/L	-0.09 [-0.17, -0.01], P=0.035 I^2=44%, P_{het}=0.010	-0.09 [-0.17, -0.01], P=0.029 I^2=32%, P_{het}=0.062	-0.09 [-0.18, -0.01], P=0.027 I^2=63%, P_{het}<0.001
ApoB, g/L	-0.05 [-0.09, -0.01], P=0.026 I^2=58%, P_{het}=0.034	-0.05 [-0.09, -0.01], P=0.019 I^2=56%, P_{het}=0.043	-0.04 [-0.08, -0.001], P=0.045 I^2=63%, P_{het}=0.019

Adiposity			
Body weight, kg	-0.66 [-0.90, -0.42], P<0.001 I^2=0%, P_{het}=0.999	-0.67 [-0.91, -0.43], P<0.001 I^2=0%, P_{het}=0.999	-0.65 [-0.88, -0.41], P<0.001 I^2=0%, P_{het}=0.997
BMI, kg/m^2	-0.38 [-0.64, -0.13], P<0.001 I^2=0%, P_{het}=0.999	-0.43 [-0.70, -0.15], P=0.002 I^2=0%, P_{het}=0.999	-0.30 [-0.52, -0.09], P=0.005 I^2=0%, P_{het}=0.990
Waist circumference, cm	-0.67 [-1.76, 0.42], P=0.226 I^2=79%, P_{het}<0.001	-0.67 [-1.77, 0.43], P=0.235 I^2=79%, P_{het}<0.001	-0.68 [-1.72, 0.37], P=0.206 I^2=79%, P_{het}<0.001
Blood Pressure			
Systolic blood pressure, mmHg	-0.14 [-2.24, 1.96], P=0.894 I^2=53% P_{het}=0.029	-0.19 [-2.30, 1.92], P=0.858 I^2=52%, P_{het}=0.032	-0.04 [-2.12, 2.03], P=0.968 I^2=55%, P_{het}=0.023
Diastolic blood pressure, mmHg	-0.50 [-1.85, 0.86], P=0.473 I^2=63%, P_{het}=0.009	-0.47 [-1.85, 0.91], P=0.503 I^2=63%, P_{het}=0.009	-0.55 [-1.86, 0.77], P=0.413 I^2=63%, P_{het}=0.008
Inflammation			
CRP, mg/L	-0.41 [-0.78, -0.04], P=0.031 I^2=24%, P_{het}=0.255	-0.41 [-0.78, -0.05], P=0.027 I^2=22%, P_{het}=0.266	-0.39 [-0.77, -0.01], P=0.044 I^2=28%, P_{het}=0.226

ApoB, apolipoprotein B; BMI, body mass index; CI, confidence interval; CRP, c-reactive protein; HbA1c, hemoglobin A1c; Het, heterogeneity; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; MD, mean difference; no., number; Non-HDL-C, non-high-density lipoprotein-cholesterol

Supplemental Table S10: GRADE assessment of study quality

Certainty assessment*									Effect		Certainty	
			Downgrades					Upgrades				
Outcomes	No of trial comparisons	Study design	Risk of bias**	Inconsistency	Indirectness***	Imprecision	Publication Bias	Dose Response	MD [95% CIs]	Interpretation of magnitude of effect****		
Glycemic control												
HbA1c, %	22	RCTs	not serious	not serious[a]	not serious	serious[b]	not serious	Linear DR[c]	-0.31 [-0.42, -0.19] Beta 0.004 [0.000, 0.008]	small important effect	⊕⊕⊕⊕	High
Fasting glucose, mmol/L	26	RCTs	not serious	not serious[d]	not serious	serious[e]	not serious[f]	None[g]	-0.36 [-0.49, -0.23]	trivial effect	⊕⊕⊕◯	Moderate
Fasting insulin, pmol/L	12	RCTs	not serious	not serious	not serious	serious[h]	serious[i]	None	-2.66 [-8.82, 3.50]	no effect	⊕⊕◯◯	Low
Blood lipids												
LDL-C, mmol/L	26	RCTs	not serious	serious[j]	not serious	serious[k]	not serious	None	-0.17 [-0.25, -0.08]	small important effect	⊕⊕◯◯	Low
Non-HDL-C, mmol/L	25	RCTs	not serious	not serious[l]	not serious	serious[m]	not serious	None	-0.20 [-0.33, -0.07]	moderate effect	⊕⊕⊕◯	Moderate
HDL-C, mmol/L	26	RCTs	not serious	not serious[n]	not serious	not serious[o]	not serious	None[p]	0.01 [-0.01, 0.04]	trivial to no effect	⊕⊕⊕⊕	High
Triglycerides, mmol/L	26	RCTs	not serious	not serious[q]	not serious	serious[r]	not serious	Linear DR[s]	-0.09 [-0.17, -0.01] Beta 0.004 [0.000, 0.007]	small important effect[t]	⊕⊕⊕◯	Moderate
ApoB, g/L	5	RCTs	not serious	not serious[u]	not serious	serious[v]	not serious[w]	None	-0.05 [-0.09, -0.01]	small important effect	⊕⊕⊕◯	Moderate
Adiposity												
Body weight, kg	24	RCTs	not serious	not serious	not serious	serious[x]	not serious	None	-0.66 [-0.90, -0.42]	small important effect	⊕⊕⊕◯	Moderate
BMI, kg/m^2	20	RCTs	not serious	not serious	not serious	serious[y]	not serious	None	-0.38 [-0.64, -0.13]	moderate effect	⊕⊕⊕◯	Moderate
Waist circumference, cm	10	RCTs	not serious	serious[z]	not serious	serious[aa]	not serious	None[ab]	-0.67 [-1.78, 0.42]	trivial to no effect	⊕⊕◯◯	Low
Blood pressure												
SBP, mmHg	9	RCTs	not serious	not serious[ac]	not serious	serious[ad]	not serious[w]	Linear DR[ae]	-0.14 [-2.24, 1.96] Beta 0.49 [0.09, 0.89]	small important effect[af]	⊕⊕⊕◯	Moderate
DBP, mmHg	8	RCTs	not serious	not serious[ag]	not serious	serious[ad]	not serious[w]	None[ah]	-0.50 [-1.85, 0.86]	no effect	⊕⊕⊕◯	Moderate
Inflammation												
CRP, mg/L	6	RCTs	not serious	not serious	not serious	serious[ai]	not serious[w]	None	-0.41 [-0.78, -0.04]	trivial effect	⊕⊕⊕◯	Moderate

Apo-B, apolipoprotein-B; BMI, body mass index; CI, confidence interval; CRP, c-reactive protein; DBP, diastolic blood pressure; HbA1c, hemoglobin A1c; LDL-C, low-density lipoprotein-cholesterol; HDL-C, high-density lipoprotein-cholesterol; MD, mean difference; N/A, not applicable; No, number; Non-HDL-C, non-high-density lipoprotein-cholesterol; RCTs, randomized controlled trials; SBP, systolic blood pressure

*Since all included studies were randomized controlled trials, the certainty of the evidence was graded as high for all outcomes by default and then downgraded based on pre-specified criteria. **Risk of Bias** - Downgraded if the majority of studies were considered to be at high risk of bias. **Inconsistency** - For inconsistency, we downgraded if there was serious inconsistency as evidence of substantial heterogeneity ($I^2 \geq 50\%$, $P < 0.10$) that was unexplained by any a priori sensitivity or subgroup analyses. If there was evidence of substantial unexplained heterogeneity by these criteria, then we confirmed this assessment by supplementing the approach with visual inspection of forest plots for the 2 additional criteria specified in the GRADE handbook: the presence of wide variance of point estimates across studies and minimal to no overlap of CIs for some studies (https://gdt.gradepro.org/app/handbook/handbook.html#h.g2dqzi9je57e). **Indirectness** - Downgraded if there were factors present relating to the participants, interventions, or outcomes that limited the generalizability of the results. **Imprecision** - Downgraded if the 95% confidence interval (95% CI) crossed the minimally important difference (MID) for benefit or harm. MIDs used for each outcome were: 0.3% for HbA1c (Committee for Medicinal Products for Human Use (CHMP). Guideline on clinical investigation of medicinal products in the treatment or prevention of diabetes mellitus (Draft Guidance). CPMP/EWP/1080/00 Rev. 2. London, UK. European Medicines Agency, 29 January 2018.), 0.5mmol/L for fasting glucose (David M. Nathan, Judith Kuenen, Rikke Borg, Hui Zheng, David Schoenfeld, and Robert J. Heine, for the A1c-Derived Average Glucose (ADAG) Study Group. Diabetes Care 2008 https://professional.diabetes.org/diapro/glucose_calc), 5pmol/L for fasting insulin (Proportional reduction to fasting glucose), 0.1mmol/L for LDL-C, HDL-C, non-HDL-C, and triglycerides (Baigent C, Blackwell L, Emberson J, et al. Efficacy and safety of more intensive lowering of LDL cholesterol: a meta-analysis of data from 170,000 participants in 26 randomised trials. Lancet 2010;376:1670-1681), 0.04g/L for apo-B, 0.5kg for body weight (Johnston BC, Kanters S, Bandayrel K, Wu P, Naji F, Siemieniuk RA, et al. Comparison of weight loss among named diet programs in overweight and obese adults: a meta-analysis. JAMA 2014;312(9):923e33), 0.2kg/m^2 for BMI, 2cm for waist circumference, 2mmHg for systolic and diastolic blood pressure (Lewington S, Clarke R, Qizilbash N, Peto R, Collins R; Prospective Studies Collaboration. Age specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet 2002;360:1903–1913) and 0.5mg/L for CRP (Reynolds Risk Score. Available at: http://www.reynoldsriskscore.org/Default.aspx [Accessed March 14, 2019]. Ridker, P.M. et al., 2008. C-reactive protein and parental history improve global cardiovascular risk prediction: the Reynolds Risk Score for men. Circulation, 118(22), pp.2243–51, 4p following 2251. Available at: http://dx.doi.org/10.1161/CIRCULATIONAHA.108.814251. Ridker, P.M. et al., 2007. Development and validation of improved algorithms for the assessment of global cardiovascular risk in women: the Reynolds Risk Score. JAMA: the journal of the American Medical Association, 297(6), pp.611–619. Available at: http://dx.doi.org/10.1001/jama.297.6.611.). **Other** – Downgraded if there was evidence of small study effects. **Upgrades** were applied if dose response analyses were justified to provide compelling evidence to warrant an upgrade to the certainty of evidence. Please refer to the **Supplemental Methods** for further details.

** No serious risk of bias since all studies were rated as either low or unclear risk of bias for each category.

***Indirectness was not downgraded for all of the outcomes since there were a variety of included studies spanning globally.

**** For the interpretation of the magnitude, we used the MIDs to assess the importance of magnitude of our point estimate using the effect size categories according to new GRADE guidance (please refer to **Supplemental Methods**).

a Although there was substantial heterogeneity in the analysis, we did not downgrade for serious inconsistency, since it was explained when the study by Cai et al. 2017 was removed as part of a priori sensitivity analyses (Original: I^2=74%, P-heterogeneity<0.001; after study removed: I^2=41%, P-heterogeneity=0.026).

b Downgrade for serious imprecision for the effect of low-GI/GL diets on HbA1c, as the 95% CIs (-0.42 to -0.19%) overlap with the minimally important difference for clinical benefit (-0.3%).

c Upgrade for positive linear dose response gradient for difference in GL and HbA1c (coefficient 0.004% [95% CI 0.000 to 0.008%], P=0.032).

d Although there was substantial heterogeneity in the analysis, we did no downgrade for serious inconsistency, since it was explained when the study by Jenkins et al. 2012 was removed as part of a priori sensitivity analyses (Original: I^2=54%, P-heterogeneity<0.001; after study removed: I^2=32%, P-heterogeneity=0.065).

e Downgrade for serious imprecision for the effect of low-GI/GL diets on fasting glucose, as although the 95% CIs (-0.49 to -0.23mmol/L) did not overlap with the minimally important difference for clinical benefit (-0.5mmol/L), the effect is not clinically relevant (<minimally important difference).

f Although asymmetry was detected in the funnel plot and the Egger's test was significant (P<0.001), the trim-and-fill method demonstrated no evidence of small-study effects for fasting glucose, where the imputation of 6 trials did not alter the mean difference and p-value (Original MD -6.01pmol/L [95% CI -10.91 to -1.11pmol/L]).

g Although we observed a reduction in fasting glucose across all trial comparisons with a non-linear dose response showing a positive linear dose response gradient up to a prescribed or in-trial achieved GI of about 50 after which it appears to plateau, we did not upgrade for this dose response because the magnitude of effect remained trivial (<1 MID, 0.5mmol/L) over the dose response range.

h Downgrade for serious imprecision for the effect of low-GI/GL diets on fasting insulin, as the 95% CIs (-8.82 to 3.50pmol/L) overlap with the minimally important difference (5pmol/L).

i Downgrade for evidence of small study effects for the effect of low-GI/GL dietary patterns on fasting insulin. Asymmetry was detected in the funnel plot and the Egger's test was significant (P=0.022). The trim-and-fill method demonstrated evidence of small-study effects for fasting insulin, where the imputation of 5 studies altered the mean difference and p-value (MD -2.66mmol/L [95% CI -8.82 to 3.49mmol/L]; imputed MD=-6.68 [95% CI -11.99 to -1.37mmol/L).

j Downgrade for serious inconsistency for the effect of low-GI/GL diets on LDL-C, due to substantial unexplained heterogeneity I^2=70%, P-heterogeneity<0.001.

k Downgrade for serious imprecision for the effect of low-GI/GL diets on LDL-C, as the 95% CIs (-0.25 to -0.08mmol/L) overlap with the minimally important difference for benefit (-0.1mmol/L).

l Although there was heterogeneity in the analysis, we did not downgrade for serious inconsistency, since it was explained when the one study by Jimenez-Cruz et al. 2004 was removed as part of a priori sensitivity analyses (Original: I^2=70%, P-heterogeneity<0.001; after study removed: I^2=34%, P-heterogeneity=0.055).

m Downgrade for serious imprecision for the effect of low-GI/GL diets on Non-HDL-C, as the 95% CIs (-0.33 to -0.07mmol/L) overlap with the minimally important difference for benefit (-0.1mmol/L).

n Although there was heterogeneity in the analysis, we did not downgrade for serious inconsistency for the effect of low-GI/GL diets on HDL-C, since it was explained by the removal of Jenkins et al. 2012, Elhayany et al. 2010 – LGL or Jenkins et al. 2008 (Original: I^2=57%, P-heterogeneity<0.001; after removal: I^2=43%, P-heterogeneity=0.014, I^2=45%, P-heterogeneity=0.008, I^2=49%, P-heterogeneity=0.003, respectively).

o No downgrade for imprecision for the effect of low-GI/GL diets on HDL-C since the 95% CIs (-0.01 to 0.04mmol/L) does not overlap with the minimally important difference (0.1mmol/L).

p Although we observed a non-linear dose response for the effect of low-GI/GL diets on HDL-C, we did not upgrade for this dose response because the magnitude of effect remained trivial (<1 MID, 0.1mmol/L) over the dose response range.

q No downgrade for serious inconsistency for the effect of low-GI/GL diets on triglycerides since I^2<50% (I^2=44%, P-heterogeneity=0.010).

r Downgrade for serious imprecision for the effect of low-GI/GL diets on triglycerides, as the 95% CIs (-0.17 to -0.01mmol/L) overlap with the minimally important difference for benefit (-0.1mmol/L) and there was instability in the estimate, as the individual removal of eight different trial comparisons in sensitivity analyses resulted in the loss of significance (ranging from P=0.051 to 0.075).

s Although we observed a linear dose response gradient for difference in GL and triglycerides, we did not upgrade since this was based on a sensitivity analysis with the removal of an outlier (Original: coefficient 0.003mmol/L [95% CI -0.001 to 0.006mmol/L], P=0.204; Sensitivity: coefficient 0.004mmol/L [95% CI 0.000 to 0.007mmol/L], P=0.043).

t Although the significant effect by the MD estimate was trivial, there was a positive linear dose response gradient for triglycerides (over the difference in GL range of -76.7 to 5.3, coefficient 0.004mmol/L [95% CI 0.000 to 0.007mmol/L] P=0.043, with the removal of a single outlier) and based on this dose response, the reduction in triglycerides met the criteria for a small important reduction in triglycerides (greater than one MID for benefit, ≥0.1mmol/L) where the reduction in GL is approximately ≥35.

u Although there was heterogeneity in the analysis, we did not downgrade for serious inconsistency for the effect of low-GI/GL diets on apoB, since it was explained when the one study by Wolever et al. 2008 was removed as part of a priori sensitivity analyses (Original: I^2=58%, P-heterogeneity=0.034; after study removed: I^2=38%, P-heterogeneity=0.168).

v Downgrade for serious imprecision for the effect of low-GI/GL diets on apoB, as the 95% CIs (-0.09 to -0.01g/L) overlap with the minimally important difference (0.04g/L) and there was instability in the estimate, as the individual removal of two different trial comparisons in sensitivity analyses resulted in the loss of significance (ranging from P=0.180 to 0.210).

w No downgrade for publication bias, as publication bias could not be assessed (for apoB, systolic or diastolic blood pressure or CRP) due to lack of power for assessing funnel plot asymmetry and small study effects (<10 trial comparisons included in the meta-analysis).

x Downgrade for serious imprecision for the effect of low-GI/GL diets on body weight, as the 95% CIs (-0.90 to -0.42kg) overlap with the minimally important difference for benefit (-0.5kg).

y Downgrade for serious imprecision for the effect of low-GI/GL diets on BMI, as the 95% CIs (-0.64, -0.13kg) overlap with the minimally important difference for benefit (-0.2kg/m^2).

z Downgrade for inconsistency for the effect of low-GI/GL diets on waist circumference due to substantial heterogeneity in the analysis (I^2=79%). Although the heterogeneity in the analysis is explained by the removal of Jenkins et al. 2014, this is a large trial which contributes a large proportion of the weight (19.78%) to the pooled estimate.

aa Downgrade for serious imprecision for the effect of low-GI/GL diets on waist circumference since removal of Jenkins et al. 2014 alters the significance of the estimate from non-significant to significant, demonstrating important instability in the estimate (Original MD -0.67cm [95% CI -1.78 to 0.42] P=0.226, I^2=79%, P-het<0.001; after study removed: MD -1.28cm [95% CI -1.95 to -0.60] P<0.001, I^2=25%, P-het=0.223).

ab Although there was a non-linear dose response for absolute test GI (and absolute test GL) and waist circumference, we did not upgrade for dose response because this was based on few observations (n<10), and thus we decided it was not sufficiently compelling to warrant an upgrade to the certainty of evidence.

ac Although there was heterogeneity in the analysis, we did not downgrade for serious inconsistency for the effect of low-GI/GL diets on SBP, since it was explained when the one study by Jenkins et al. 2012 was removed as part of a priori sensitivity analyses (Original: I^2=53%, P-heterogeneity=0.029; after study removed: I^2=0%, P-heterogeneity=0.668).

ad Downgrade for serious imprecision for the effect of low-GI/GL diets on SBP and DBP, as the 95% CIs (-2.24 to 1.96mmHg and -2.14 to 2.26mmHg, respectively) overlap with the minimally important difference (2mmHg).

ae Although there was a dose response for SBP, we did not upgrade for this dose response because it was based on few observations (n<10), and thus we decided it was not sufficiently compelling to warrant an upgrade to the certainty of evidence.

af Although there was no effect by the MD estimate, there was a linear dose response for SBP (over the GI dose range of 43 to 57, coefficient 0.49mmHg [95% CI, 0.09 to 0.89] P=0.016). Based on this dose response, the reduction in SBP met the criteria for a small important reduction in SBP (greater than one MID for benefit, ≥2mmHg) where the in-trial achieved dietary GI is ≤48.

ag Although there was heterogeneity in the analysis, we did not downgrade for serious inconsistency for the effect of low-GI/GL diets on DBP since it was explained when the one study by Jenkins et al. 2012 was removed as part of a priori sensitivity analyses (Original: I^2=63%, P-heterogeneity=0.009; after study removed: I^2=43%, P-heterogeneity=0.104).

ah Although there was a dose response for difference in GL and DBP, we did not upgrade for this dose response because it was based on few observations (n<10), and thus we decided it was not sufficiently compelling to warrant an upgrade to the certainty of evidence.

ai Downgrade for serious imprecision for the effect of low-GI/GL diets on CRP, as the 95% CIs (-0.78 to -0.04mg/L) overlap with the minimally important difference for benefit (-0.5mg/L).

Supplemental Table S11: Potential mechanisms to explain the observed effects of low-GI/GL dietary patterns

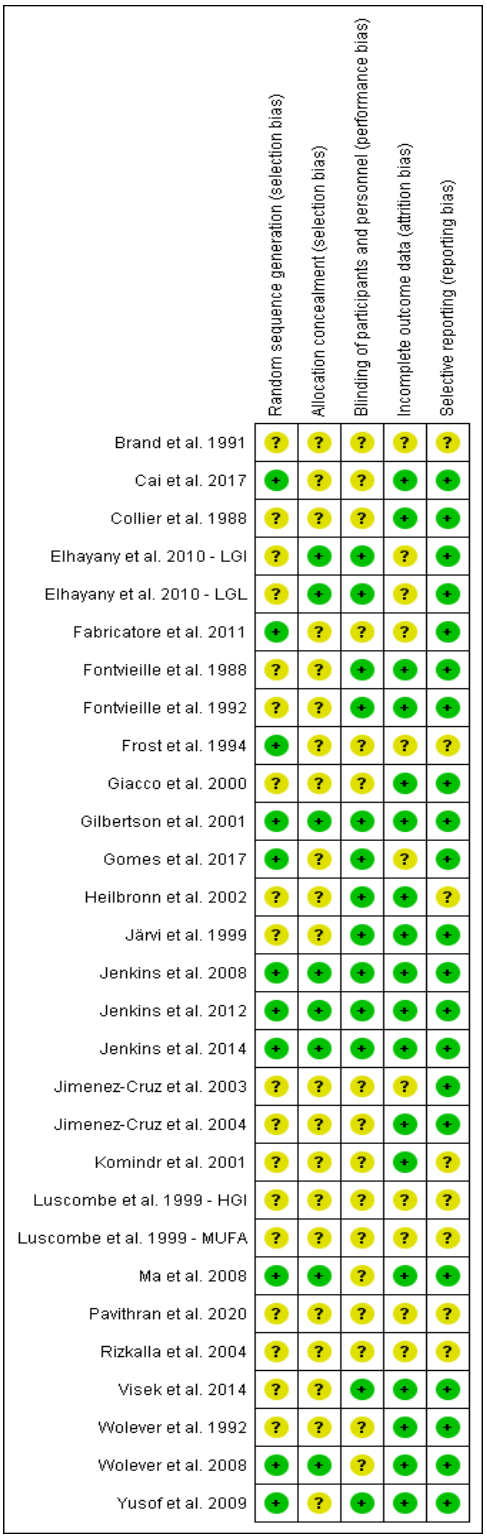
Potential mechanism	Description	references
Low-GI foods slow digestion and reduce absorption	Low-GI foods reduce the rate of carbohydrate absorption and cause a lower rise in blood glucose compared to higher GI foods[1]. Sustained over the longer term through consumption of low-GI/GL diets, the slowed absorption may result in an overall improvement in glycated proteins, as observed in the present analysis as a significant reduction in HbA1c.	1. Jenkins DJ, Kendall CW, Augustin LS, Franceschi S, Hamidi M, Marchie A, Jenkins AL and Axelsen M. Glycemic index: overview of implications in health and disease. Am J Clin Nutr. 2002;76:266S-73S.
Low-GI foods may reduce intrahepatic recycling of bile acids	The higher viscous fiber content of low-GI diets may also explain the cholesterol-lowering effects[2-4] observed in the present analysis for LDL-C and non-HDL-C. Higher prescribed or in-trial achieved fibre intake on the low-GI/GL diets or the difference in fibre between the low-GI/GL and control diets was associated with a reduction in LDL-C and non-HDL-C ($P<0.05$), where higher fibre in both cases resulted in greater reductions.	2. Wolever TM, Tosh SM, Gibbs AL, Brand-Miller J, Duncan AM, Hart V, Lamarche B, Thomson BA, Duss R and Wood PJ. Physicochemical properties of oat beta-glucan influence its ability to reduce serum LDL cholesterol in humans: a randomized clinical trial. Am J Clin Nutr. 2010;92:723-32. 3. Jenkins DJ, Wolever TM, Rao AV, Hegele RA, Mitchell SJ, Ransom TP, Boctor DL, Spadafora PJ, Jenkins AL, Mehling C and et al. Effect on blood lipids of very high intakes of fiber in diets low in saturated fat and cholesterol. N Engl J Med. 1993;329:21-6. 4. Administration UFaD. Food Labeling: Health Claims; Soluble Fiber from Certain Foods and Coronary Heart Disease. Rockville, MD. Docket No. 96P-0338. 1998.
Low-GI foods reduce glycemic variability	The slowed absorption may also result in reductions in glycemic fluctuations, which may also contribute to an overall improvement in glycated proteins. Reduced glycemic fluctuations may lower the demand for insulin and thus reduce circulating insulin along with related gastrointestinal incretin hormone, such as gastric inhibitory polypeptide (GIP) and glucagon-like peptide-1 (GLP-1)[5]. Glycemic variability has been demonstrated to activate oxidative stress[6-8] whereas, by creating a more blunted and sustained glycemic response with a low-GI diet, oxidative stress, as well as the production of advanced glycation end products, would be reduced. Thus, this may explain the significant reduction in CRP observed in the present analysis.	5. Drucker DJ. Deciphering metabolic messages from the gut drives therapeutic innovation: the 2014 Banting Lecture. Diabetes. 2015;64:317-26. 6 Ceriello A and Ihnat MA. 'Glycaemic variability': a new therapeutic challenge in diabetes and the critical care setting. Diabetic medicine : a journal of the British Diabetic Association. 2010;27:862-7. 7. Monnier L, Mas E, Ginet C, Michel F, Villon L, Cristol JP and Colette C. Activation of oxidative stress by acute glucose fluctuations compared with sustained chronic hyperglycemia in patients with type 2 diabetes. JAMA. 2006;295:1681-7. 8. Brownlee M and Hirsch IB. Glycemic variability: a hemoglobin A1c-independent risk factor for diabetic complications. JAMA. 2006;295:1707-8.

Low-GI foods improve satiety and hunger	The reduced circulating insulin and related incretin hormones. These effects may explain the greater reductions in body weight and BMI via increased satiety after low-GI meals[9] and delayed hunger and thus a reduced subsequent energy intake[10-11]. Typically, the fibre content of low-GI dietary patterns is higher[12-13], which may also contribute to improvements in satiety and hunger[14].	9. Ludwig DS. Dietary glycemic index and obesity. J Nutr 2000;130:280S–3 10. Colagiuri S, Dickinson S, Girgis S and R C. National Evidence Based Guideline for Blood Glucose Control in Type 2 Diabetes. Diabetes Australia and the NHMRC. 2009. 11. Jenkins DJ, Kendall CW, Augustin LS, Mitchell S, Sahye-Pudaruth S, Blanco Mejia S, Chiavaroli L, Mirrahimi A, Ireland C, Bashyam B, Vidgen E, de Souza RJ, Sievenpiper JL, Coveney J, Leiter LA and Josse RG. Effect of legumes as part of a low-GLycemic index diet on glycemic control and cardiovascular risk factors in type 2 diabetes mellitus: a randomized controlled trial. Arch Intern Med. 2012;172:1653-60. 12. Atkinson FS, Foster-Powell K, Brand-Miller JC. International tables of glycemic index and glycemic load values: 2008. Diabetes Care 2008;31:2281–3 13. Riccardi G, Rivellese AA, Giacco R. Role of glycemic index and glycemic load in the healthy state, in prediabetes, and in diabetes. Am J Clin Nutr 2008;87:269S–74 14. Slavin JL. Dietary fiber and body weight. Nutrition 2005;21:411–8

BMI, body mass index; CRP, c-reactive protein; GI, glycemic index; GIP, gastric inhibitory polypeptide; GL, glycemic load; GLP-1, glucagon-like peptide-1; HbA1c, hemoglobin A1c; LDL-C, low-density lipoprotein-cholesterol; non-HDL-C, non-high-density lipoprotein-cholesterol

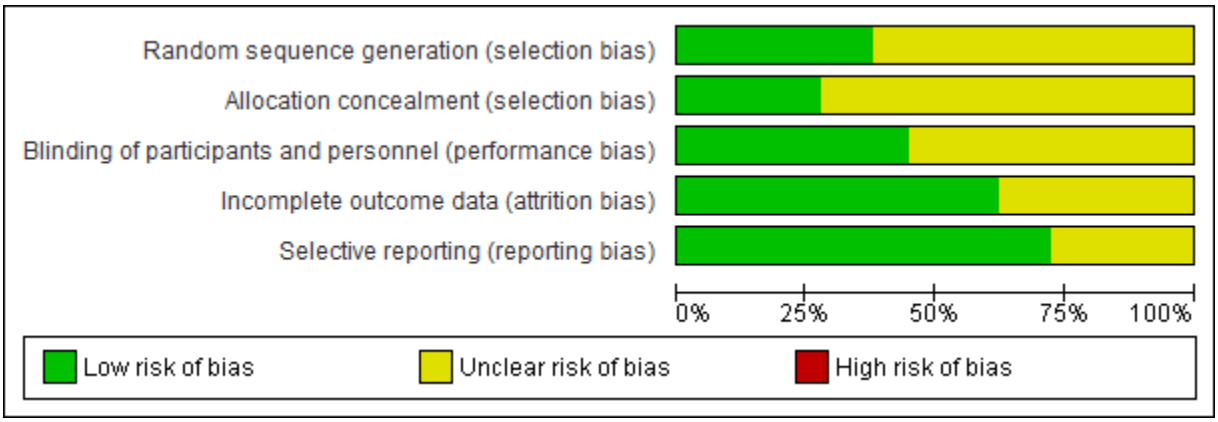
Supplemental Figures

Supplemental Figure S1: Cochrane risk of bias summary for all included trial comparisons



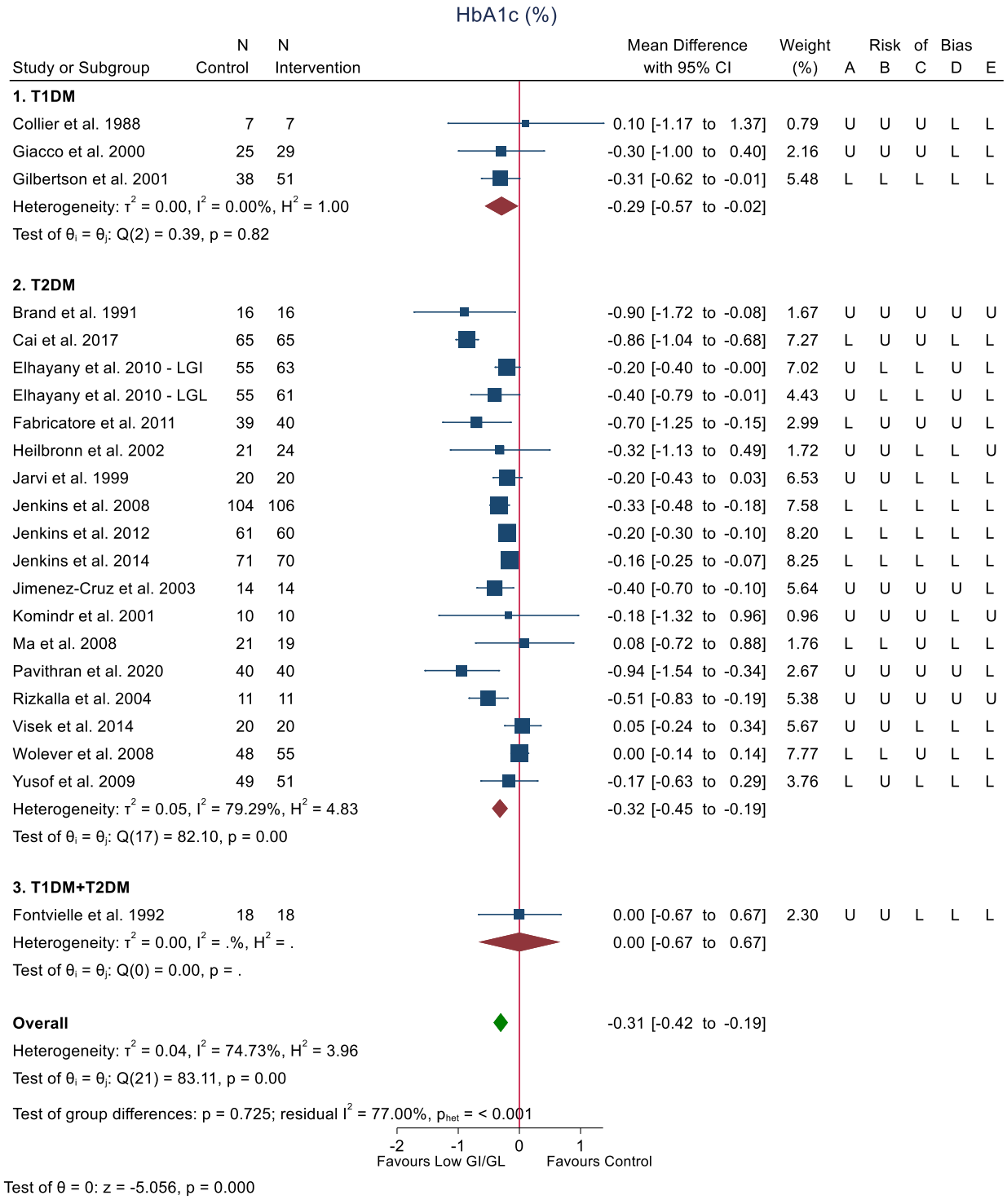
Summary of risk of bias ratings for each individual trial comparison included in the meta-analysis. LGI, low-glycemic index; LGL, low-glycemic load; MUFA, monounsaturated fatty acids

Supplemental Figure S2: Risk of bias proportion graph for all included trial comparisons



Colored bars represent the proportion of trial comparisons assessed as low (green), unclear (yellow) or high (red) risk of bias for the 5 domains of bias above according to criteria set by the Cochrane Risk of Bias tool in the 29 included randomized controlled trial comparisons.

Supplemental Figure S3: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on HbA1c (%) in diabetes

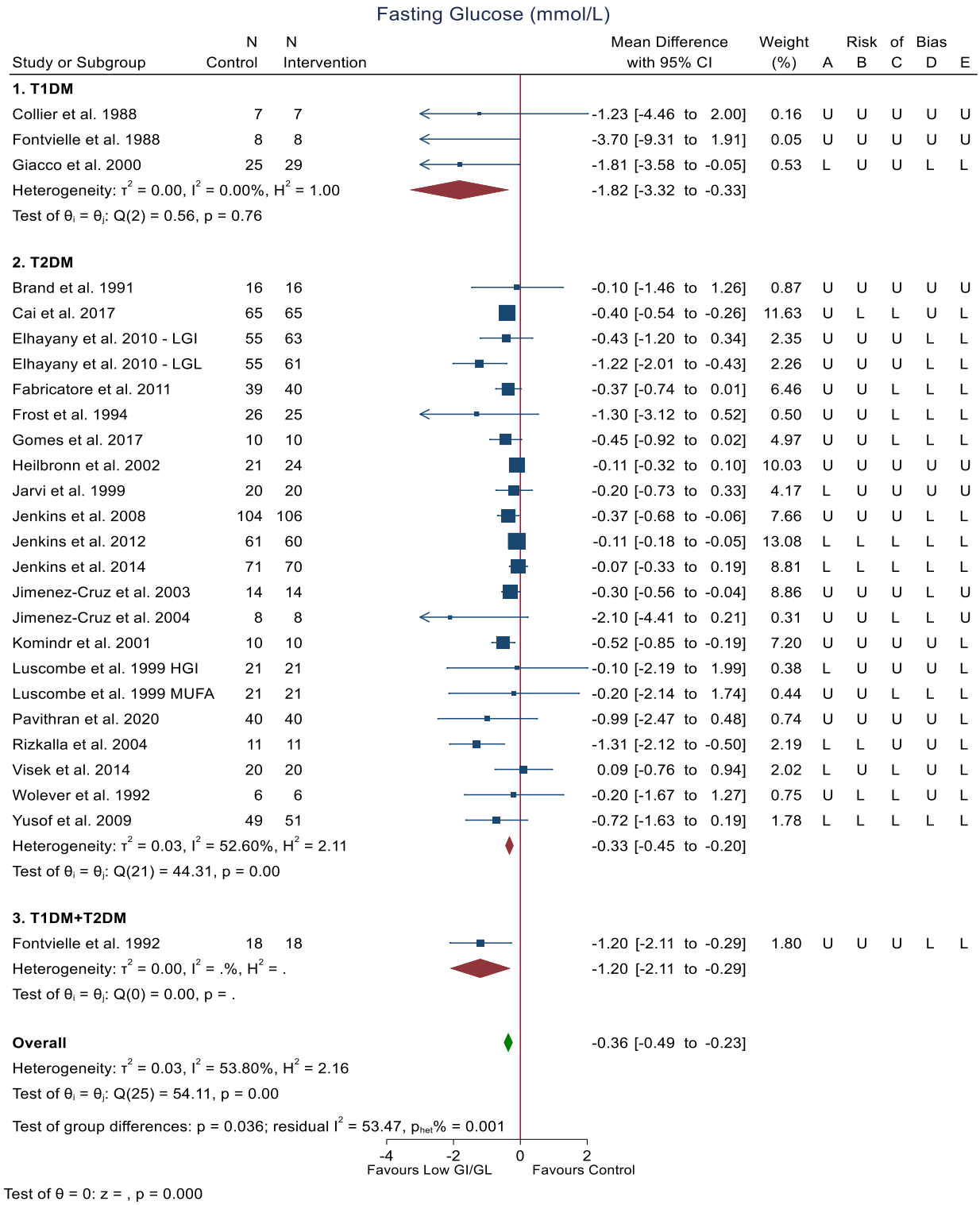


Test of θ = 0: z = -5.056, p = 0.000

Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

CI, confidence interval; GI, glycemic index; GL, glycemic load; HbA1c, hemoglobin A1c; LGI, low-GI; LGL, low-GL; T1DM, type 1 diabetes; T2DM, type 2 diabetes

Supplemental Figure S4: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on fasting glucose (mmol/L) in diabetes

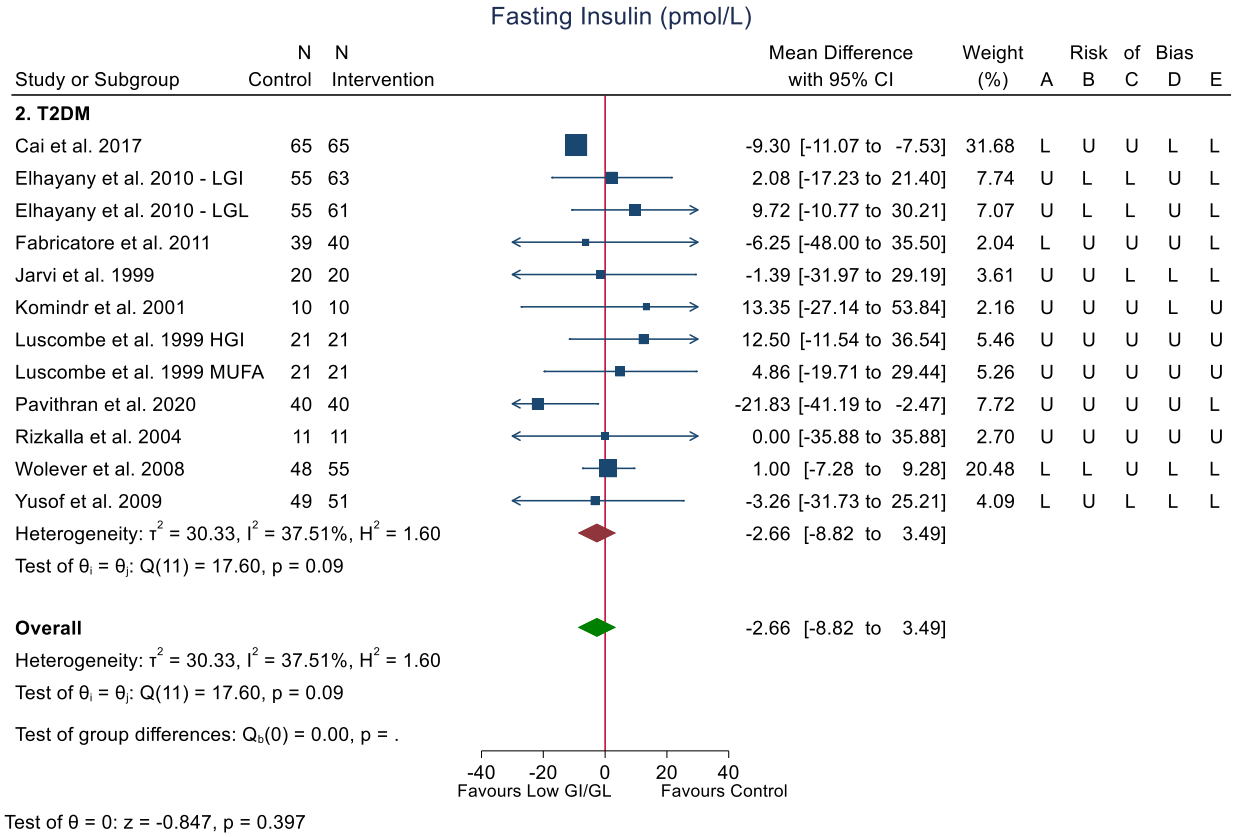


Test of θ = 0: z = , p = 0.000

Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fat; T1DM, type 1 diabetes; T2DM, type 2 diabetes

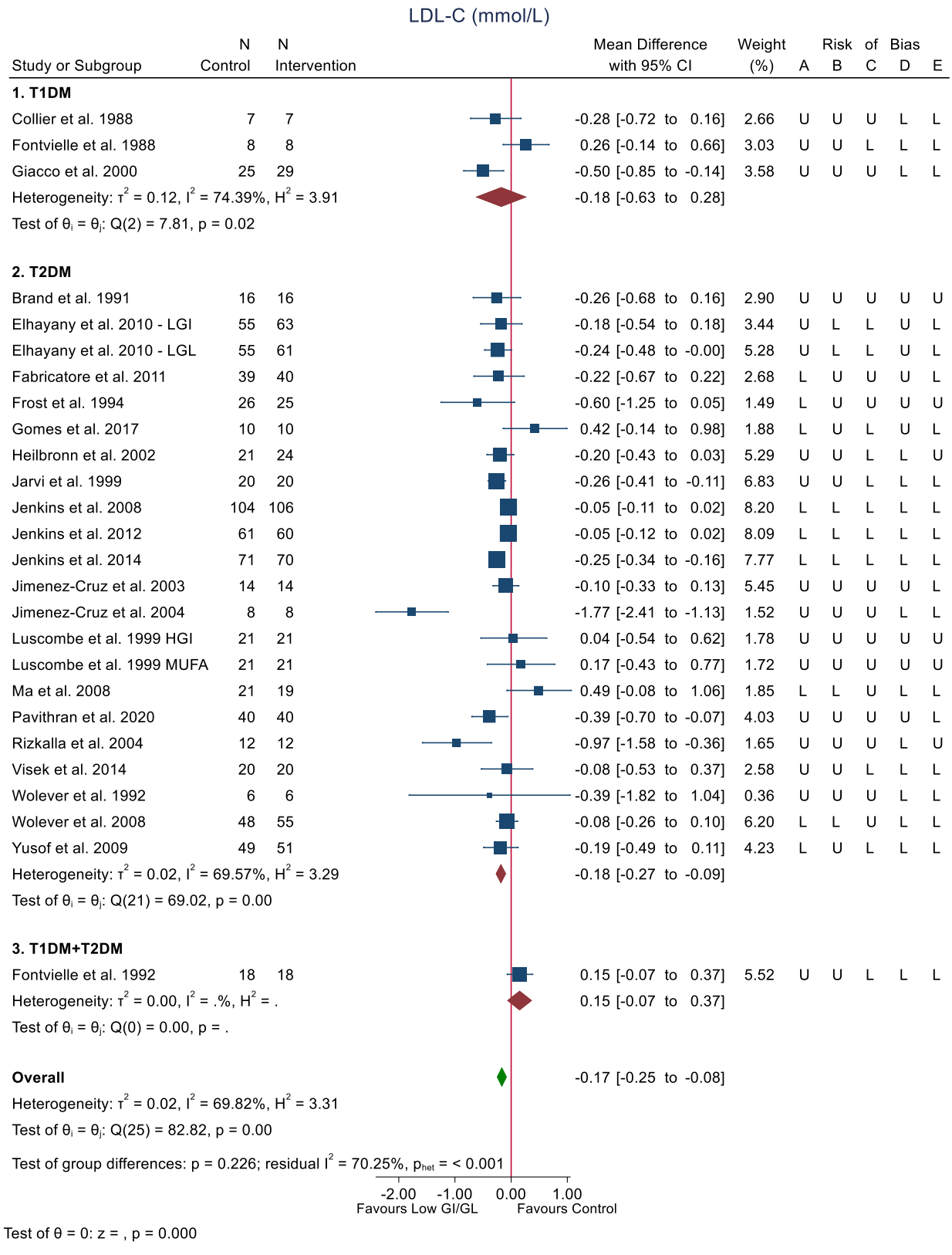
Supplemental Figure S5: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on fasting insulin (pmol/L) in diabetes



Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at P<0.10 and I^2>50% considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test. The conversion from mIU/L to pmol/L in Pavithran et al. 2020 produced implausible differences in the MD estimates and variances. We therefore treated the mIU/L as pmol/L. If we convert the mIU/L to pmol/L, then the direction, magnitude (<1 MID of 5pmol/L) and significance of the estimates and the evidence for heterogeneity do not change meaningfully (MD -0.70pmol/L [95% CI: -7.86 to 6.46], P=0.847; I^2=45%, P-het=0.04).

CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fat; T2DM, type 2 diabetes

Supplemental Figure S6: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on LDL-C (mmol/L) in diabetes



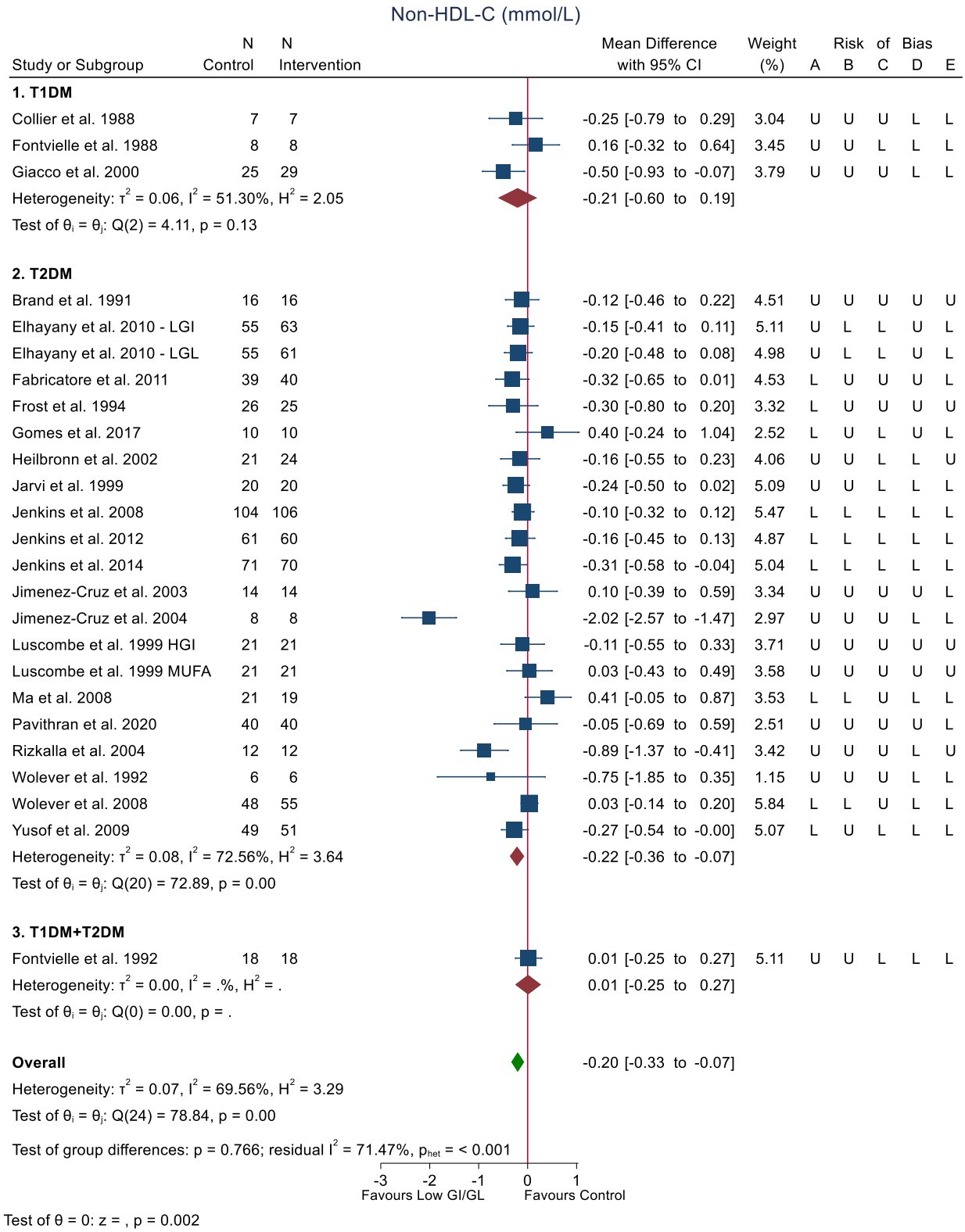
Test of θ = 0: z = , p = 0.000

Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

Note that in 5 studies, the Friedewald equation was used to calculate LDL-C (LDL-C = total cholesterol – HDL-C – triglycerides*0.45, where units are all in mmol/L) (Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem. 1972;18(6):499-5024337382) and the SDs were calculated using a standard formula using the SDs of total cholesterol, HDL-C and triglycerides (Cohen, J. (1988), Statistical Power Analysis for the Behavioral Sciences, 2nd Edition. Hillsdale: Lawrence Erlbaum. Hedges L. V., Olkin I. (1985). Statistical methods for meta-analysis. San Diego, CA: Academic Press https://www.statisticshowto.datasciencecentral.com/pooled-standard-deviation/).

CI, confidence interval; GI, glycemic index; GL, glycemic load; HDL-C, high-density lipoprotein-cholesterol; HGI, high GI; LGI, low-GI; LGL, low-GL; LDL-C, low-density lipoprotein-cholesterol; MUFA, monounsaturated fat; SD, standard deviation; T1DM, type 1 diabetes; T2DM, type 2 diabetes

Supplemental Figure S7: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on Non-HDL-C (mmol/L) in diabetes



Test of θ = 0: z = , p = 0.002

Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

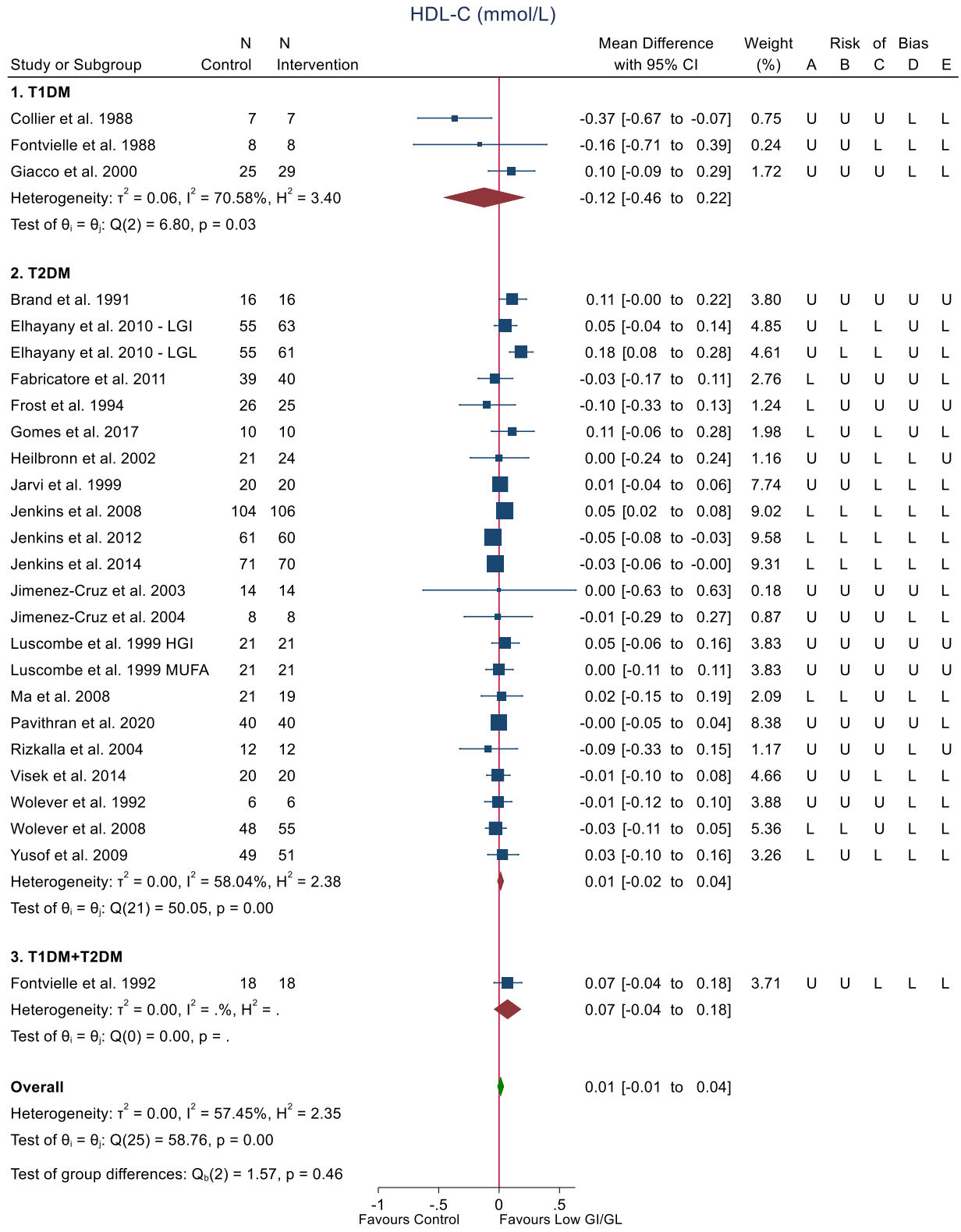
For all studies, non-HDL-C was not explicitly reported. However, non-HDL-C was determined using studies that reported both total cholesterol and HDL-C by calculating the difference between the means. The SDs for non-HDL-C were calculated using the inverse variance law using the SDs of total cholesterol

$$SD_{X\pm Y} = \frac{1}{\sqrt{k}}\sqrt{SD_1^2 + SD_2^2}$$

and HDL-C, (Harry Ku (1966). Notes on the Use of Propagation of Error Formulas, J Research of National Bureau of Standards-C. Engineering and Instrumentation, Vol. 70C, No.4, pp. 263-273.)

CI, confidence interval; GI, glycemic index; GL, glycemic load; HDL-C, high-density lipoprotein-cholesterol; HGI, high GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fat; Non-HDL-C, non-high-density lipoprotein-cholesterol; SD, standard deviation; T1DM, type 1 diabetes; T2DM, type 2 diabetes

Supplemental Figure S8: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on HDL-C (mmol/L) in diabetes

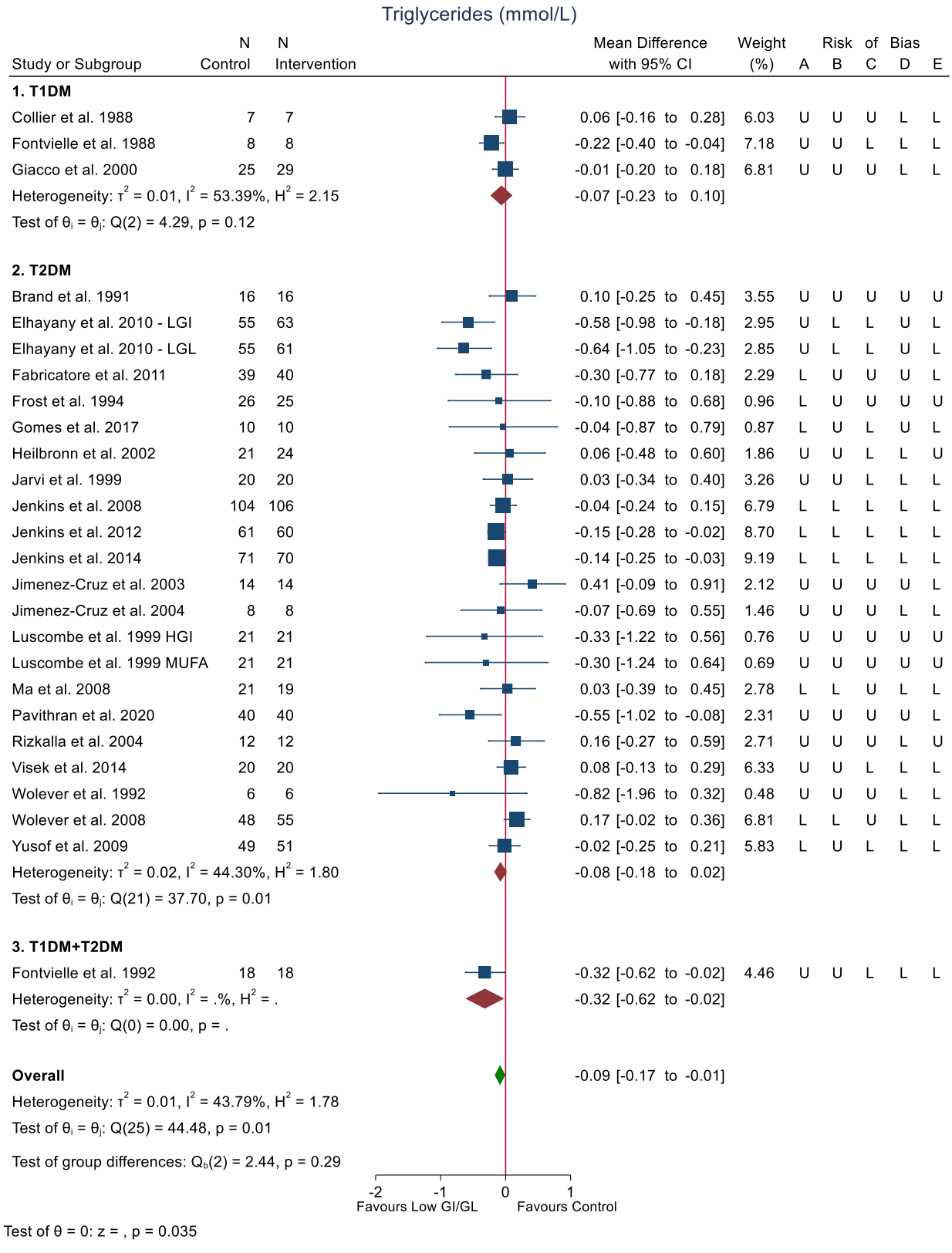


Test of θ = 0: z = , p = 0.351

Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

CI, confidence interval; GI, glycemic index; GL, glycemic load; HDL-C, high-density lipoprotein-cholesterol; HGI, high GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fat; T1DM, type 1 diabetes; T2DM, type 2 diabetes

Supplemental Figure S9: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on triglycerides (mmol/L) in diabetes

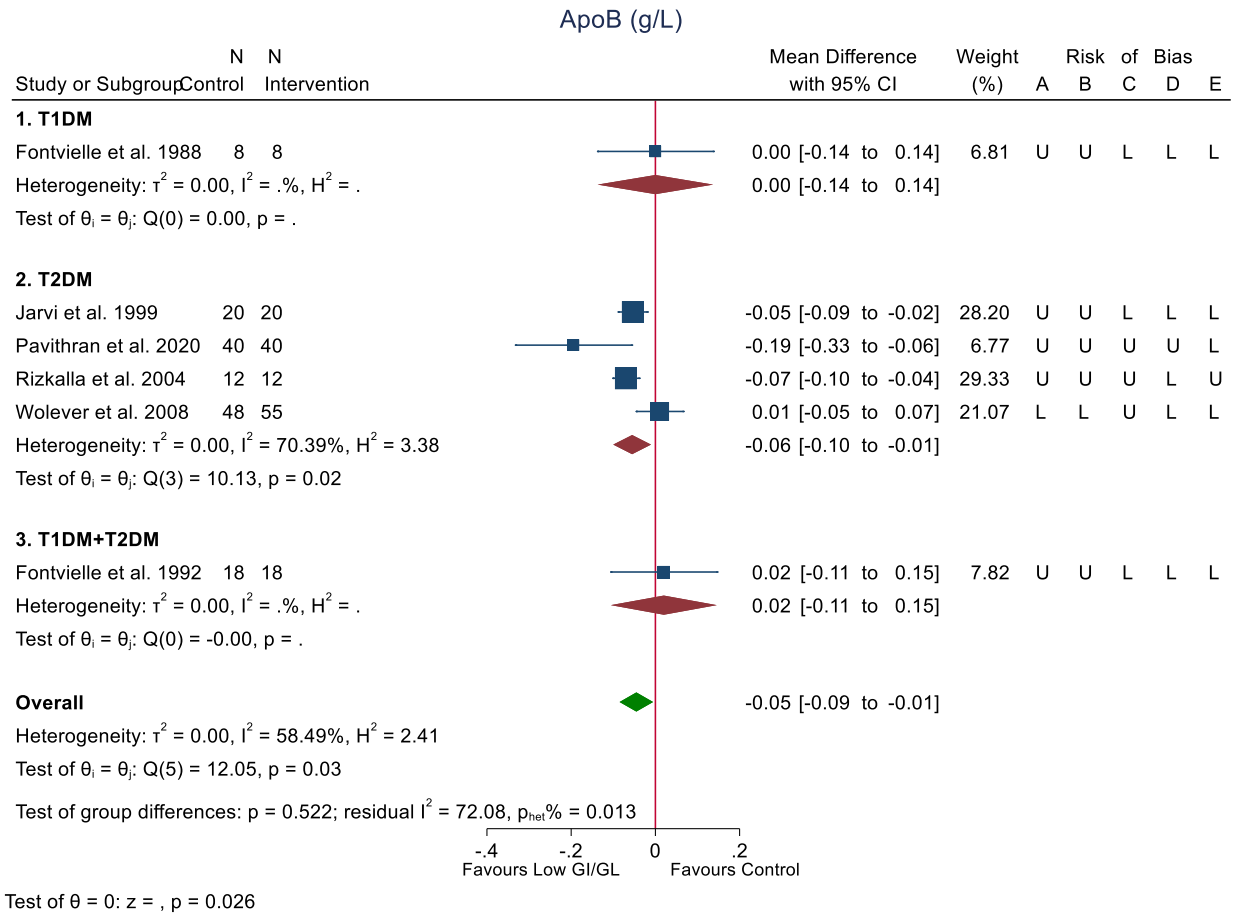


Test of θ = 0: z = , p = 0.035

Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fat; T1DM, type 1 diabetes; T2DM, type 2 diabetes

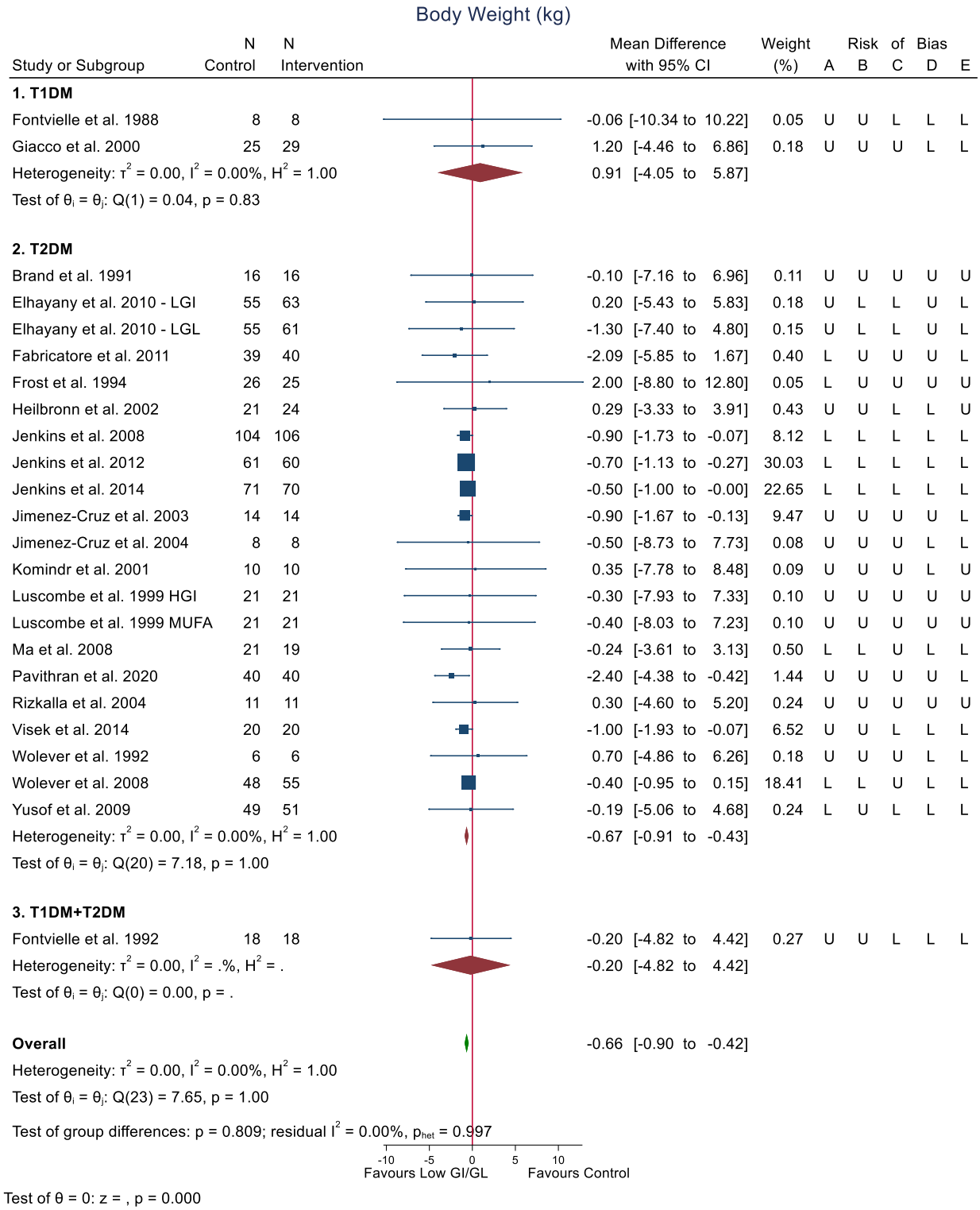
Supplemental Figure S10: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on apoB (g/L) in diabetes



Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at P<0.10 and I^2>50% considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

ApoB, apolipoprotein B; CI, confidence interval; GI, glycemic index; GL, glycemic load; T1DM, type 1 diabetes; T2DM, type 2 diabetes

Supplemental Figure S11: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on body weight (kg) in diabetes

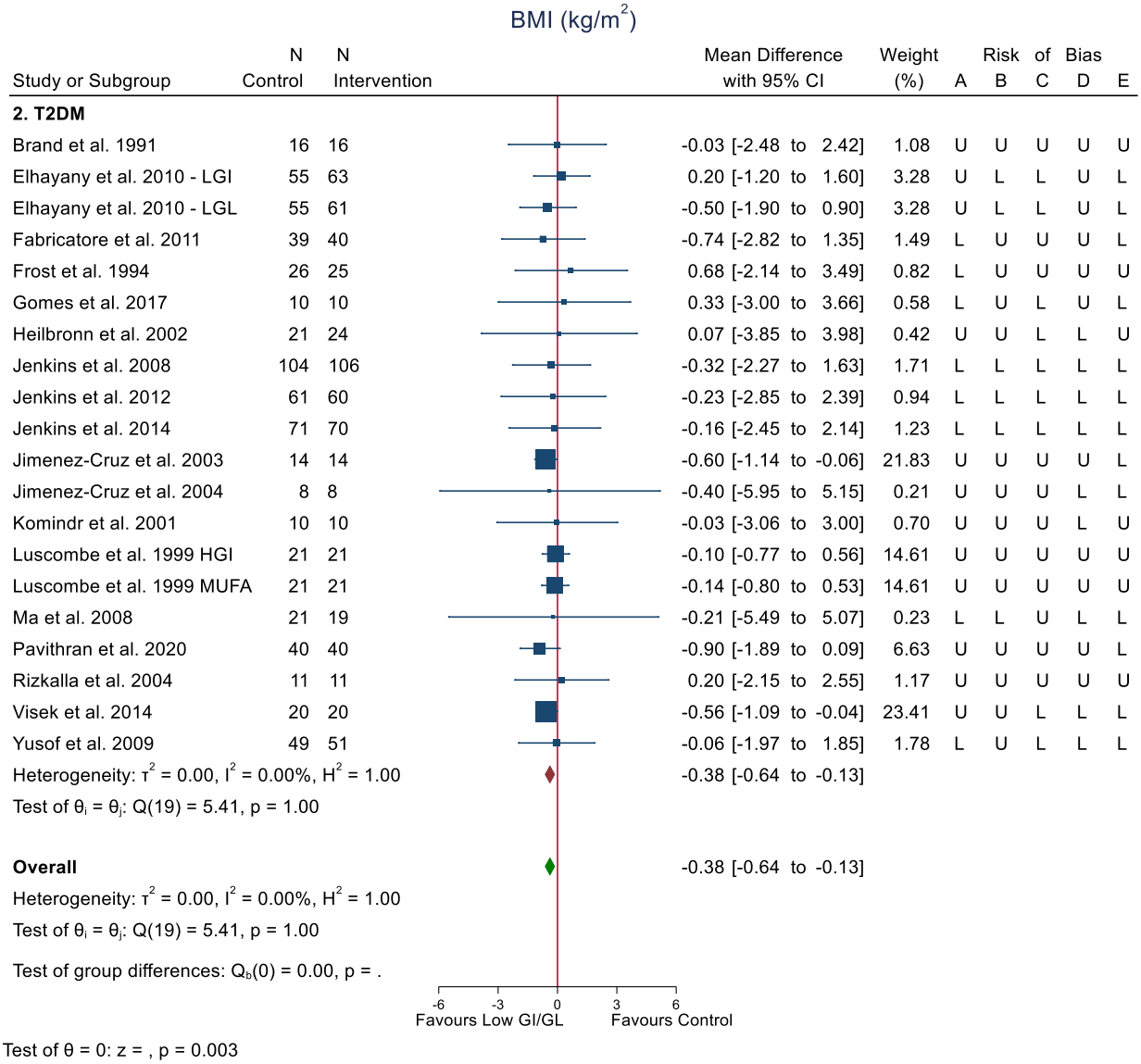


Test of θ = 0: z = , p = 0.000

Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fat; T1DM, type 1 diabetes; T2DM, type 2 diabetes

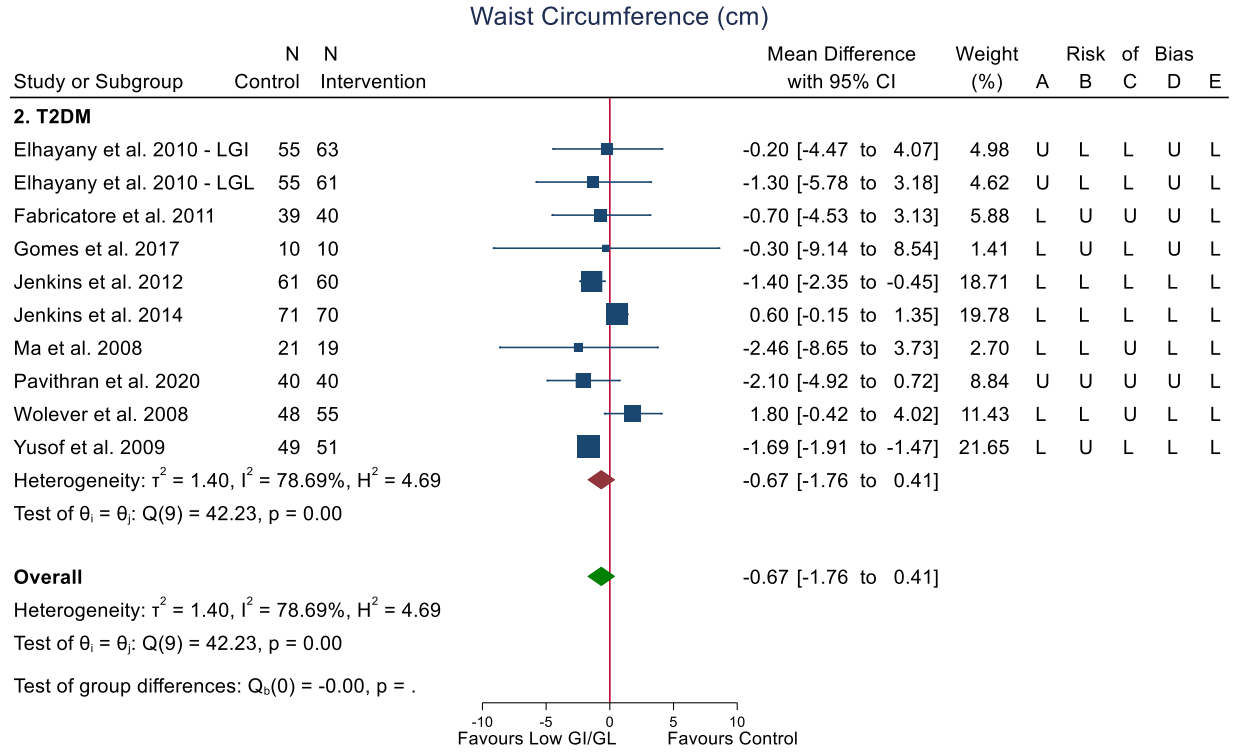
Supplemental Figure S12: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on BMI (kg/m²) in diabetes



Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

BMI, body mass index; CI, confidence interval; GI, glycemic index; GL, glycemic load; LGI, low-GI; LGL, low-GL; T1DM, type 1 diabetes; T2DM, type 2 diabetes

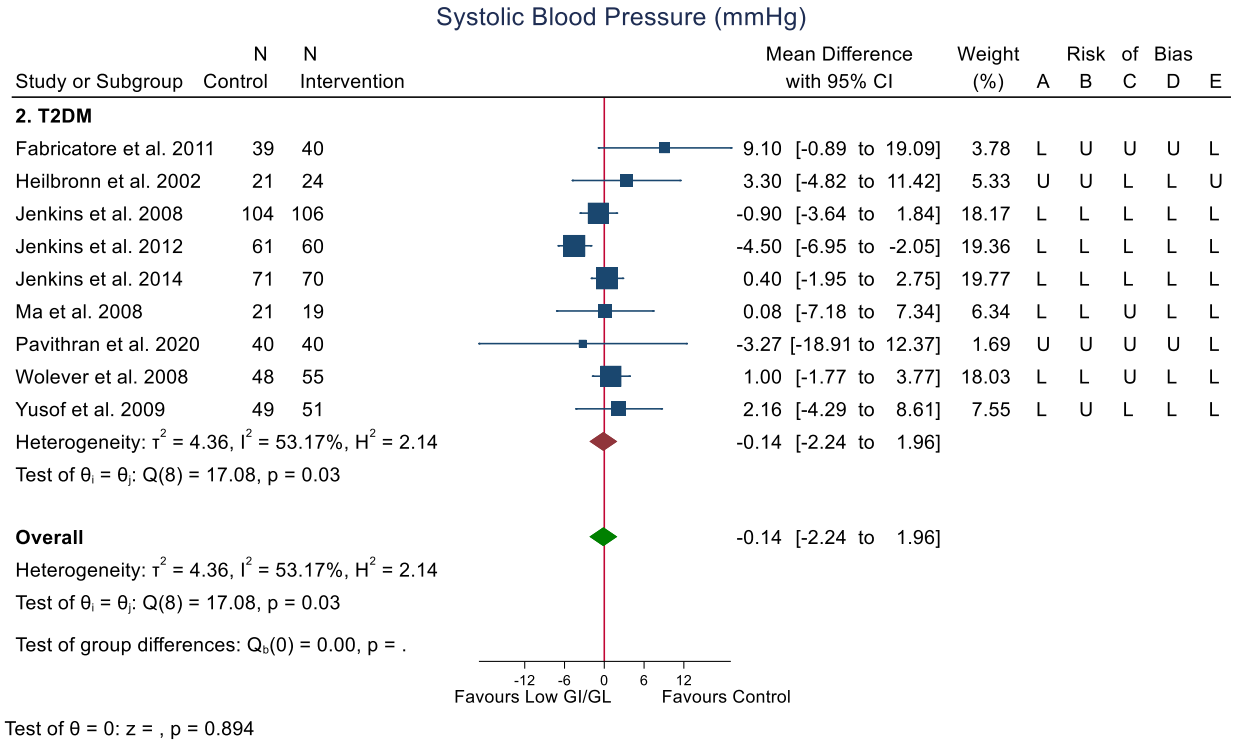
Supplemental Figure S13: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on waist circumference (cm) in diabetes



Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

CI, confidence interval; GI, glycemic index; GL, glycemic load; LGI, low-GI; LGL, low-GL; T2DM, type 2 diabetes

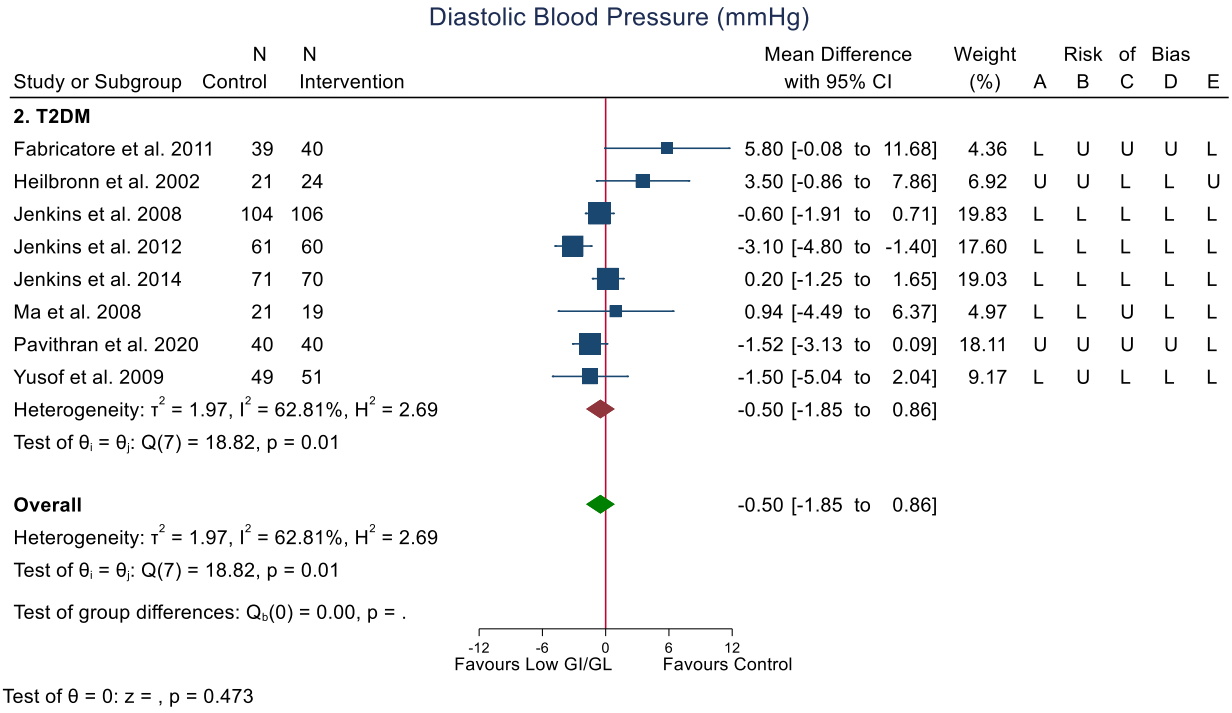
Supplemental Figure S14: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on systolic blood pressure (mmHg) in diabetes



Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

CI, confidence interval; GI, glycemic index; GL, glycemic load; T2DM, type 2 diabetes

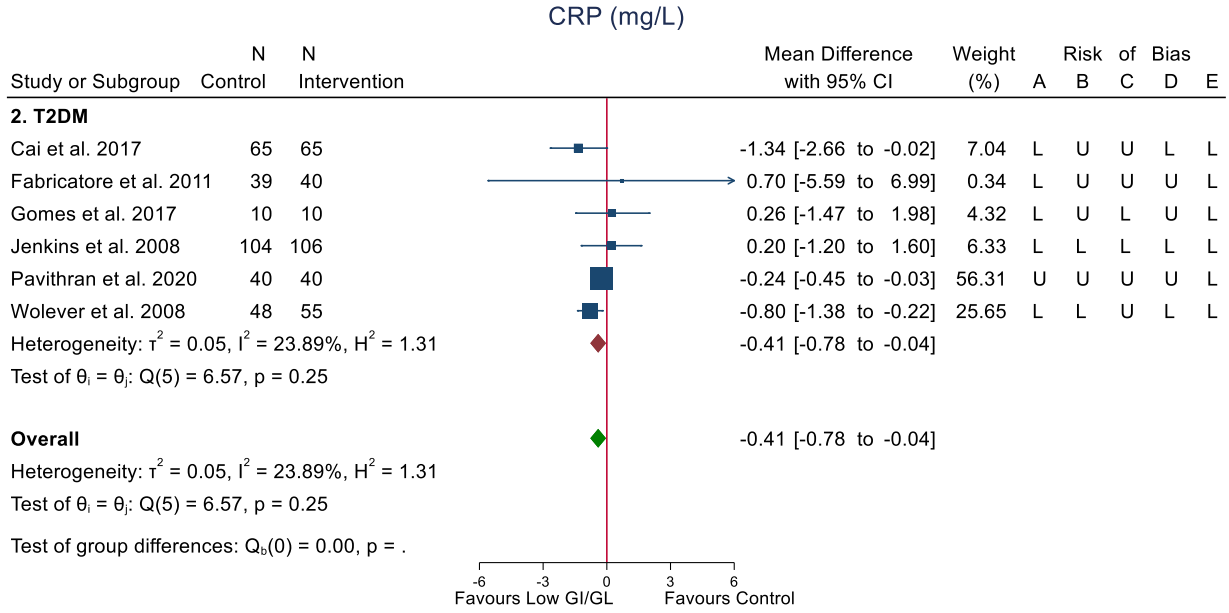
Supplemental Figure S15: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on diastolic blood pressure (mmHg) in diabetes



Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

CI, confidence interval; GI, glycemic index; GL, glycemic load; T2DM, type 2 diabetes

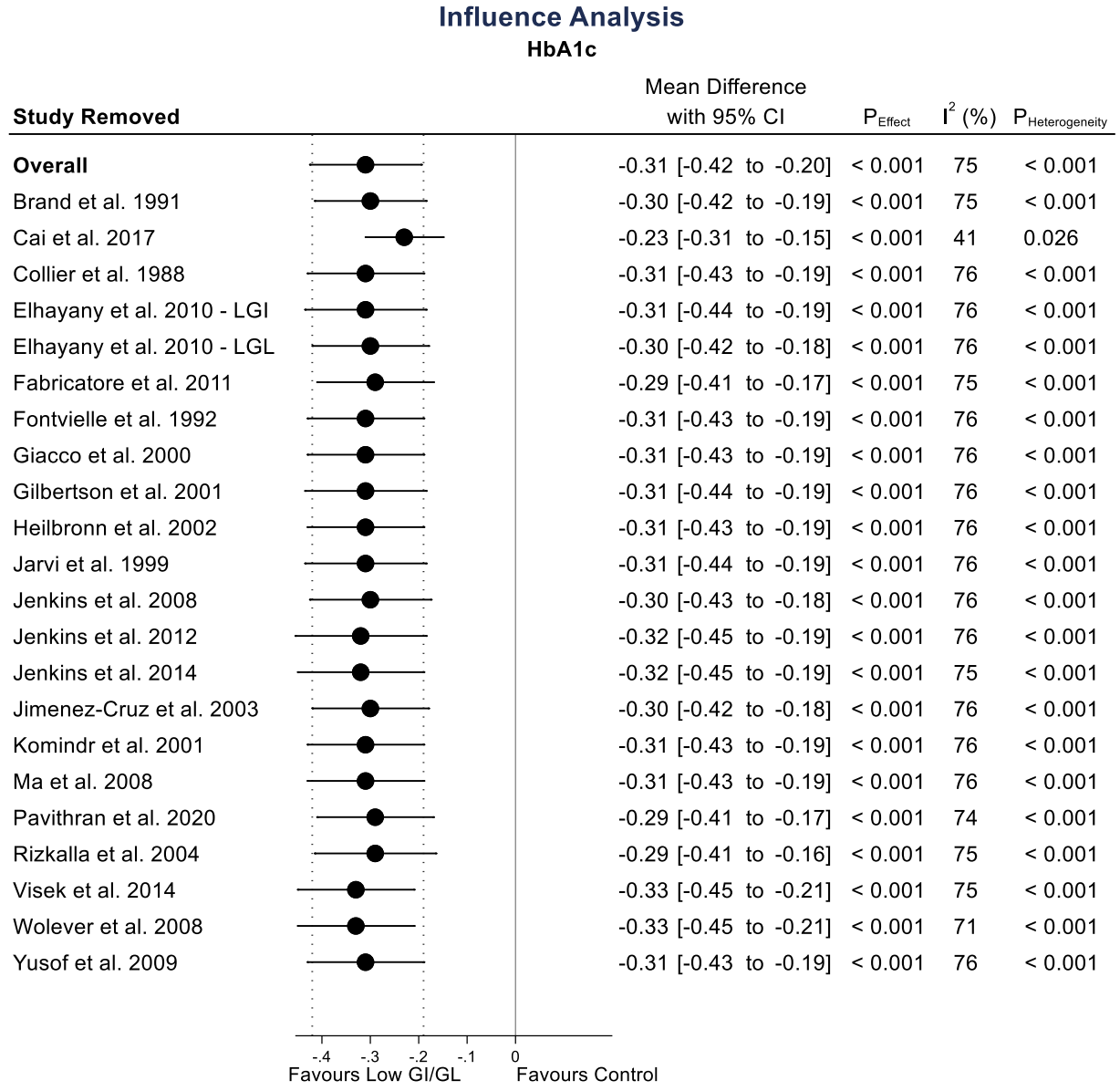
Supplemental Figure S16: Forest plot of randomized controlled trials of the effect of low-GI/GL dietary patterns on CRP (mg/L) in diabetes



Pooled effect estimates for each subgroup and overall effect are represented by the diamonds. Data are expressed as weighted mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Paired analyses were applied to all crossover trials. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. Risk of Bias Legend: (H) High Risk; (L) Low Risk; (U) Unclear. The letters represent the following risk of bias domains: A, random sequence generation (selection bias); B, allocation concealment (selection bias); C, blinding of participants and personnel and outcome assessors (performance bias); D, incomplete outcome data (attrition bias); and E, selective reporting (reporting bias). Pooled effect summary calculated with the χ^2 test. Test for group differences calculated with meta-regression, which uses the Wald test.

CI, confidence interval; CRP, c-reactive protein; GI, glycemic index; GL, glycemic load; T2DM, type 2 diabetes

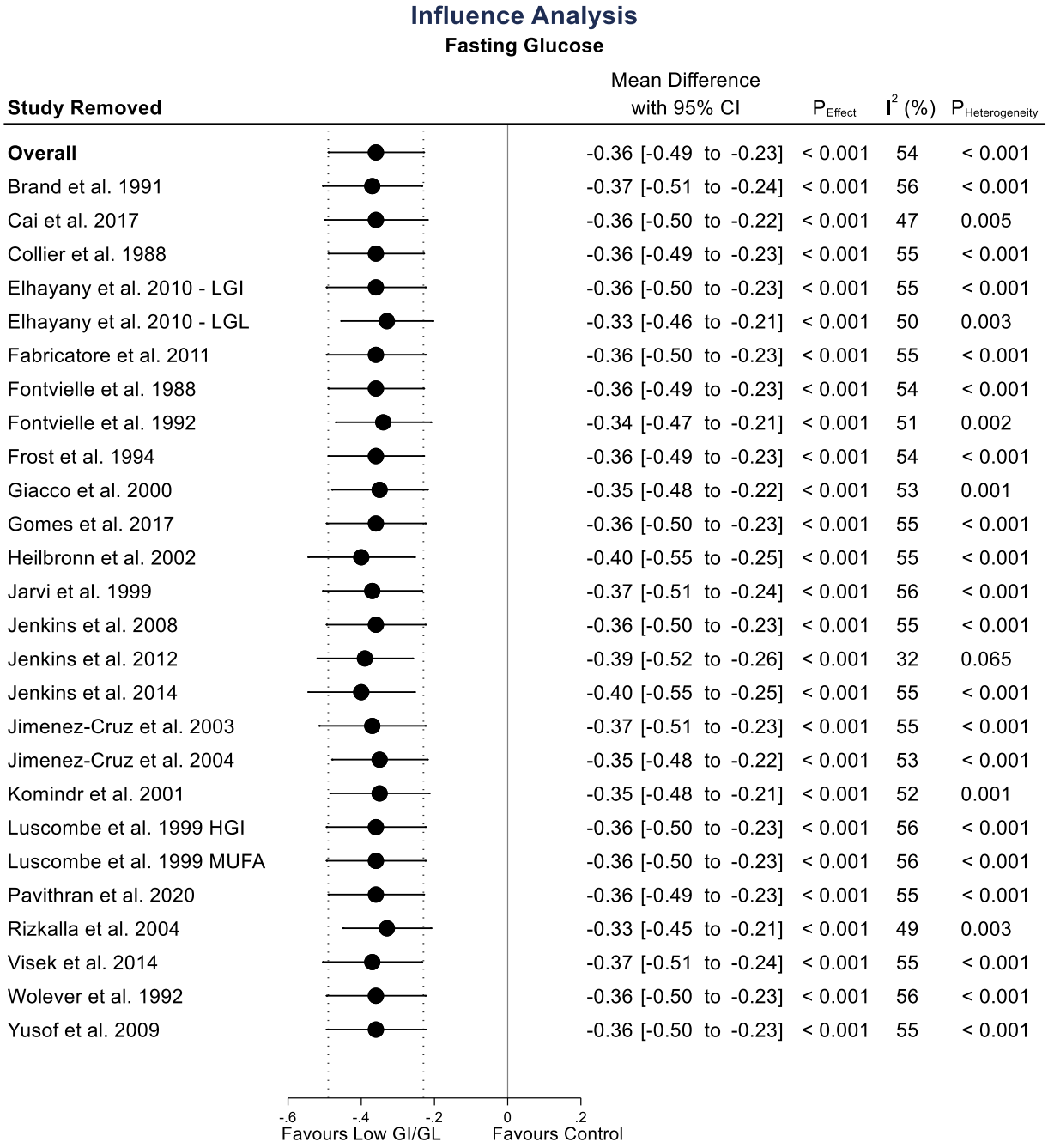
Supplemental Figure S17: Sensitivity analysis of the systematic removal of each trial comparison for HbA1c (%)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; GI, glycemic index; GL, glycemic load; HbA1c, hemoglobin A1c; HGI, high-GI; LGI, low-GI; LGL, low-GL

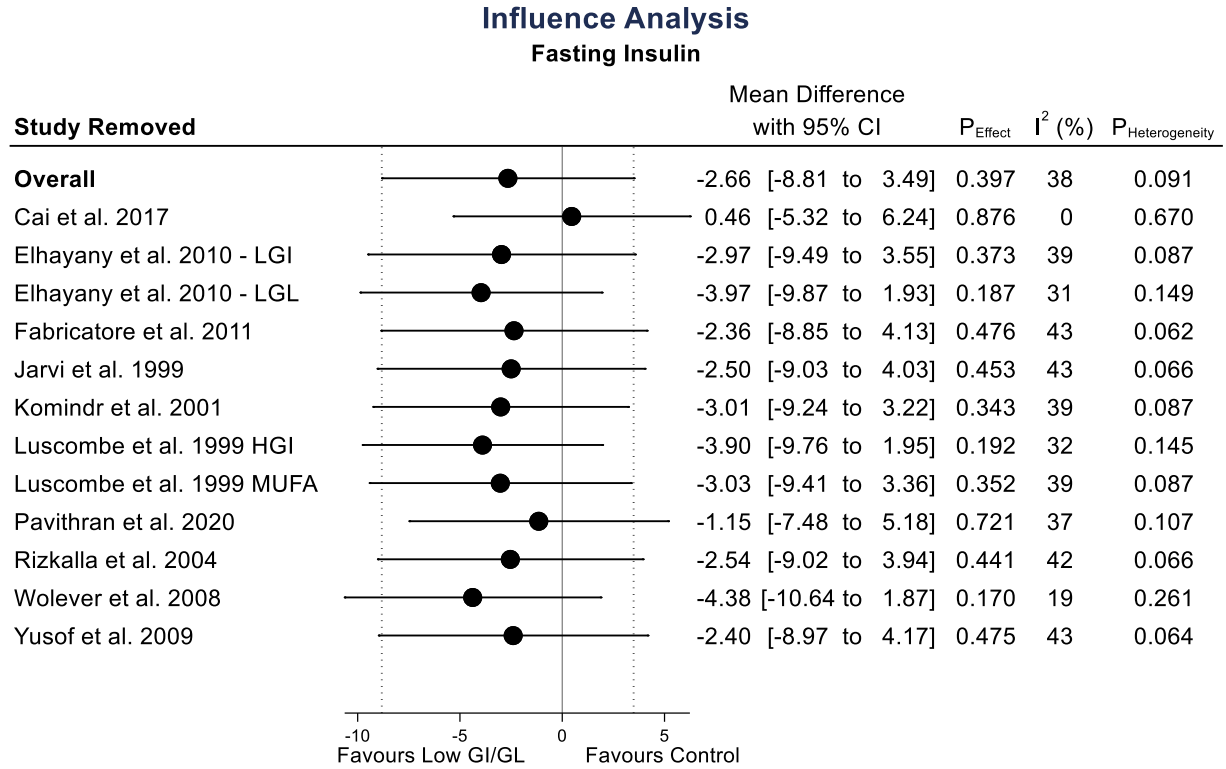
Supplemental Figure S18: Sensitivity analysis of the systematic removal of each trial comparison for fasting glucose (mmol/L)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high-GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fatty acids

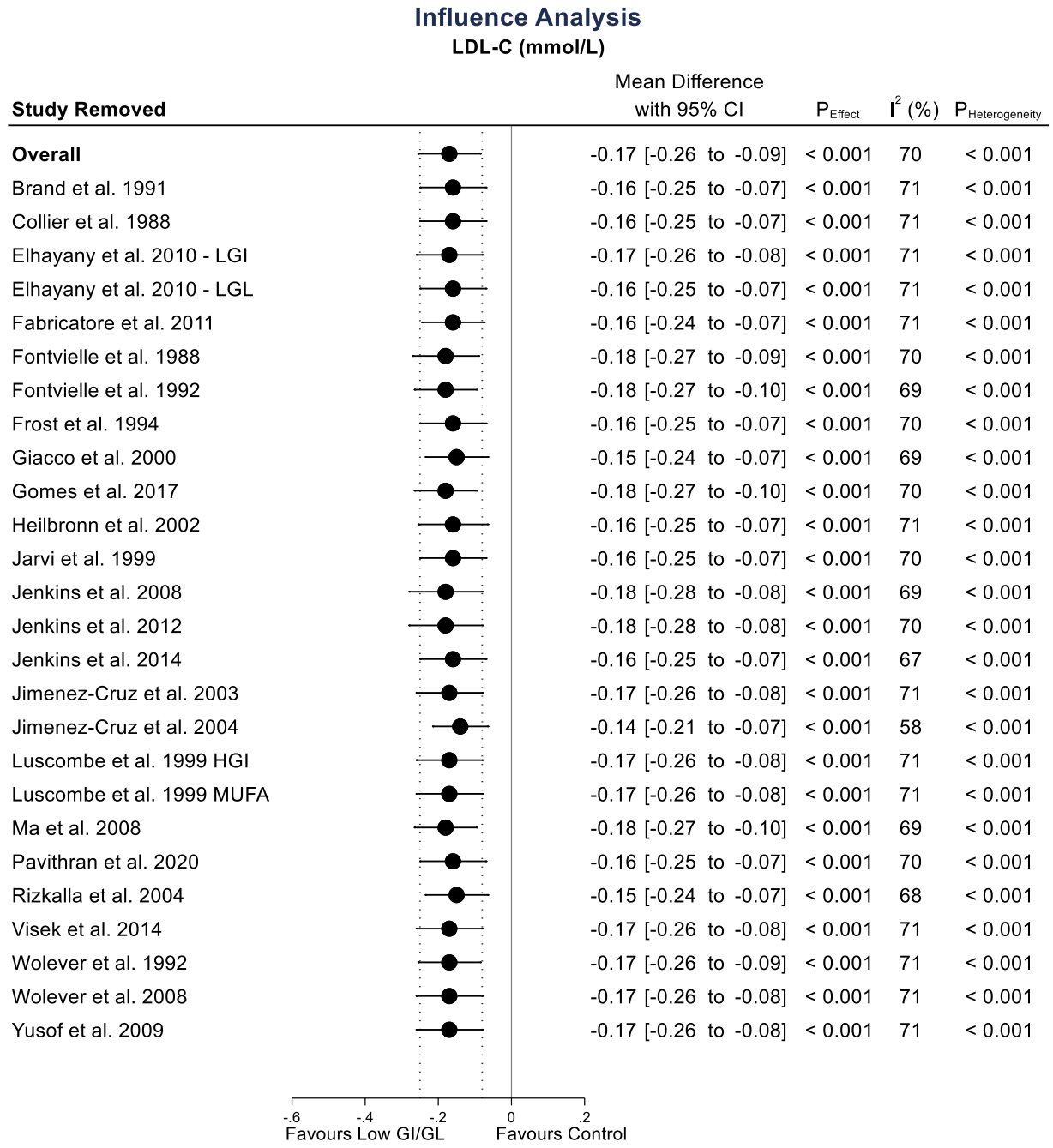
Supplemental Figure S19: Sensitivity analysis of the systematic removal of each trial comparison for fasting insulin (pmol/L)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high-GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fatty acids

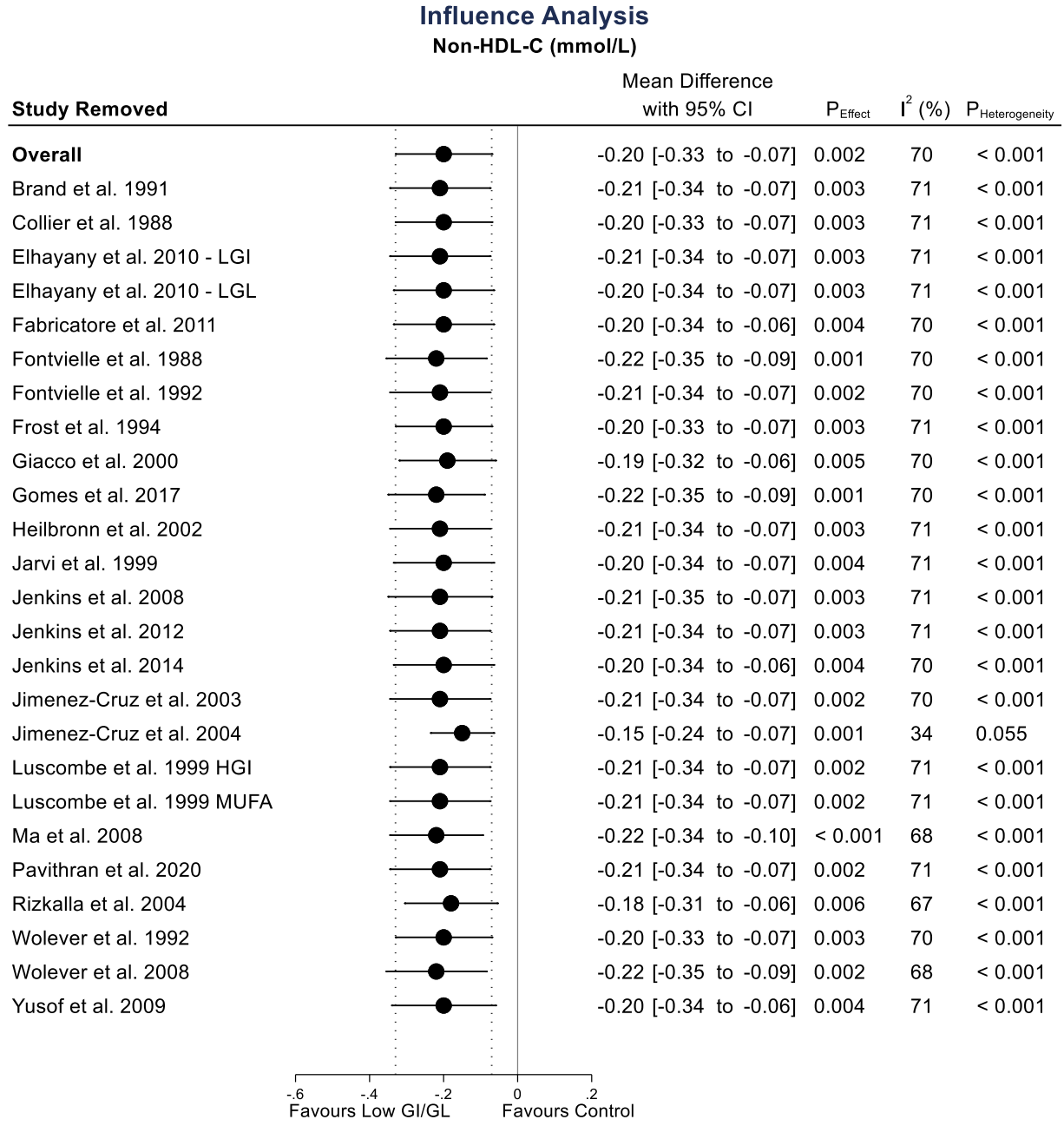
Supplemental Figure S20: Sensitivity analysis of the systematic removal of each trial comparison for LDL-C (mmol/L)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high-GI; LGI, low-GI; LGL, low-GL; LDL-C, low-density lipoprotein-cholesterol; MUFA, monounsaturated fatty acids

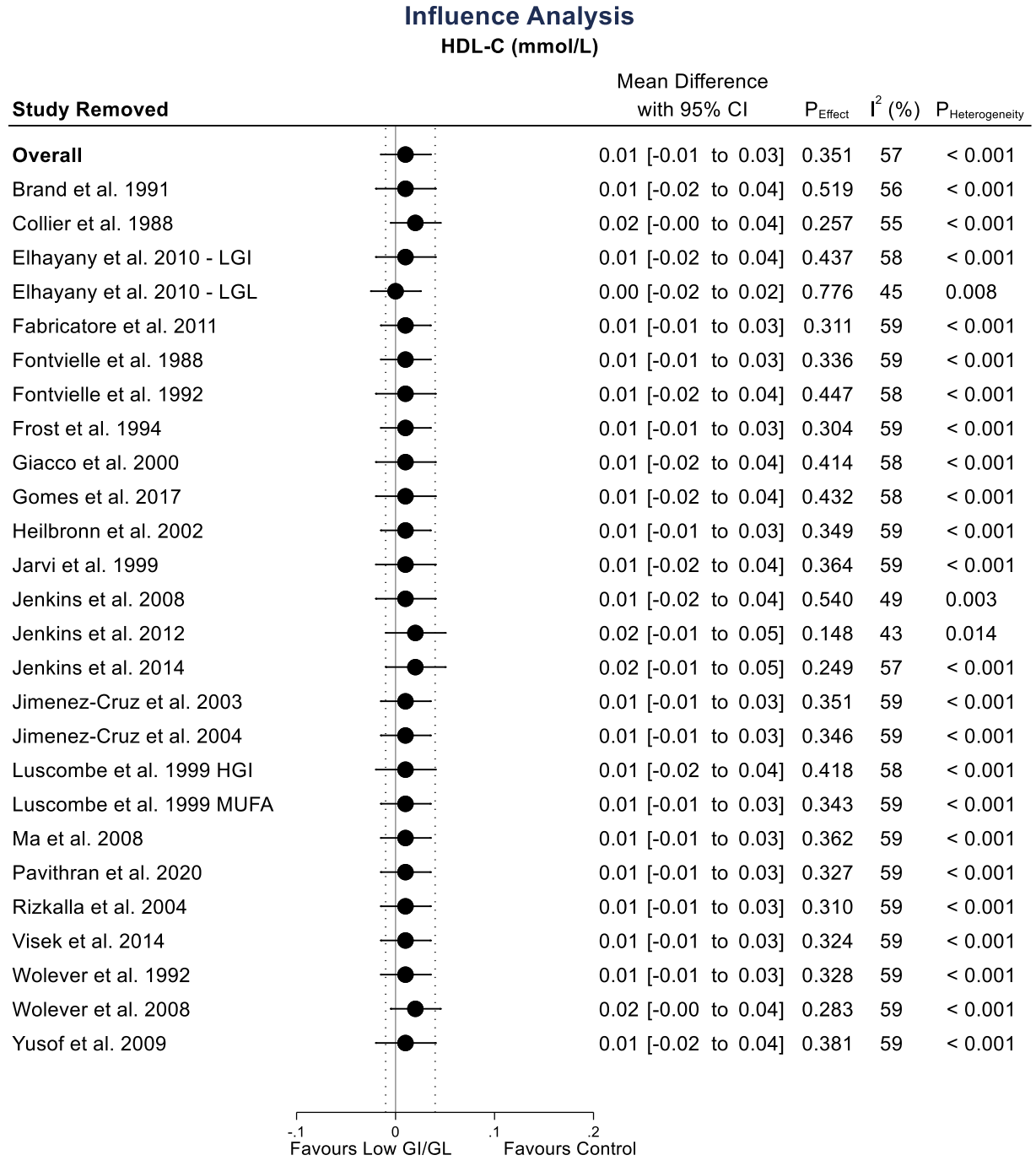
Supplemental Figure S21: Sensitivity analysis of the systematic removal of each trial comparison for Non-HDL-C (mmol/L)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high-GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fatty acids; Non-HDL-C, non-high-density lipoprotein-cholesterol

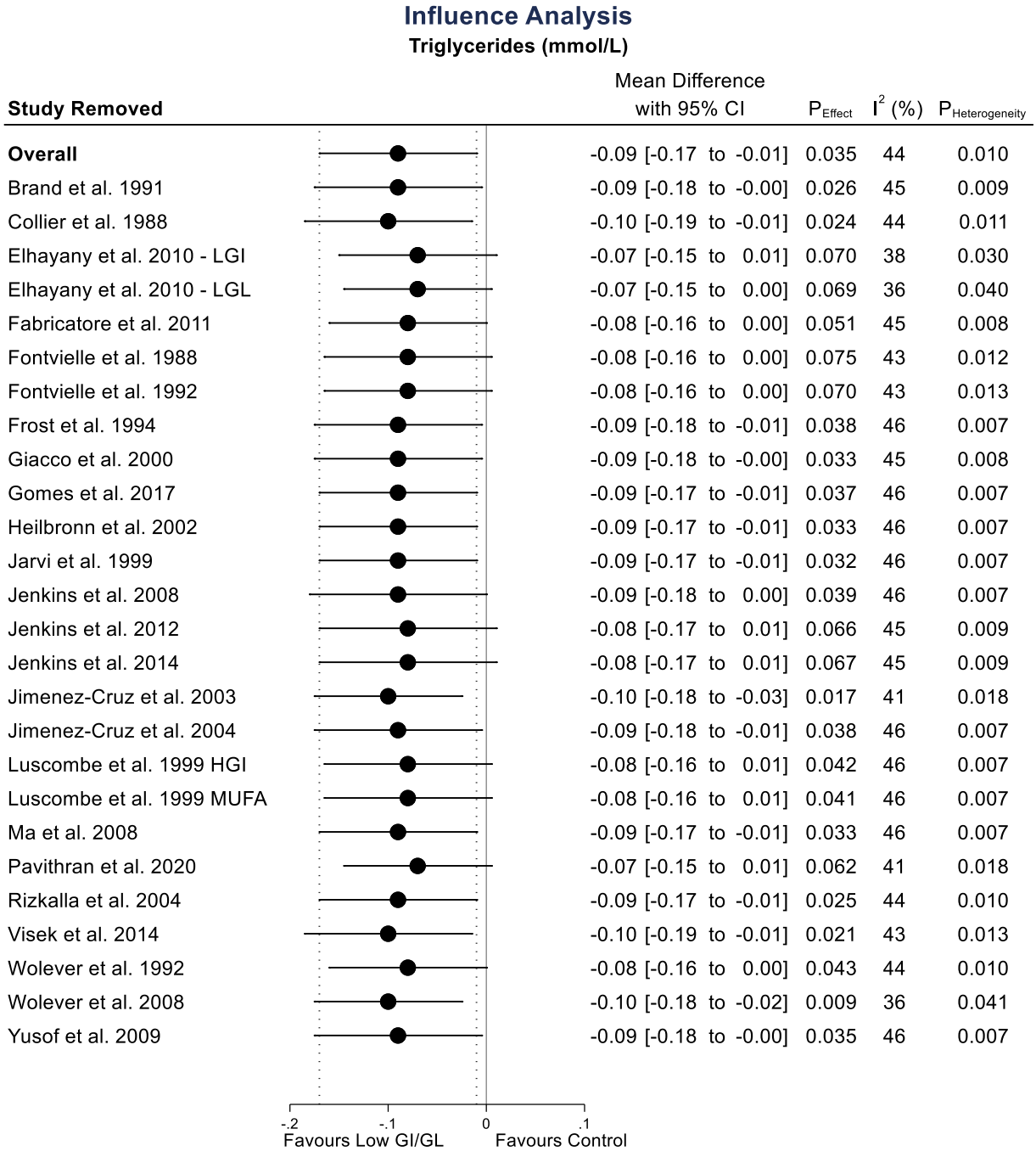
Supplemental Figure S22: Sensitivity analysis of the systematic removal of each trial comparison for HDL-C (mmol/L)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

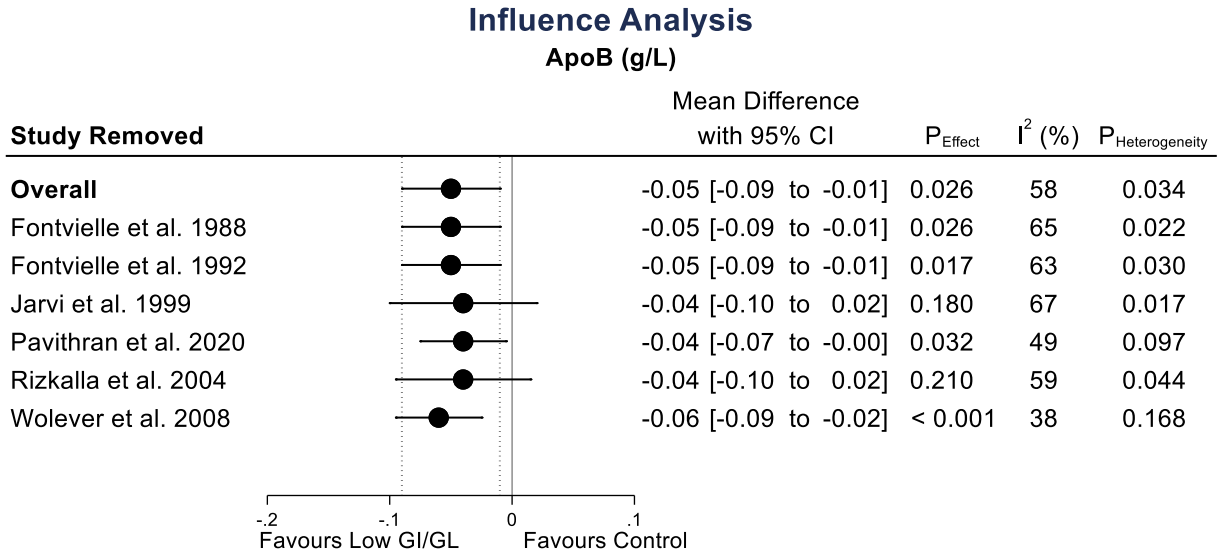
CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high-GI; HDL-C, high-density lipoprotein-cholesterol; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fatty acids

Supplemental Figure S23: Sensitivity analysis of the systematic removal of each trial comparison for triglycerides (mmol/L)



CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high-GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fatty acids

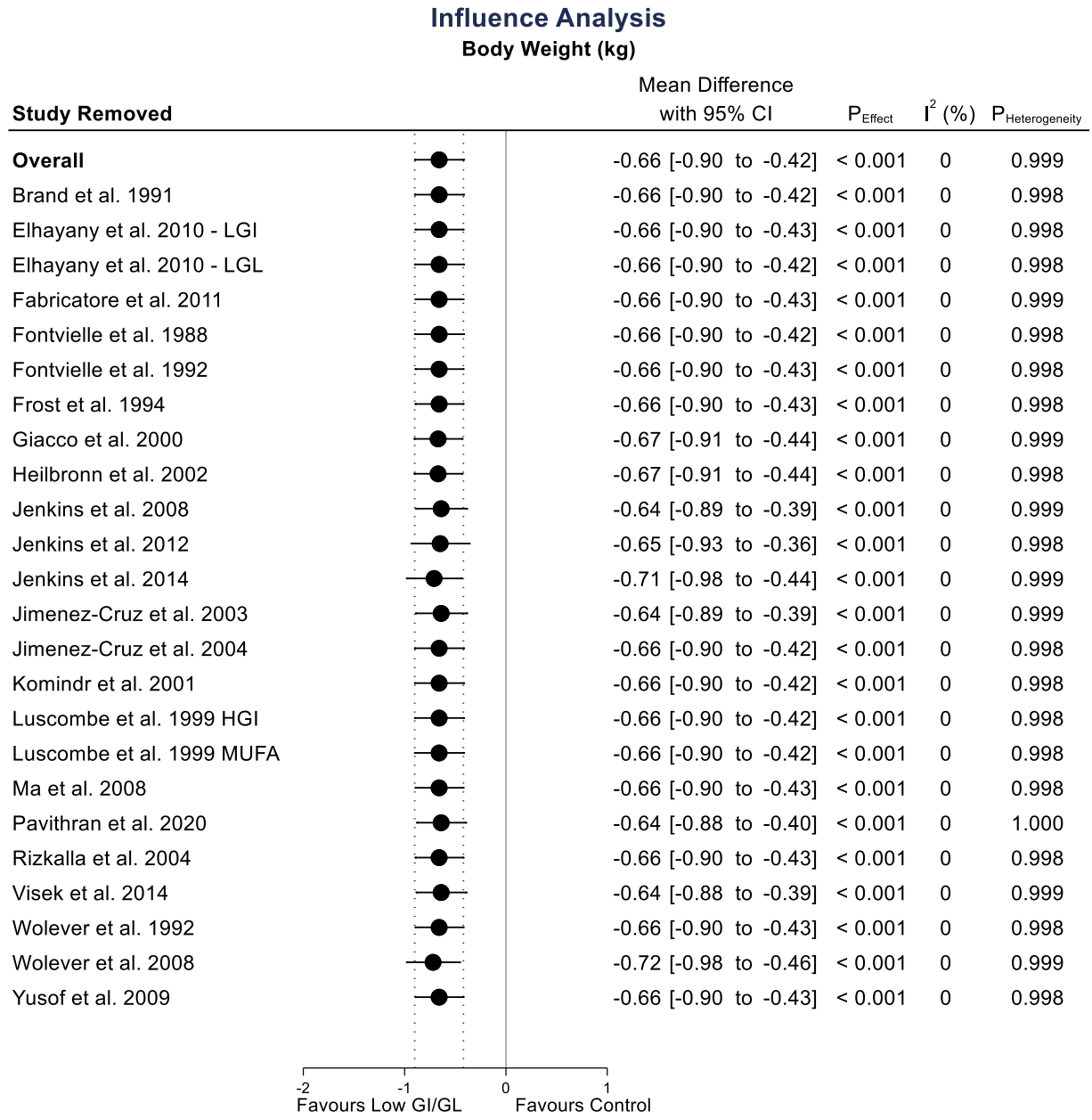
Supplemental Figure S24: Sensitivity analysis of the systematic removal of each trial comparison for ApoB (g/L)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

ApoB; apolipoprotein B; CI, confidence interval; GI, glycemic index; GL, glycemic load

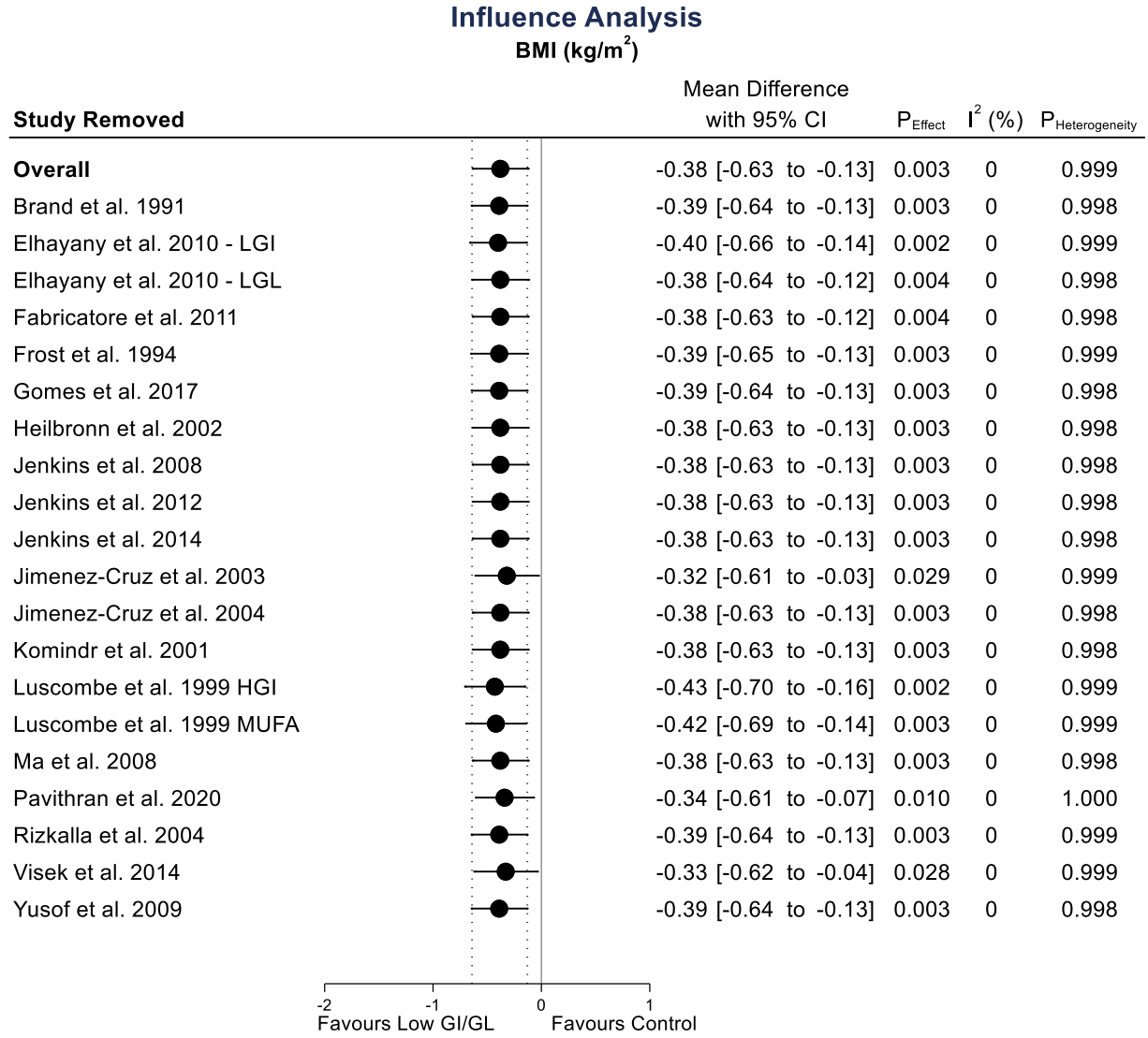
Supplemental Figure S25: Sensitivity analysis of the systematic removal of each trial comparison for body weight (kg)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; GI, glycemic index; GL, glycemic load; HGI, high-GI; LGI, low-GI; LGL, low-GL; MUFA, monounsaturated fatty acids

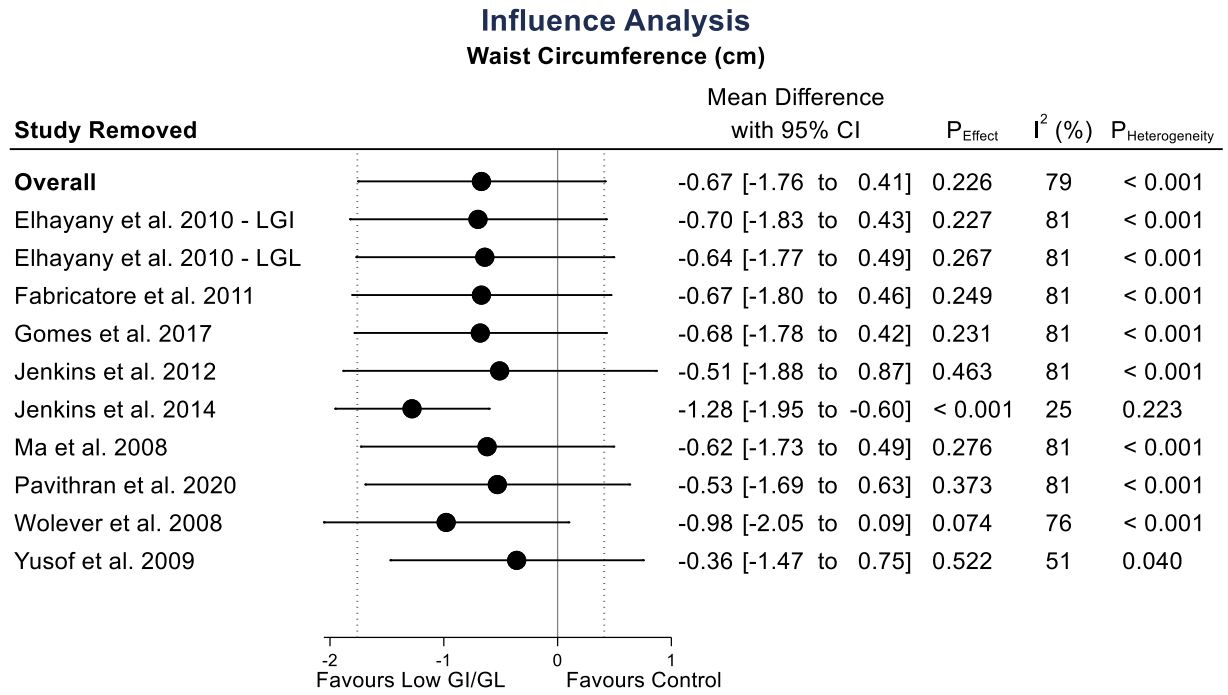
Supplemental Figure S26: Sensitivity analysis of the systematic removal of each trial comparison for BMI (kg/m²)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

BMI, body mass index; CI, confidence interval; GI, glycemic index; GL, glycemic load; LGI, low-GI; LGL, low-GL

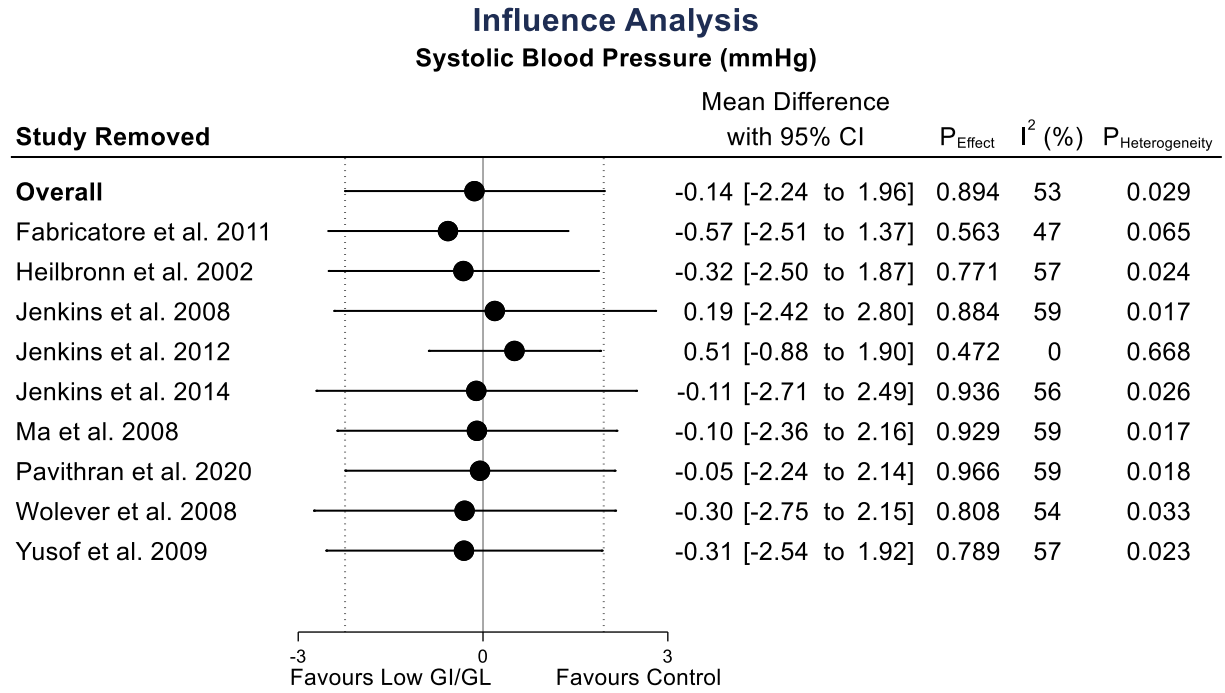
Supplemental Figure S27: Sensitivity analysis of the systematic removal of each trial comparison for waist circumference (cm)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; GI, glycemic index; GL, glycemic load; LGI, low-GI; LGL, low-GL

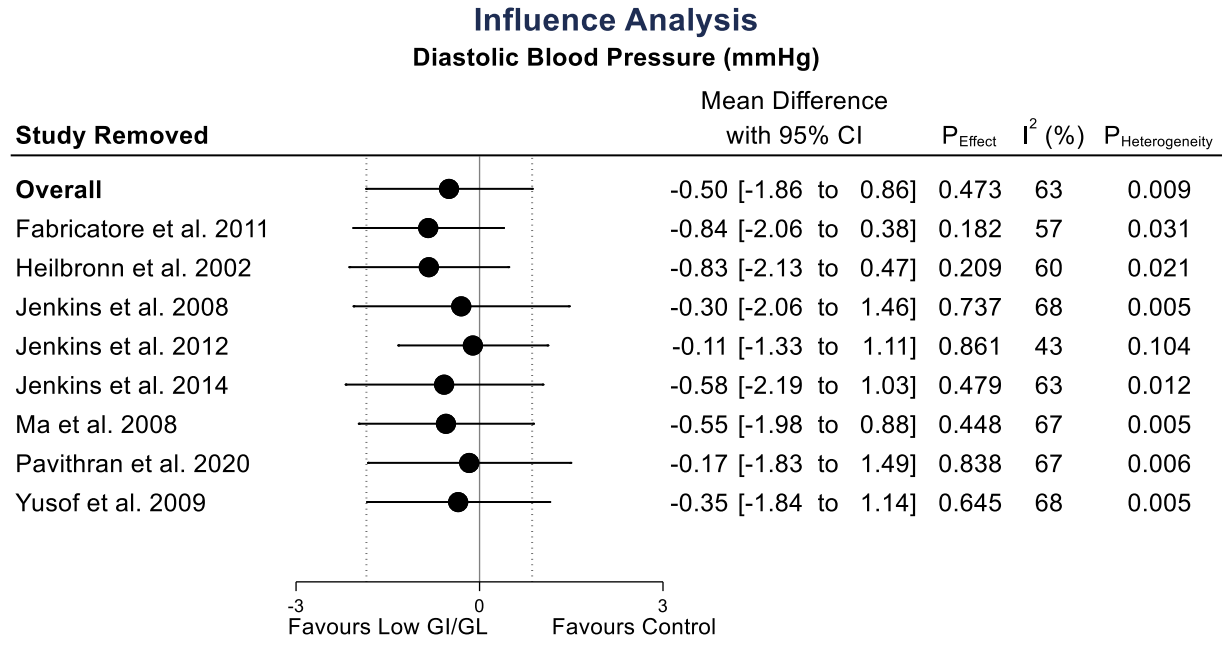
Supplemental Figure S28: Sensitivity analysis of the systematic removal of each trial comparison for systolic blood pressure (mmHg)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; GI, glycemic index; GL, glycemic load

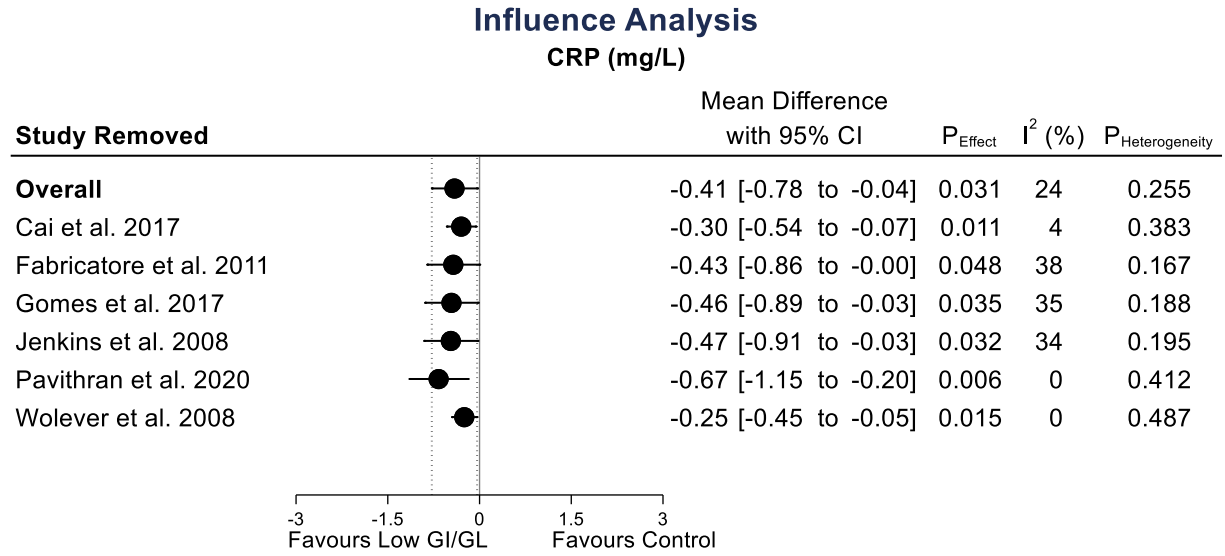
Supplemental Figure S29: Sensitivity analysis of the systematic removal of each trial comparison for diastolic blood pressure (mmHg)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; GI, glycemic index; GL, glycemic load

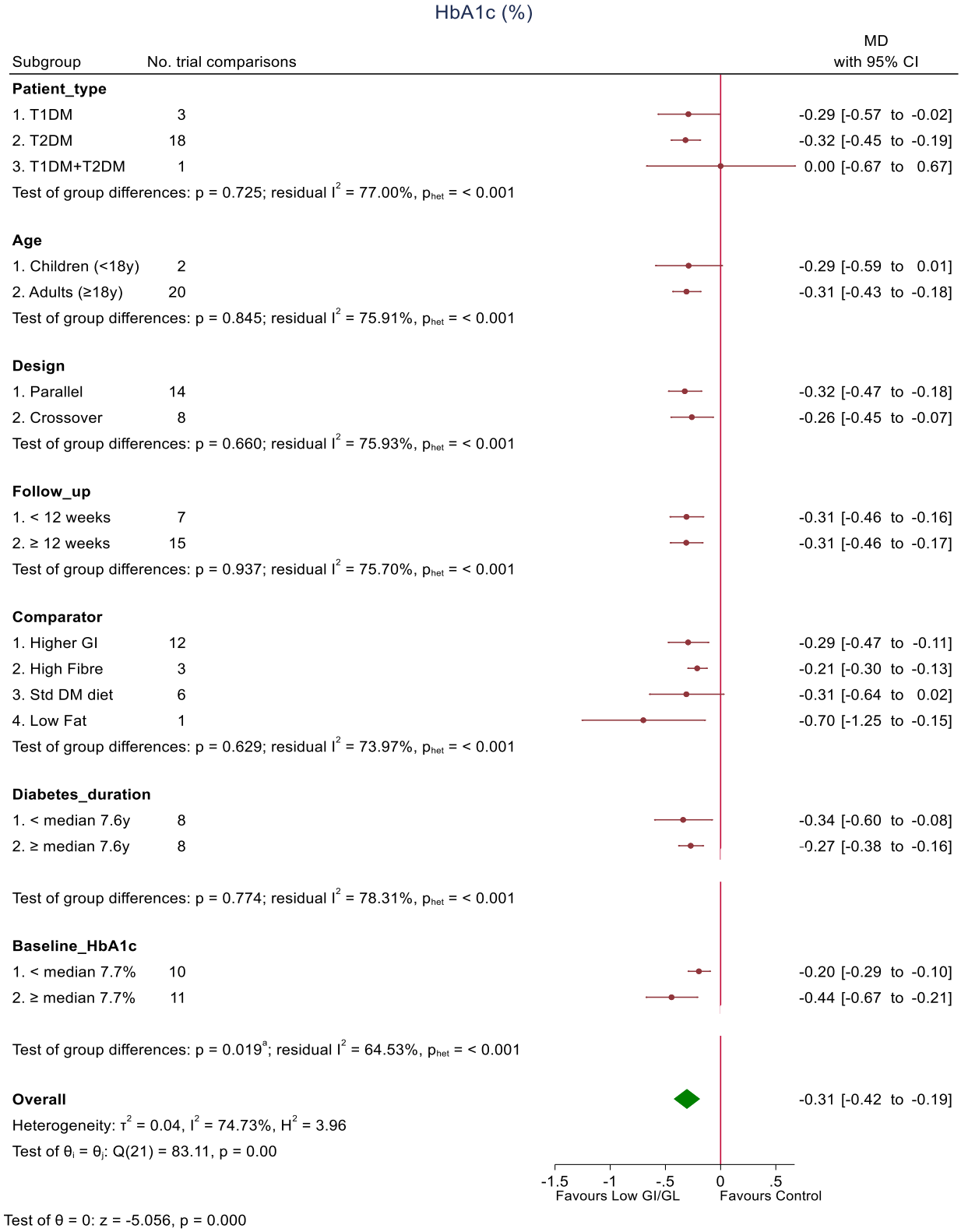
Supplemental Figure S30: Sensitivity analysis of the systematic removal of each trial comparison for CRP (mg/L)



Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

CI, confidence interval; CRP, c-reactive peptide; GI, glycemic index; GL, glycemic load

Supplemental Figure S31 (1 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on HbA1c (%) in diabetes*



Test of θ = 0: z = -5.056, p = 0.000

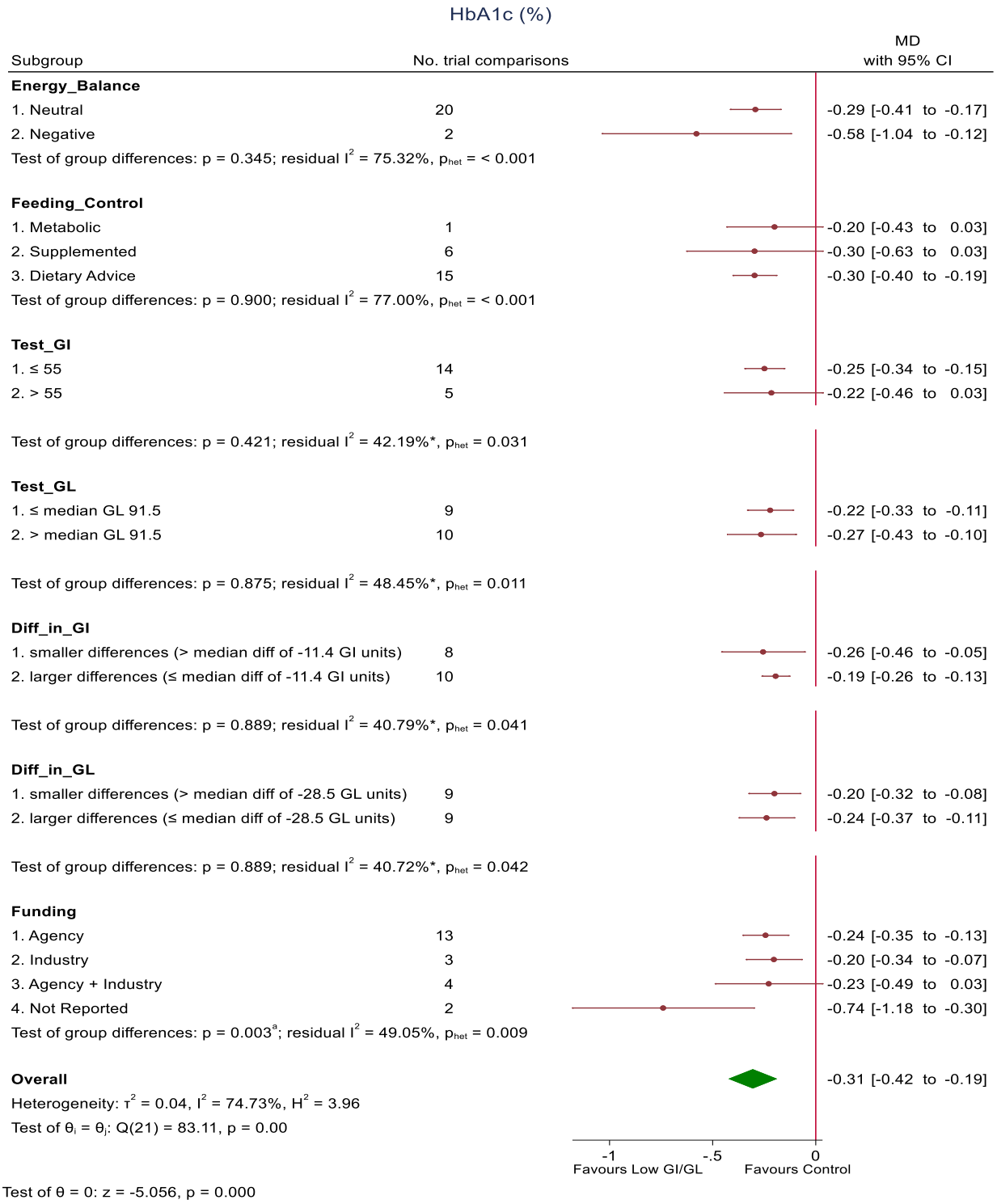
The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on HbA1c. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=6 trial comparisons missing data for disease duration, N=1 missing data for baseline HbA1c

[a]Pairwise between-subgroup mean differences (95% CIs) for Baseline HbA1c were as follows: -0.25% (-0.46 to -0.04%) (1 vs. 2).

CI, confidence interval; GI, glycemic index; GL, glycemic load; HbA1c, hemoglobin A1c; MD, mean difference; Std DM diet, standard diabetes diet; T1DM, type 1 diabetes; T2DM, type 2 diabetes; y, years

Supplemental Figure S31 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on HbA1c (%) in diabetes*



The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on HbA1c. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95%

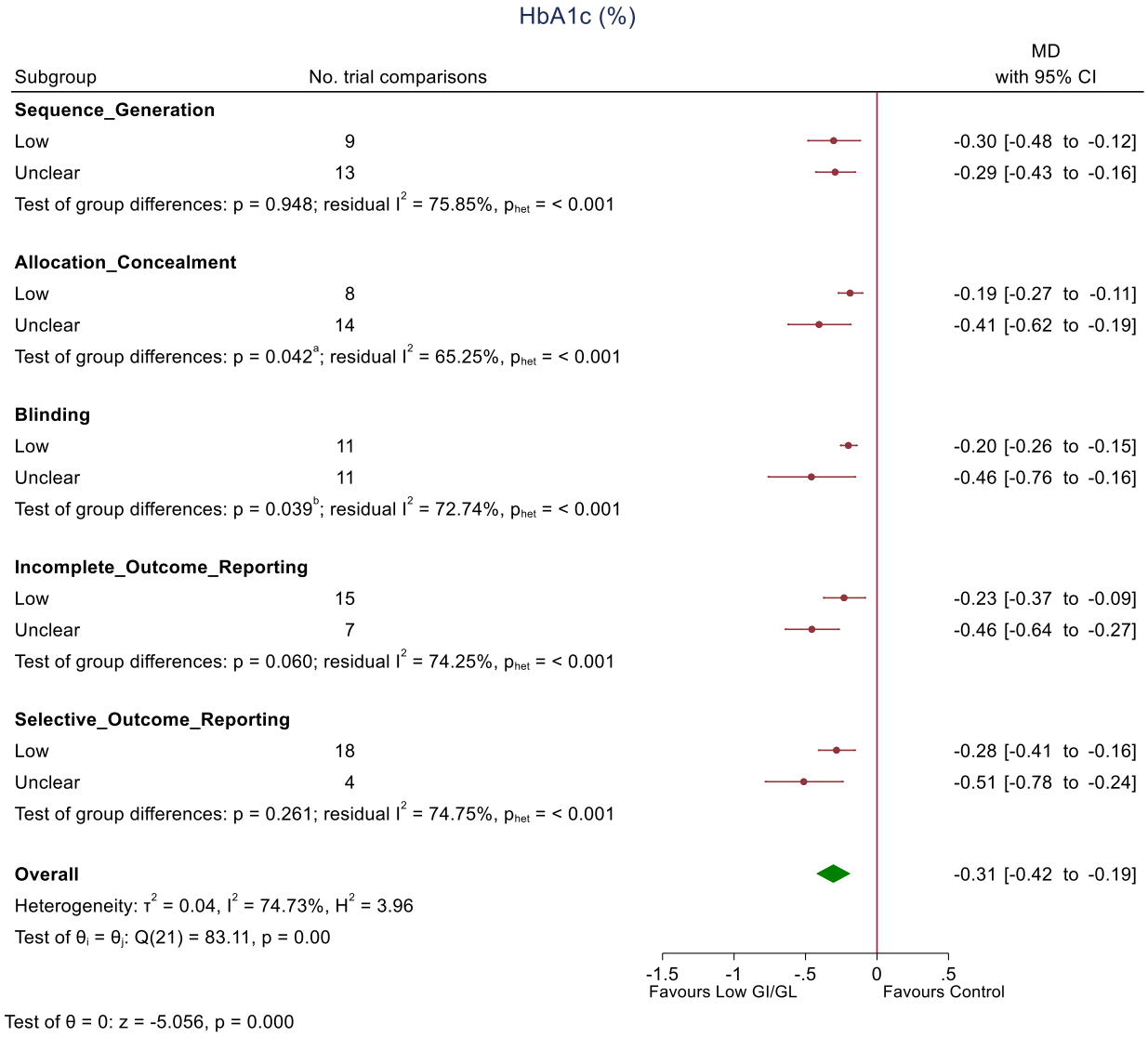
CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=3 trial comparisons missing data for Test GI and Test GL, and N=4 trial comparisons for Diff in GI and Diff in GL. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

a Pairwise between-subgroup mean differences (95% CIs) for Funding were as follows: 0.02% (-0.22, 0.26) (1 vs. 2), 0.04% (-0.18, 0.25) (1 vs. 3), -0.55% (-0.85, -0.24) (1 vs. 4), 0.02% (-0.26, 0.29) (2 vs. 3), -0.57% (-0.92, -0.22) (2 vs. 4), -0.58% (-0.91, -0.25) (3 vs. 4).

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; HbA1c, hemoglobin A1c; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S32: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on HbA1c (%) in diabetes

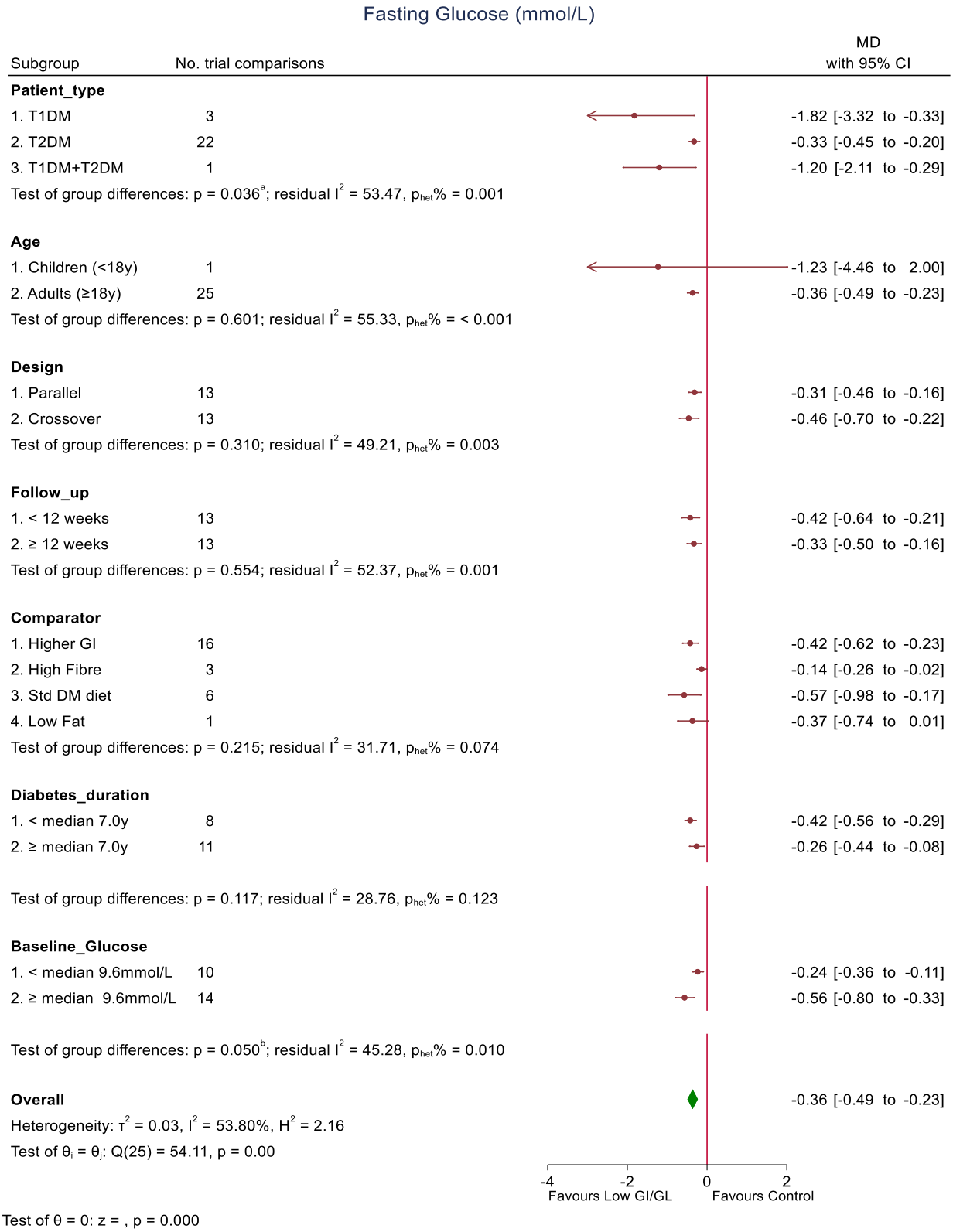


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on HbA1c. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at P<0.10 and I^2>50% considered to be evidence of substantial heterogeneity. P<0.05 indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.

a Pairwise between-subgroup mean differences (95% CIs) for Allocation concealment were as follows: -0.24% (-0.42, -0.01) (low vs unclear).
b Pairwise between-subgroup mean differences (95% CIs) for Blinding were as follows: -0.25% (-0.49, -0.01) (low vs unclear).

CI, confidence interval; HbA1c, hemoglobin A1c; GI, glycemic index; GL, glycemic load; MD, mean difference

Supplemental Figure S33 (1 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on fasting glucose (mmol/L) in diabetes*



The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on fasting glucose. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

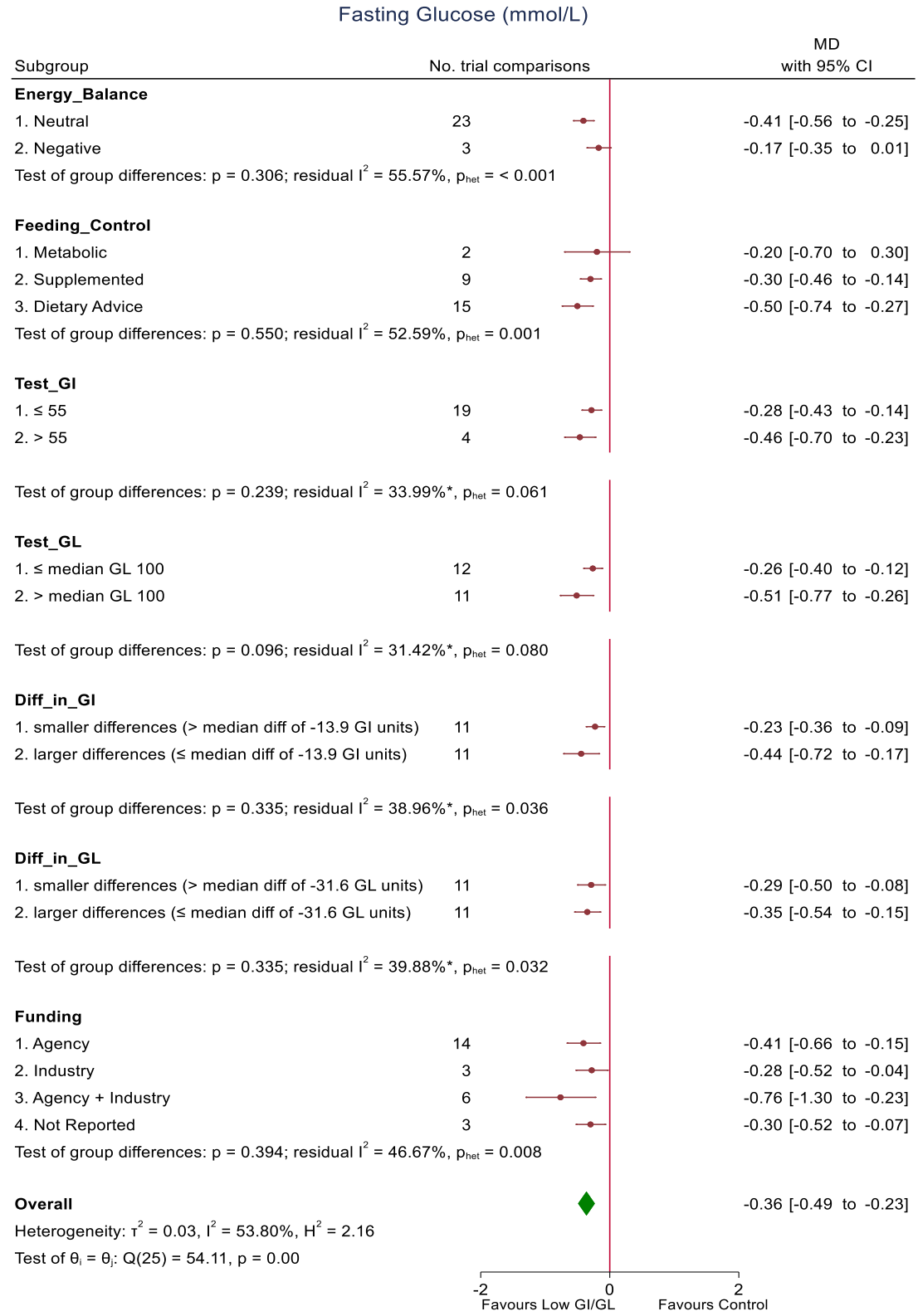
*N=7 trial comparisons missing data for disease duration, N=2 missing data for baseline fasting glucose.

[a]Pairwise between-subgroup mean differences (95% CIs) for patient type were as follows: -1.49mmol/L (-3.01, 0.03) (1 vs. 2), -0.87 (-1.85, 0.109) (1 vs. 3), 0.624 (-1.17, 2.42) (2 vs. 3).

[b]Pairwise between-subgroup mean differences (95% CIs) for Baseline glucose were as follows: -0.29mmol/L (-0.57, 0.00) (1 vs. 2).

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference; Std DM diet, standard diabetes diet; T1DM, type 1 diabetes; T2DM, type 2 diabetes; y, years

Supplemental Figure S33 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on fasting glucose (mmol/L) in diabetes*

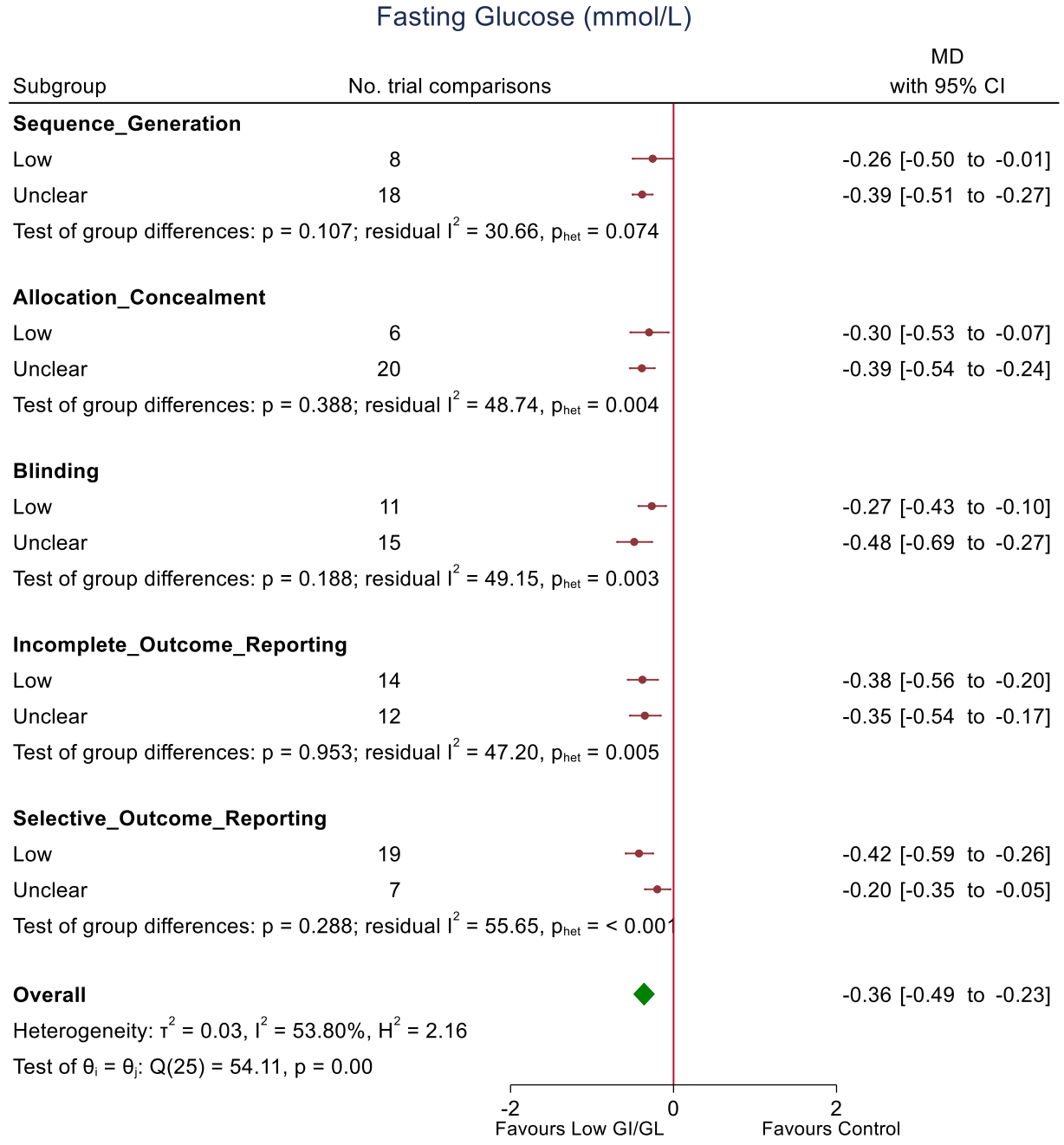


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on fasting glucose. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=3 trial comparisons missing data for absolute Test GI and Test GL, and 4 trial comparisons missing data for Diff in GI and Diff in GL.
Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

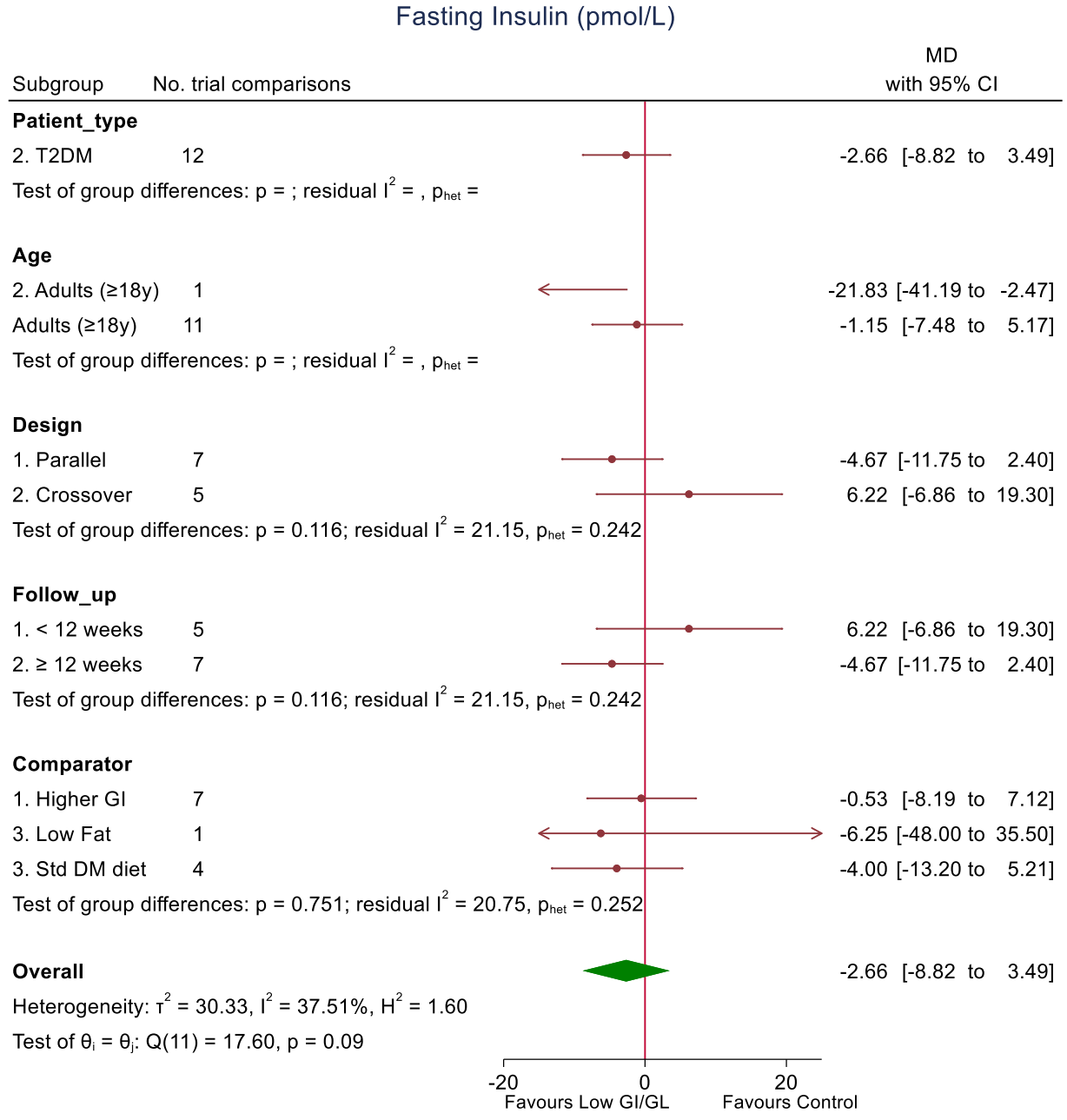
Supplemental Figure S34: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on fasting glucose (mmol/L) in diabetes



The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on fasting glucose. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity.

P<0.05 indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.
CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference

Supplemental Figure S35 (1 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on fasting insulin (pmol/L) in diabetes*



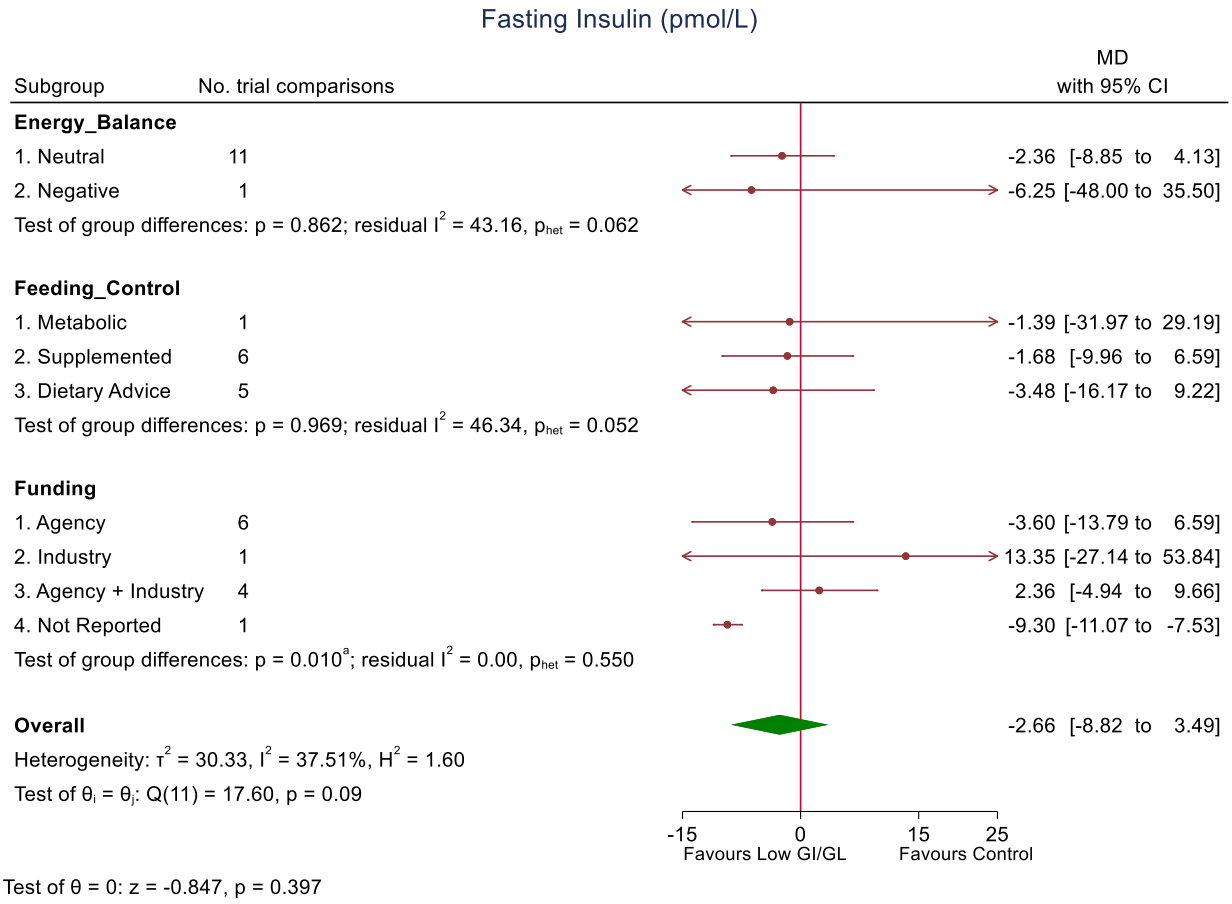
The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on fasting insulin. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic,

with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

* N=5 trial comparisons did not report baseline diabetes duration and N=3 did not report baseline insulin. Thus, since there were <10 trial comparisons, these subgroup analyses were not reported.

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference; Std DM diet, standard diabetes diet; T2DM, type 2 diabetes; y, years

Supplemental Figure S35 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on fasting insulin (pmol/L) in diabetes*



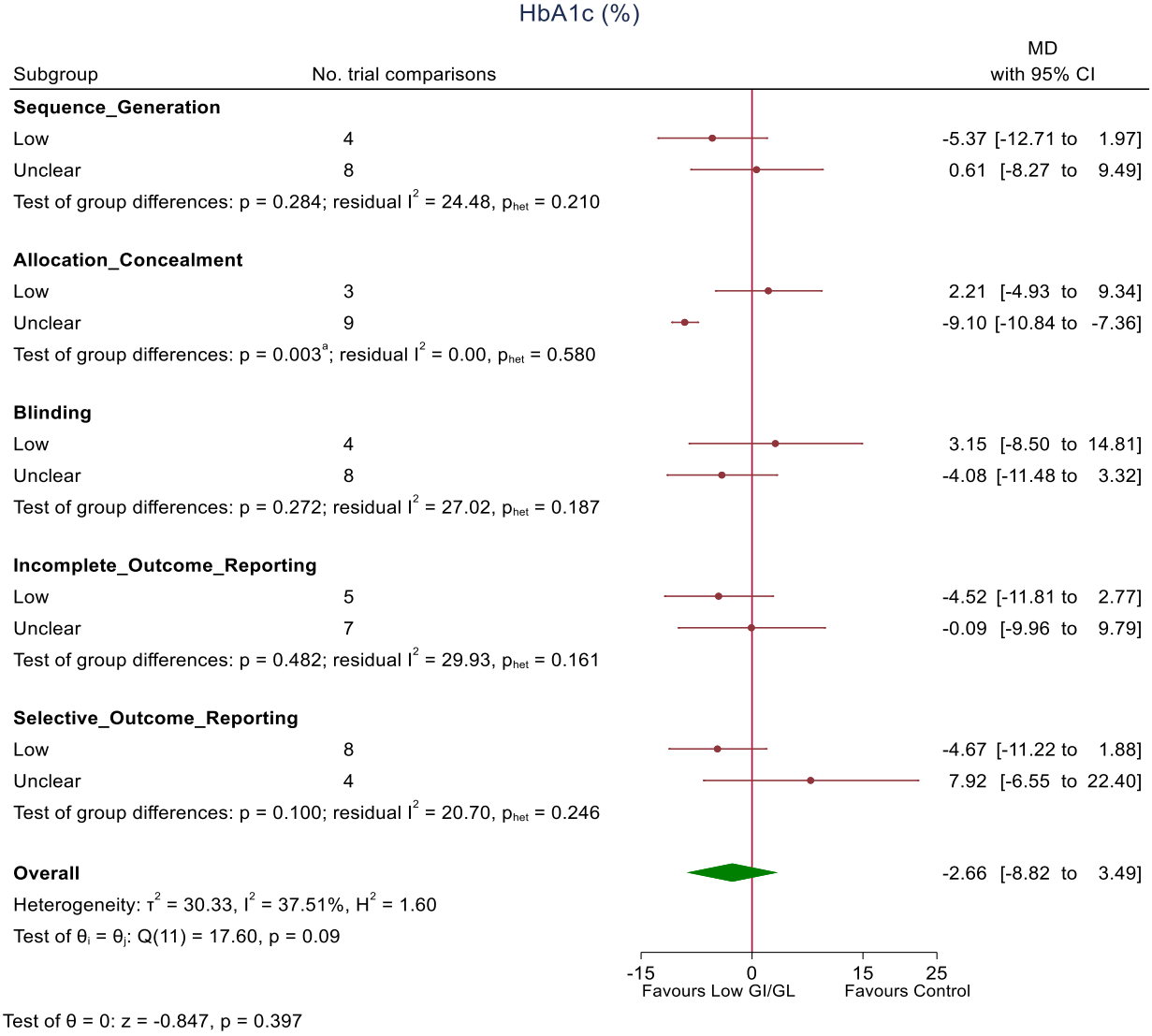
The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on fasting insulin. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at P<0.10 and I^2>50% considered to be evidence of substantial heterogeneity. P<0.05 indicates that the effect size differed between levels of the subgroup.

* N=4 trial comparisons did not report absolute Test GI and Test GL and N=5 trial comparisons did not report the data for Diff in GI and Diff in GL. Thus, since there were <10 trial comparisons, these subgroup analyses were not reported.
Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

a Pairwise between-subgroup mean differences (95% CIs) for Funding were as follows: 17.0pmol/L (-24.6, 58.6) (1 vs. 2) to 6.0pmol/L (-6.15, 18.2) (1 vs. 3) to -5.66pmol/L (-15.5, 4.21) (1 vs. 4) to -11.0pmol/L (-52.1, 30.2) (2 vs. 3) to -22.6pmol/L (-63.2, 17.9) (2 vs. 4) to -11.7pmol/L (-19.2, -4.15) (3 vs. 4).

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S36: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on fasting insulin (pmol/L) in diabetes

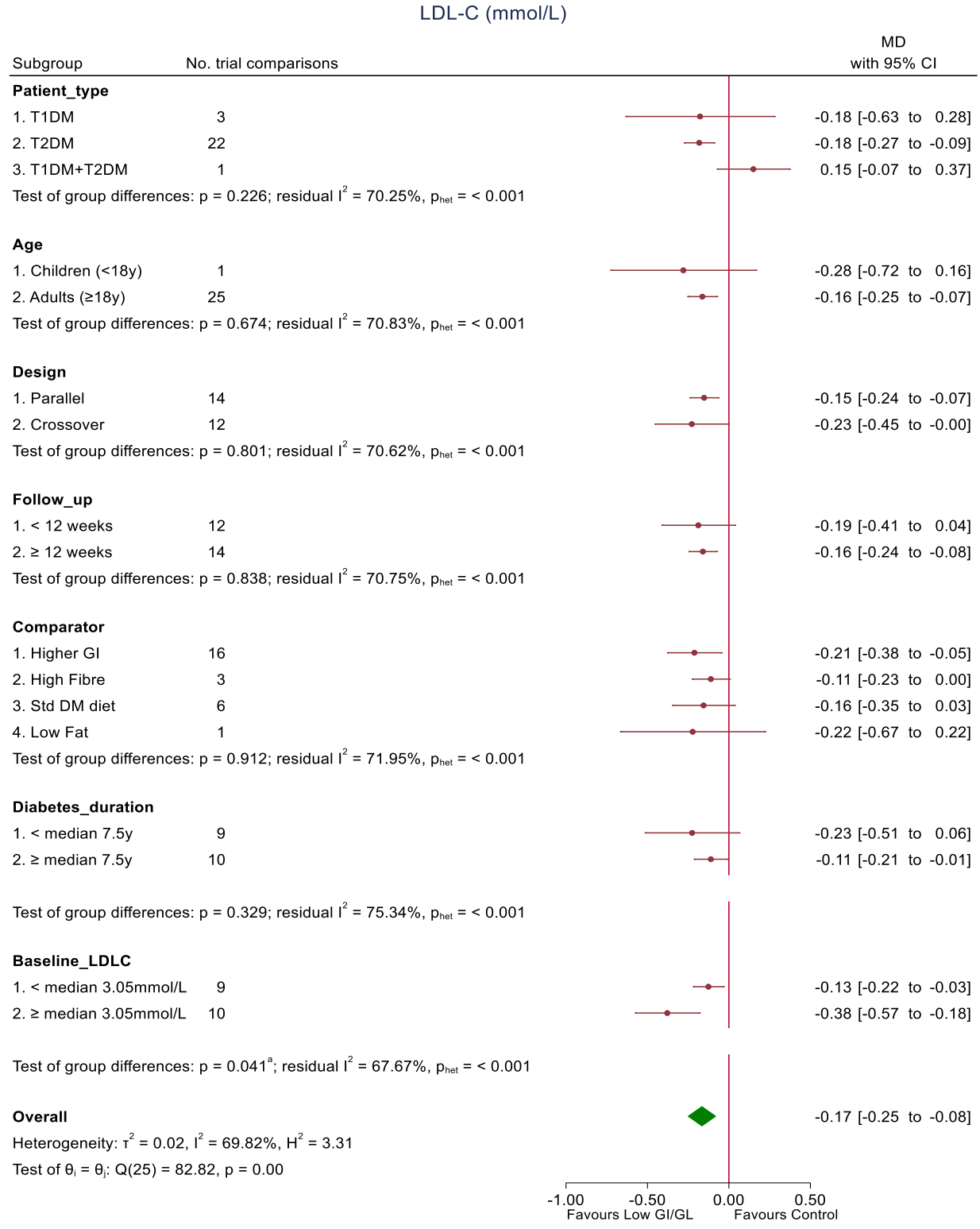


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on fasting insulin. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at P<0.10 and I^2>50% considered to be evidence of substantial heterogeneity. P<0.05 indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.

a Pairwise between-subgroup mean differences (95% CIs) for allocation concealment were as follows: -11.3pmol/L (-18.6, -3.96) (Low vs Unclear).

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference

Supplemental Figure S37 (1 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on LDL-C (mmol/L) in diabetes*



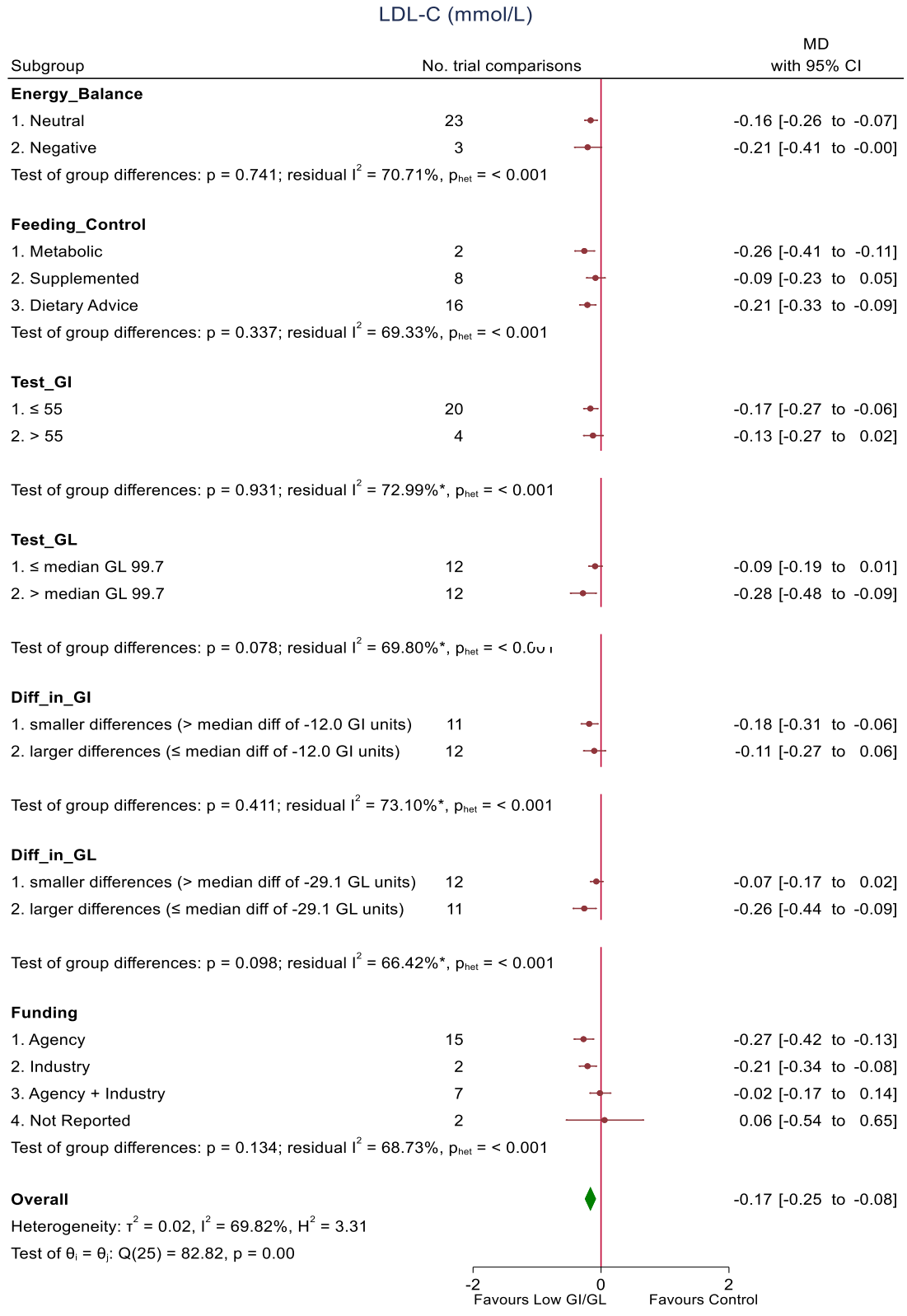
The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on LDL-C. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=7 trial comparisons were missing data for disease duration, N=7 missing data for baseline LDL-C

[a]Pairwise between-subgroup mean differences (95% CIs) for Baseline LDL-C were as follows: -0.19mmol/L (-0.37, -0.01) (1 vs. 2).

CI, confidence interval; GI, glycemic index; GL, glycemic load; LDL-C, low-density lipoprotein-cholesterol; MD, mean difference; Std DM diet, standard diabetes diet; T1DM, type 1 diabetes; T2DM, type 2 diabetes; y, years

Supplemental Figure S37 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on LDL-C (mmol/L) in diabetes*

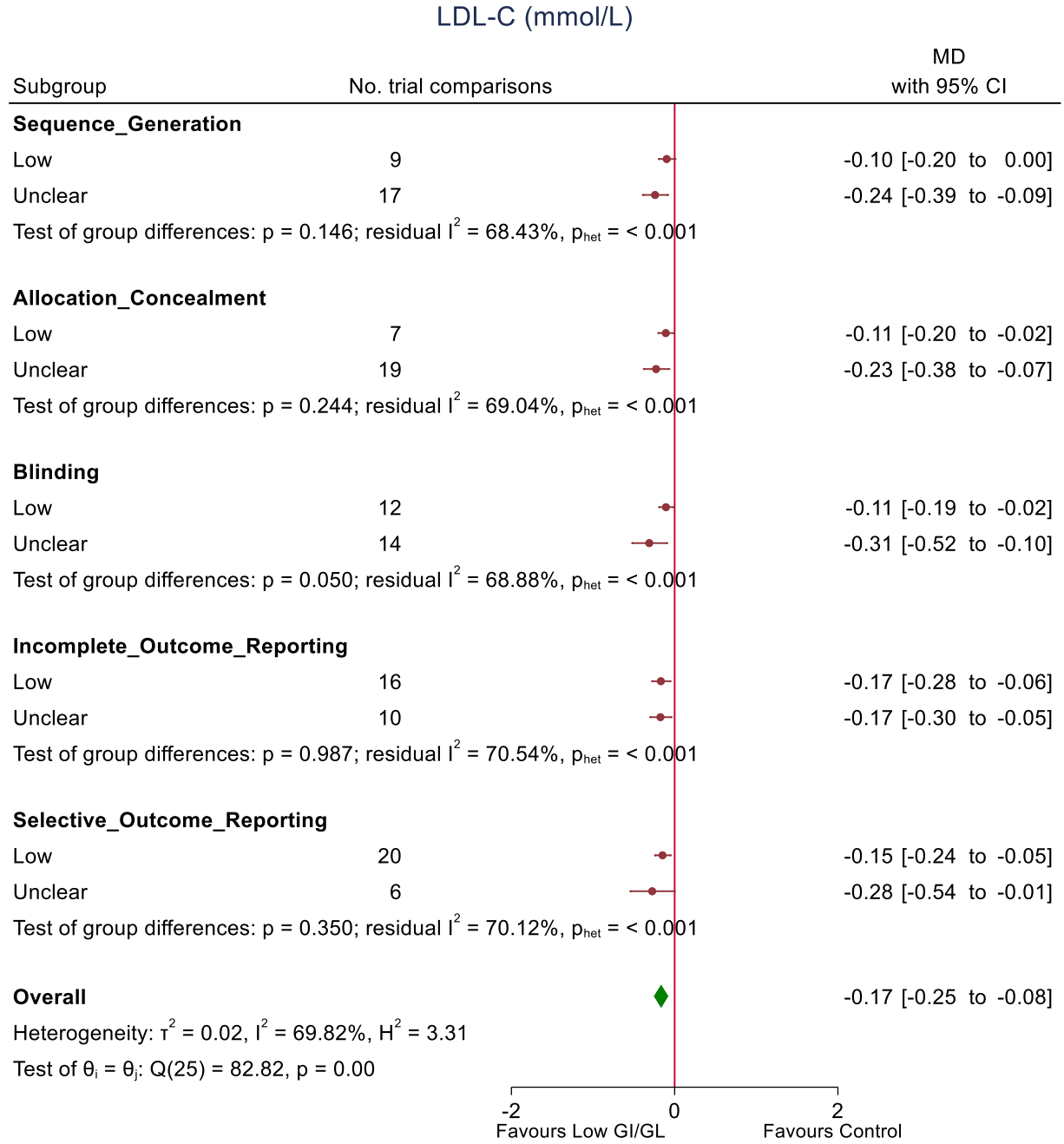


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on LDL-C. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=2 trial comparisons missing data for Test GI and Test GL, and 3 trial comparisons missing data for Diff in GI and Diff in GL. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; LDL-C, low-density lipoprotein-cholesterol; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S38: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on LDL-C (mmol/L) in diabetes

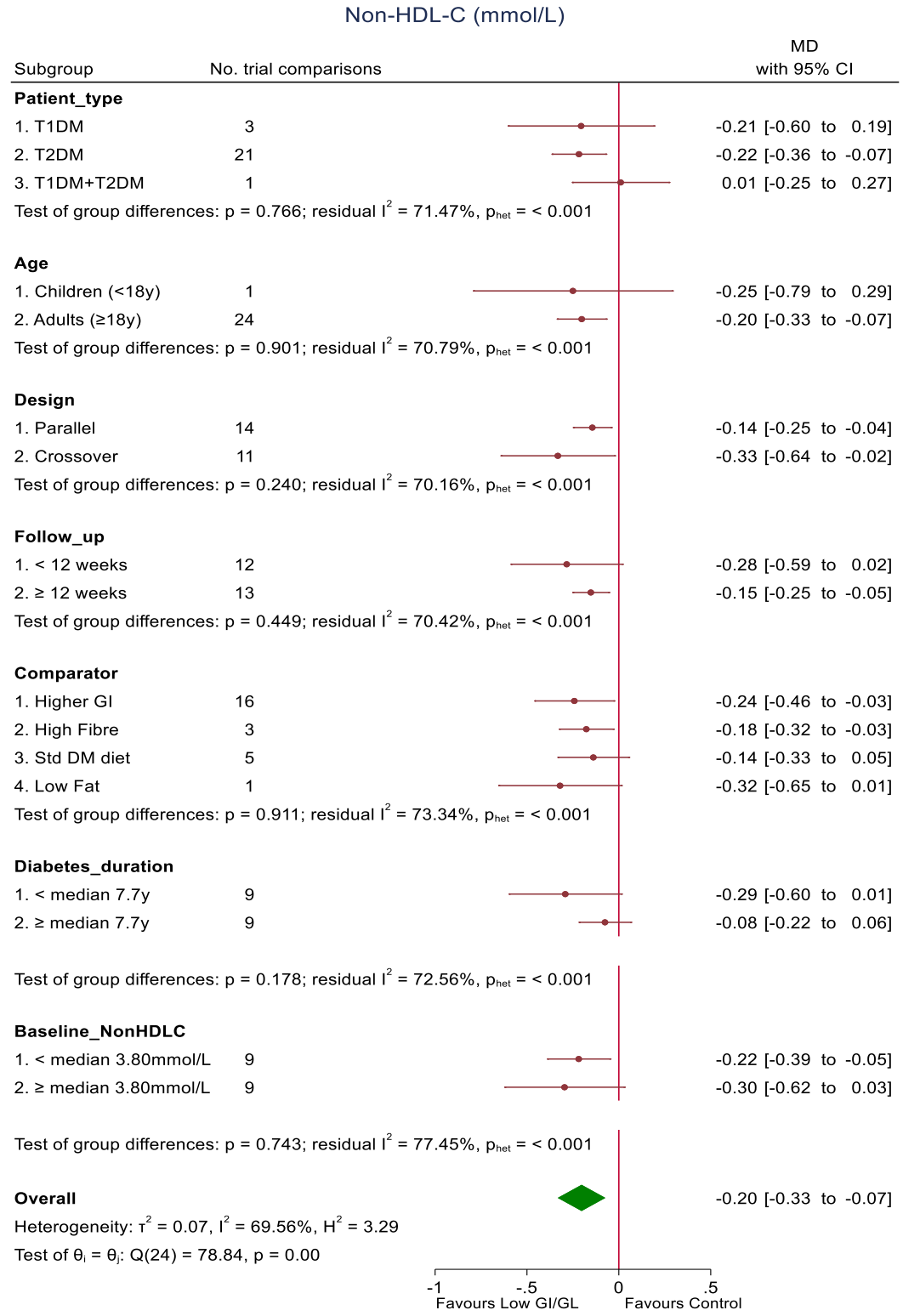


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on LDL-C. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study

heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.

CI, confidence interval; GI, glycemic index; GL, glycemic load; LDL-C, low-density lipoprotein-cholesterol; MD, mean difference

Supplemental Figure S39 (1of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on non-HDL-C (mmol/L) in diabetes*



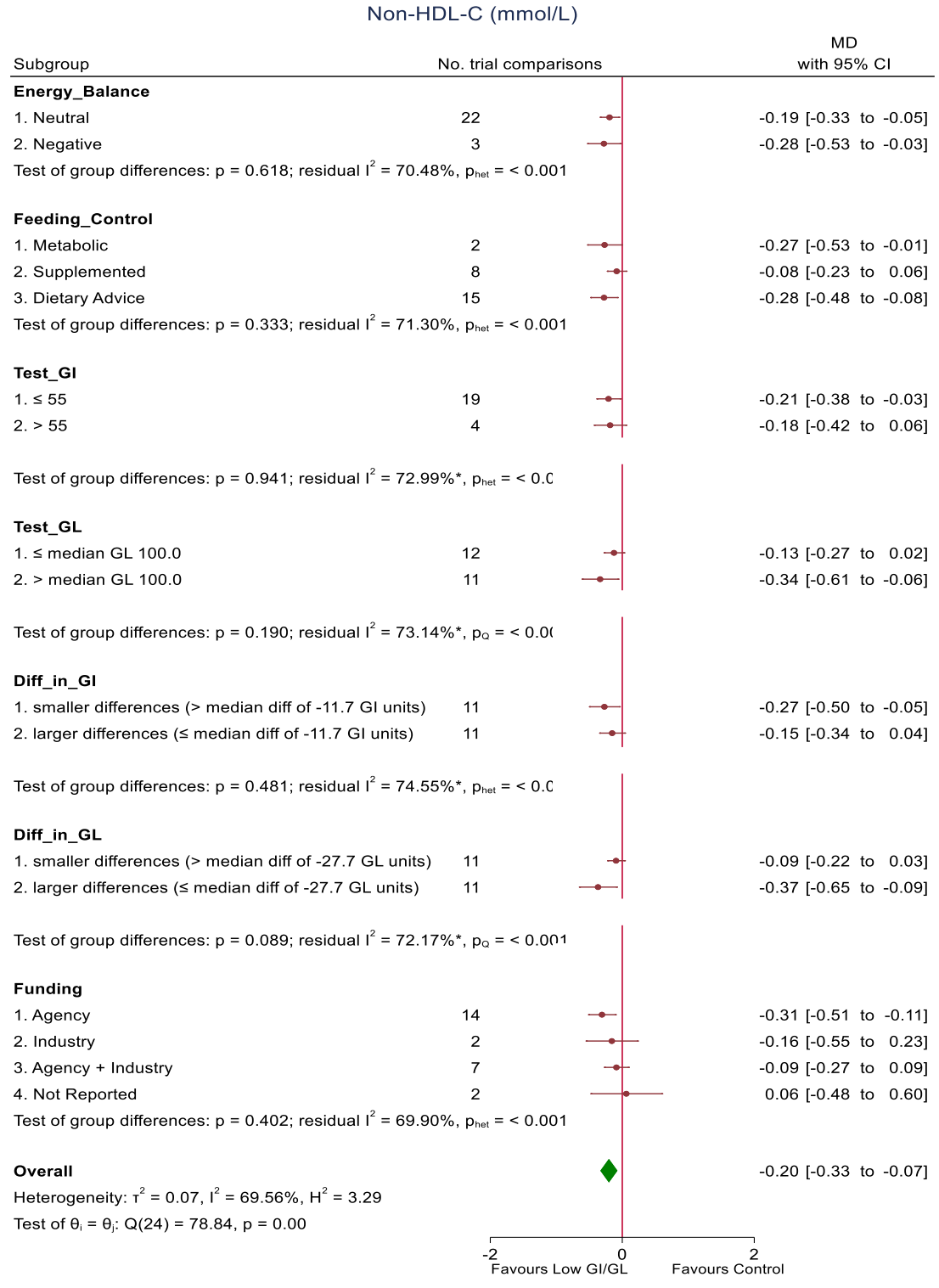
Test of θ = 0: z = -3.071, p = 0.002

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on Non-HDL-C. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=7 trial comparisons were missing data for disease duration, N=7 missing data for baseline Non-HDL-C

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference; Non-HDL-C, non-high-density lipoprotein-cholesterol; Std DM diet, standard diabetes diet; T1DM, type 1 diabetes; T2DM, type 2 diabetes; y, years

Supplemental Figure S39 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on non-HDL-C (mmol/L) in diabetes*



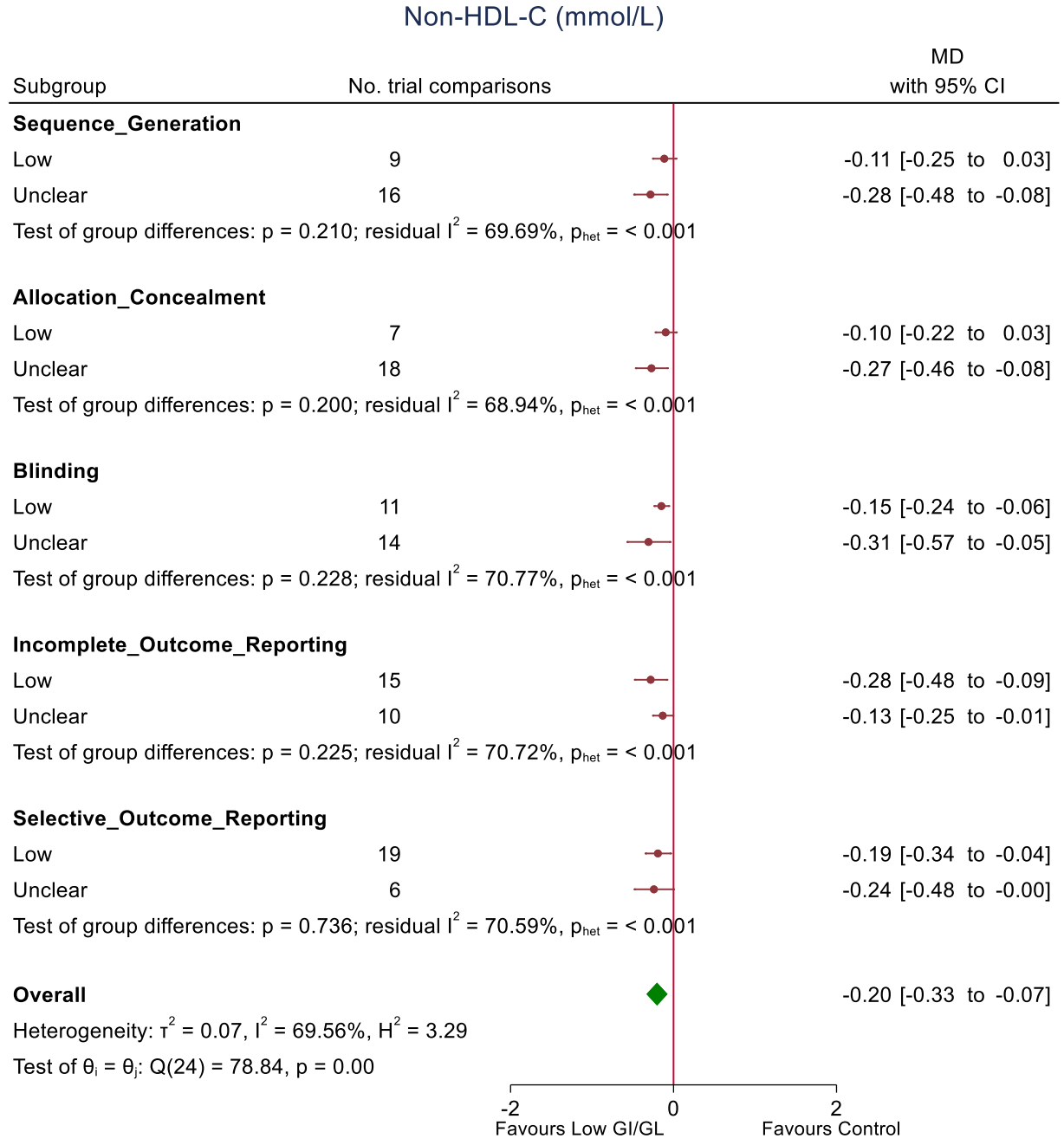
Test of θ = 0: z = -3.071, p = 0.002

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on Non-HDL-C. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=2 trial comparisons missing data for Test GI and Test GL, and 3 trial comparisons missing data for Diff in GI and Diff in GL. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; Non-HDL-C, non-high-density lipoprotein-cholesterol; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S40: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on non-HDL-C (mmol/L) in diabetes



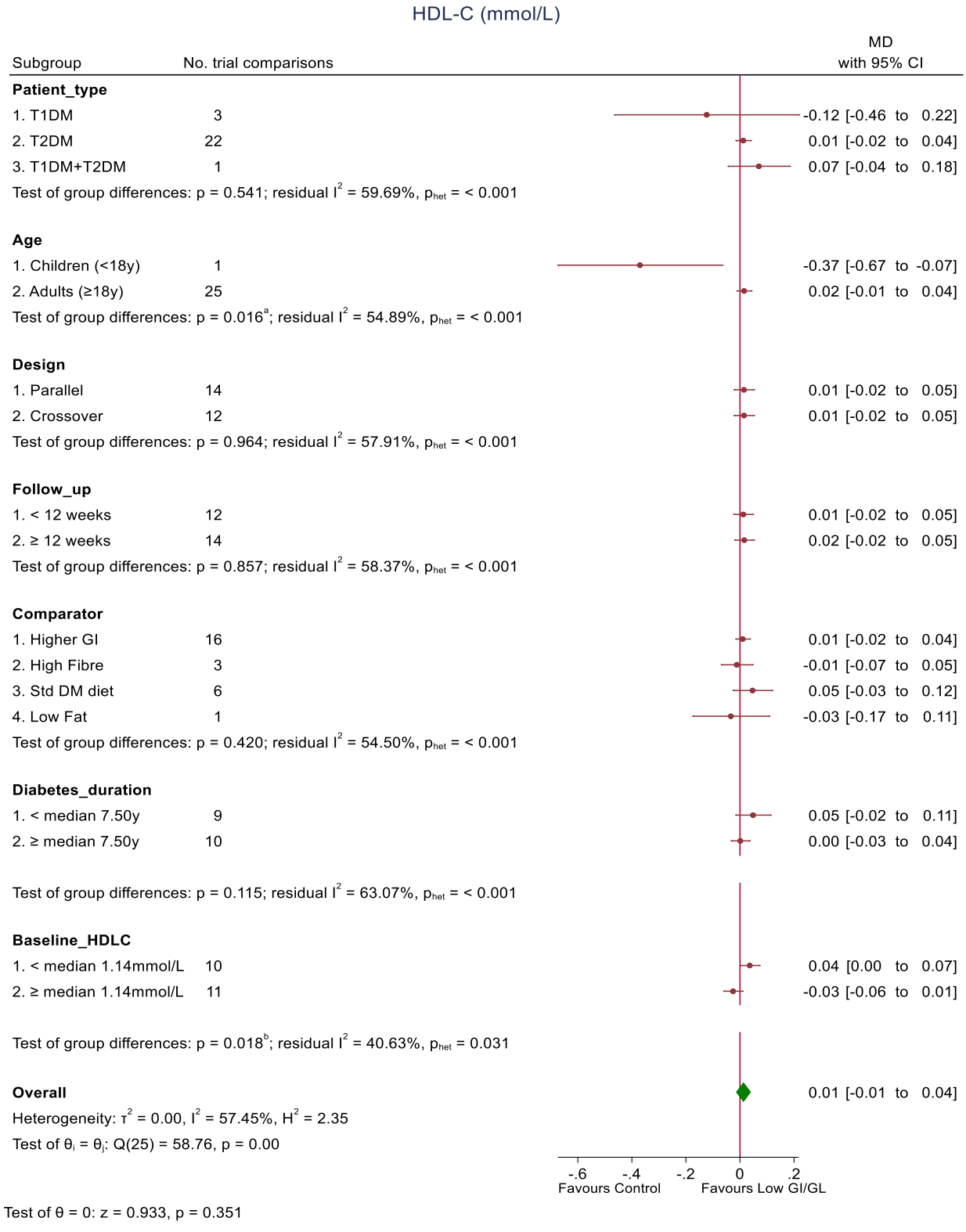
Test of θ = 0: z = -3.071, p = 0.002

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on Non-HDL-C. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic,

with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference; Non-HDL-C, non-high-density lipoprotein-cholesterol

Supplemental Figure S41 (1 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on HDL-C (mmol/L) in diabetes*



The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on HDL-C. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

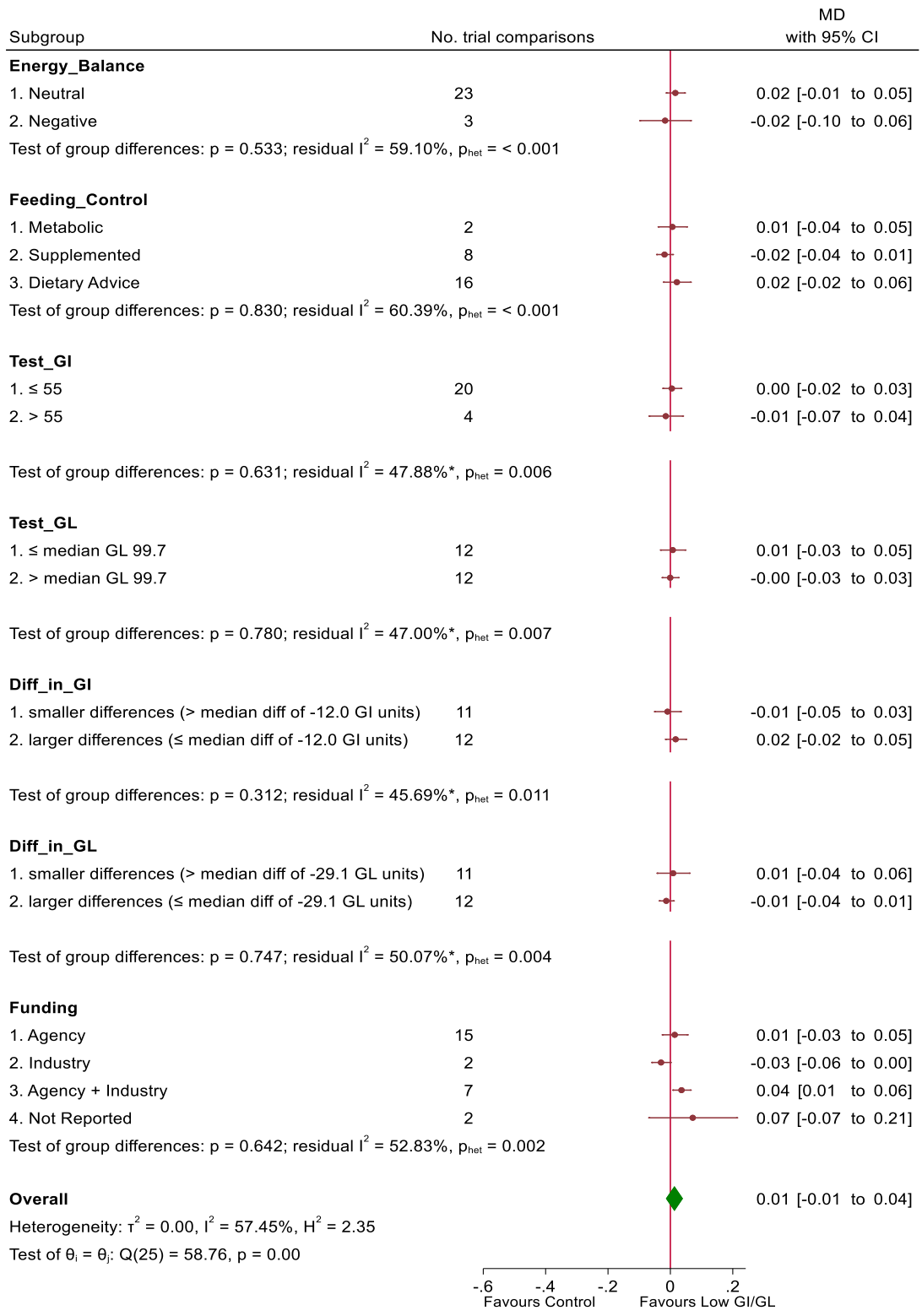
*N=7 trial comparisons were missing data for disease duration, N=5 missing data for baseline HDL-C

[a]Pairwise between-subgroup mean differences (95% CIs) for Age were as follows: -0.39mmol/L (-0.70, -0.07) (1 vs. 2).

[b]Pairwise between-subgroup mean differences (95% CIs) for Baseline HDL-C were as follows: -0.06mmol/L (-0.11, -0.01) (1 vs. 2).

CI, confidence interval; GI, glycemic index; GL, glycemic load; HDL-C, high-density lipoprotein-cholesterol; MD, mean difference; Std DM diet, standard diabetes diet; T1DM, type 1 diabetes; T2DM, type 2 diabetes; y, years

Supplemental Figure S41 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on HDL-C (mmol/L) in diabetes*



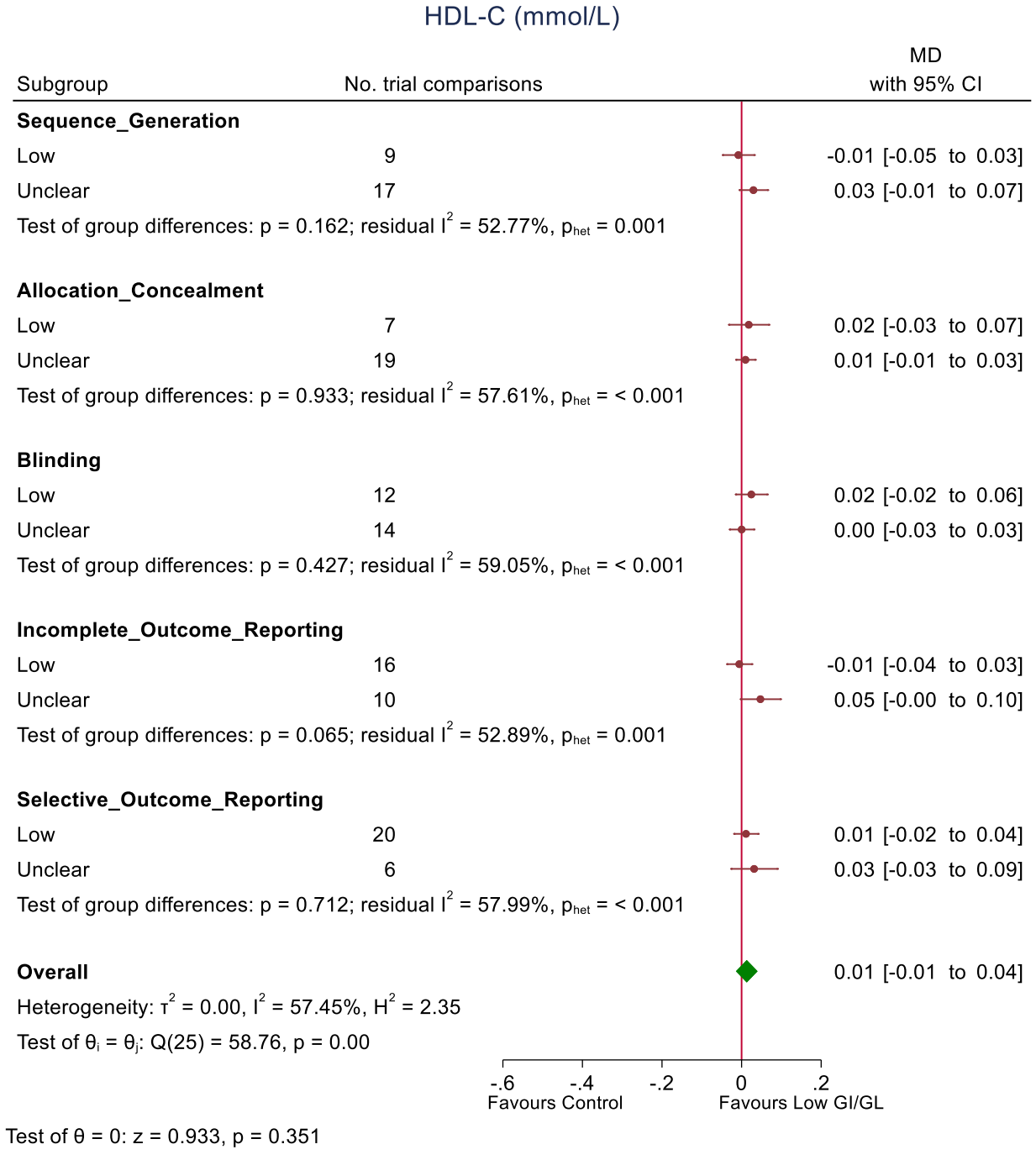
Test of θ = 0: z = 0.933, p = 0.351

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on HDL-C. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=2 trial comparisons missing data for absolute Test GI and Test GL, and 3 trial comparisons missing data for Diff in GI and Diff in GL. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; HDL-C, high-density lipoprotein-cholesterol; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S42: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on HDL-C (mmol/L) in diabetes

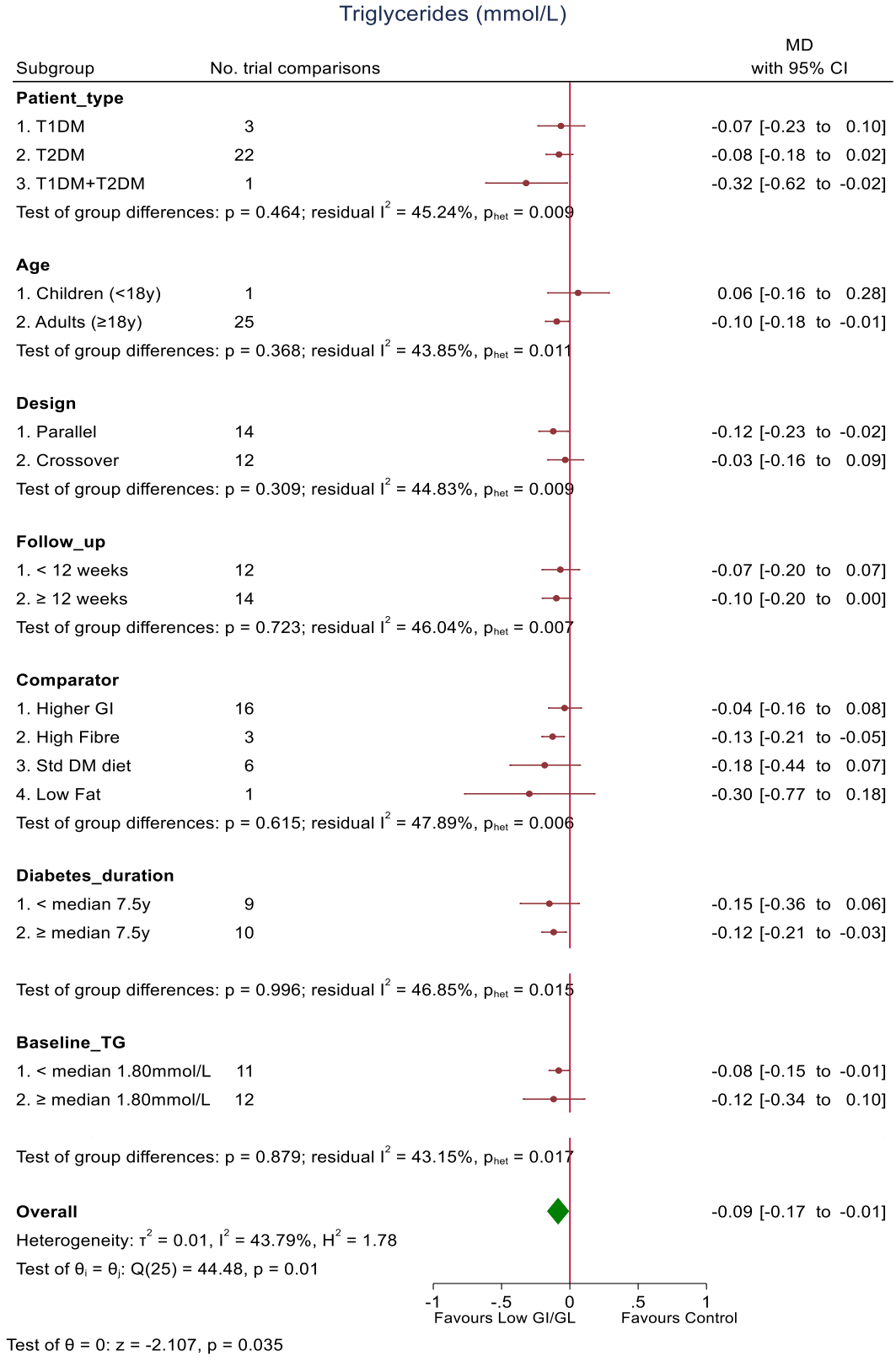


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on HDL-C. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study

heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.

CI, confidence interval; GI, glycemic index; GL, glycemic load; HDL-C, high-density lipoprotein-cholesterol; MD, mean difference

Supplemental Figure S43 (1 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on triglycerides (mmol/L) in diabetes*



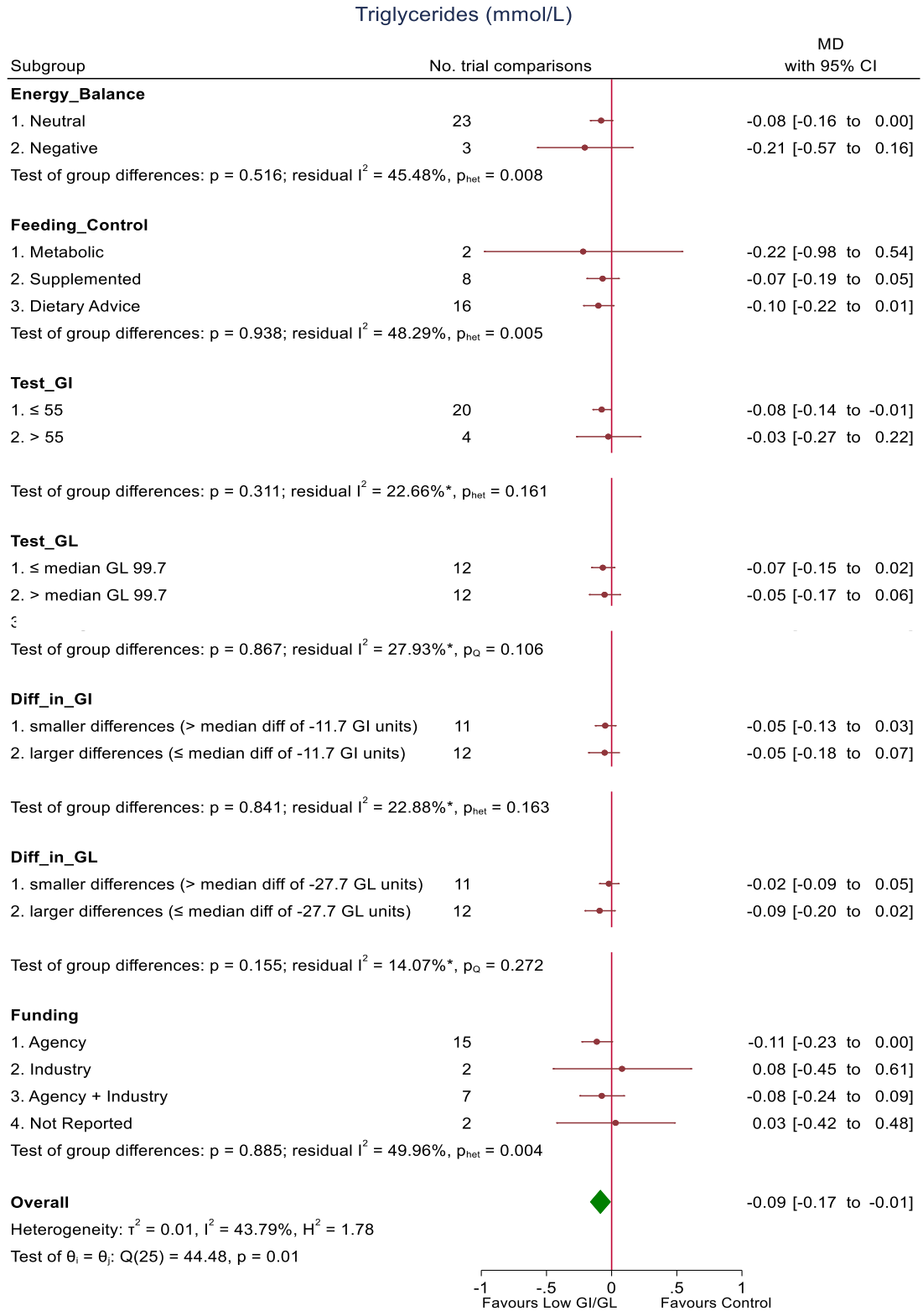
Test of θ = 0: z = -2.107, p = 0.035

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on triglycerides. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=7 trial comparisons were missing data for disease duration, N=3 missing data for baseline triglycerides

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference; Std DM diet, standard diabetes diet; T1DM, type 1 diabetes; T2DM, type 2 diabetes; TG, triglycerides; y, years

Supplemental Figure S43 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on triglycerides (mmol/L) in diabetes*



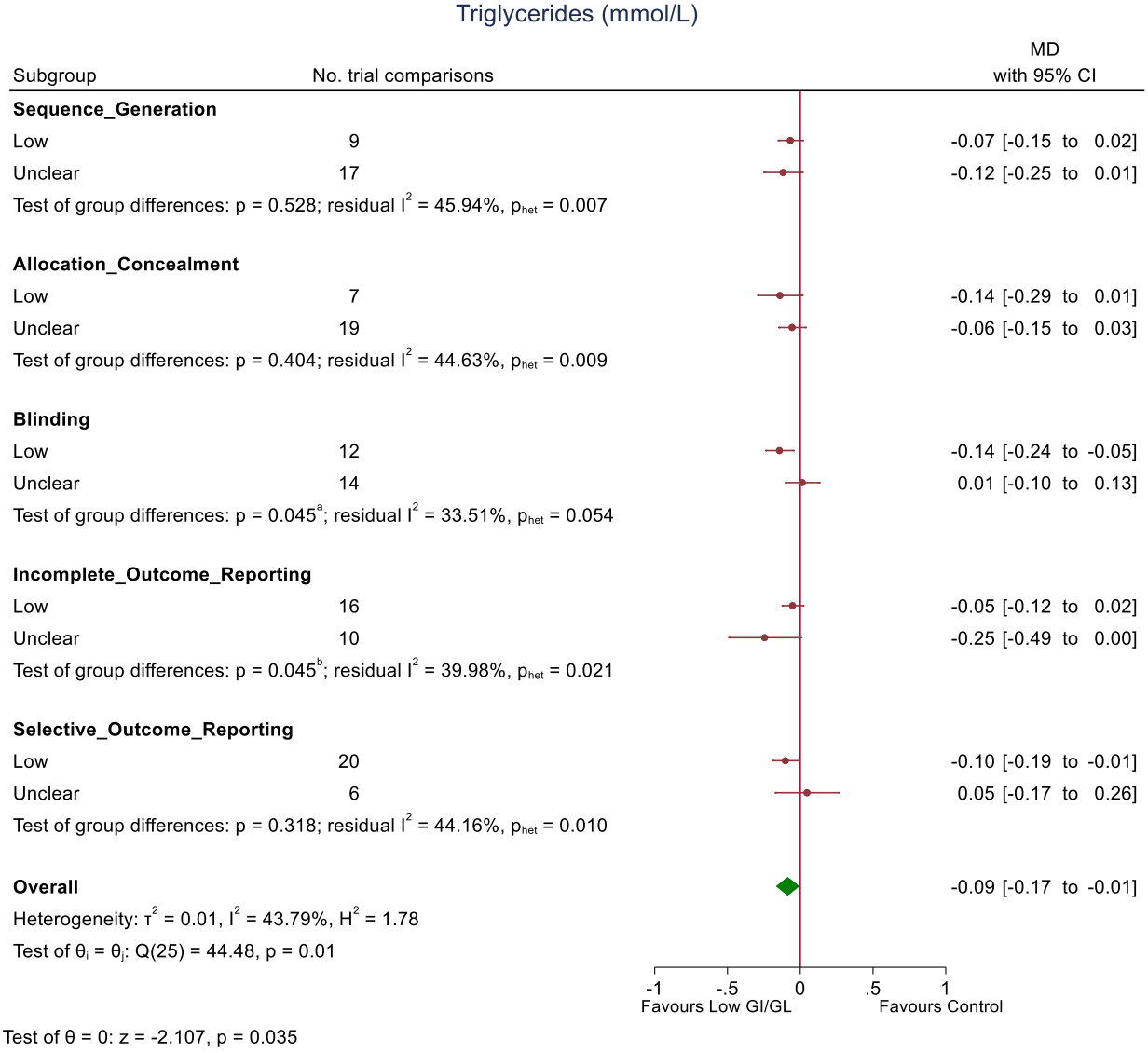
Test of θ = 0: z = -2.107, p = 0.035

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on triglycerides. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=2 trial comparisons missing data for absolute Test GI and Test GL, and 3 trial comparisons missing data for Diff in GI and Diff in GL. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S44: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on triglycerides (mmol/L) in diabetes



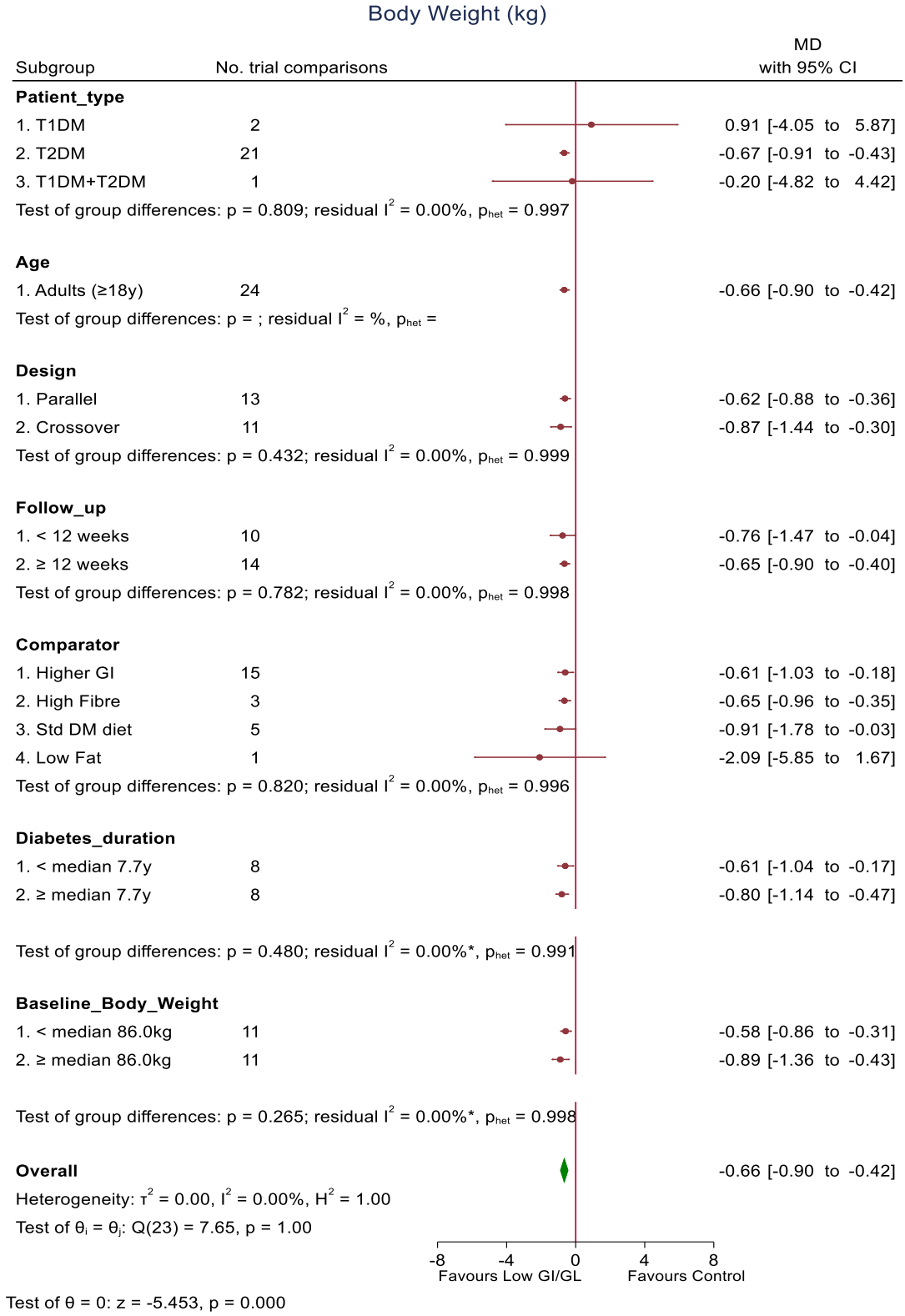
The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on triglycerides. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.

[a]Pairwise between-subgroup mean differences in triglycerides (95% CIs) for Blinding were as follows: -0.15mmol/L (0.00, 0.31) (1 vs. 2).

[b]Pairwise between-subgroup mean differences in triglycerides (95% CIs) for Incomplete outcome were as follows: -0.20mmol/L (-0.40, -0.00) (1 vs. 2).

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference

Supplemental Figure S45 (1 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on body weight (kg) in diabetes*



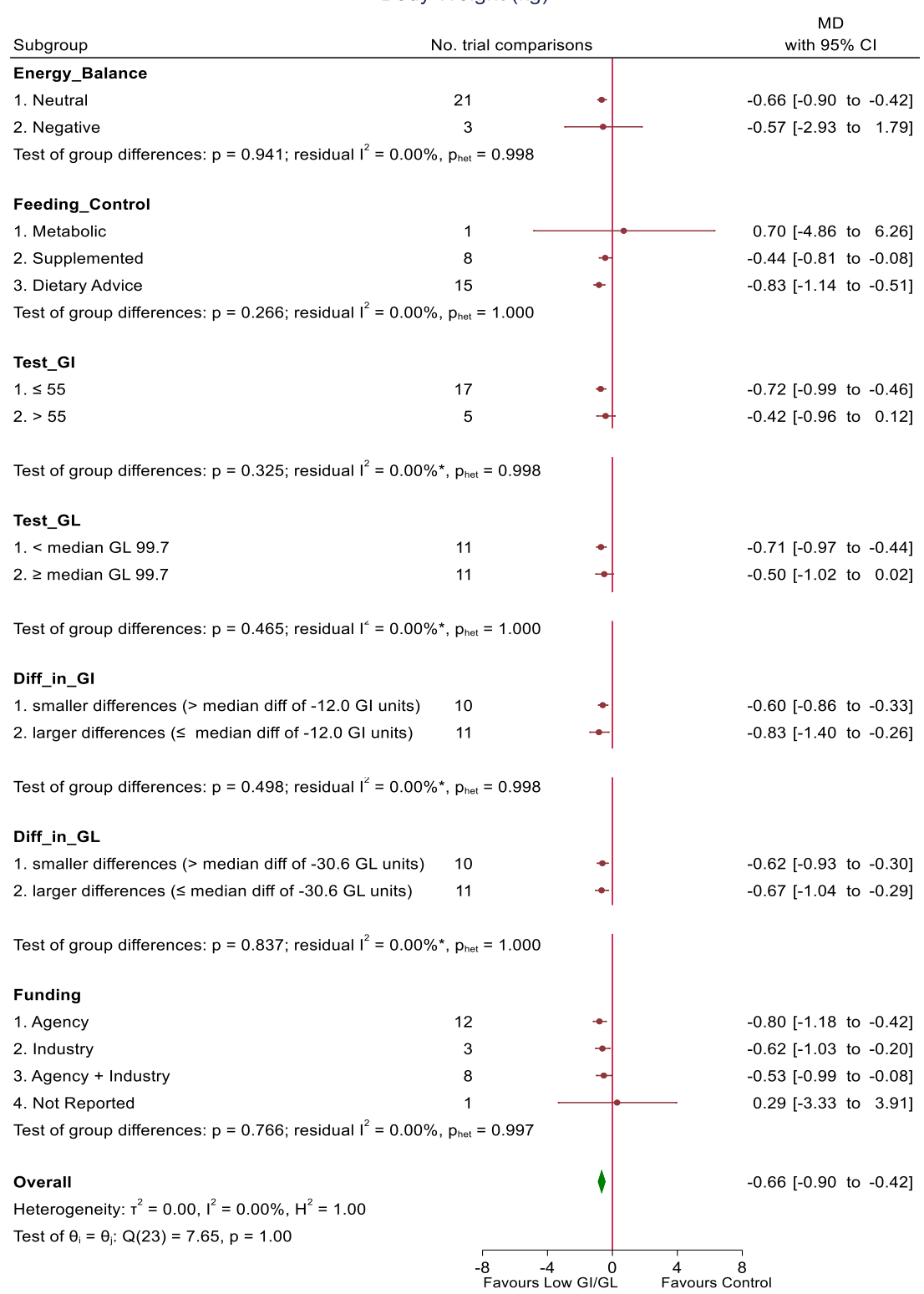
Test of θ = 0: z = -5.453, p = 0.000

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on body weight. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=8 trial comparisons were missing data for disease duration, N=2 missing data for baseline body weight.

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference; Std DM diet, standard diabetes diet; T1DM, type 1 diabetes; T2DM, type 2 diabetes; y, years

Supplemental Figure S45 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on body weight (kg) in diabetes*



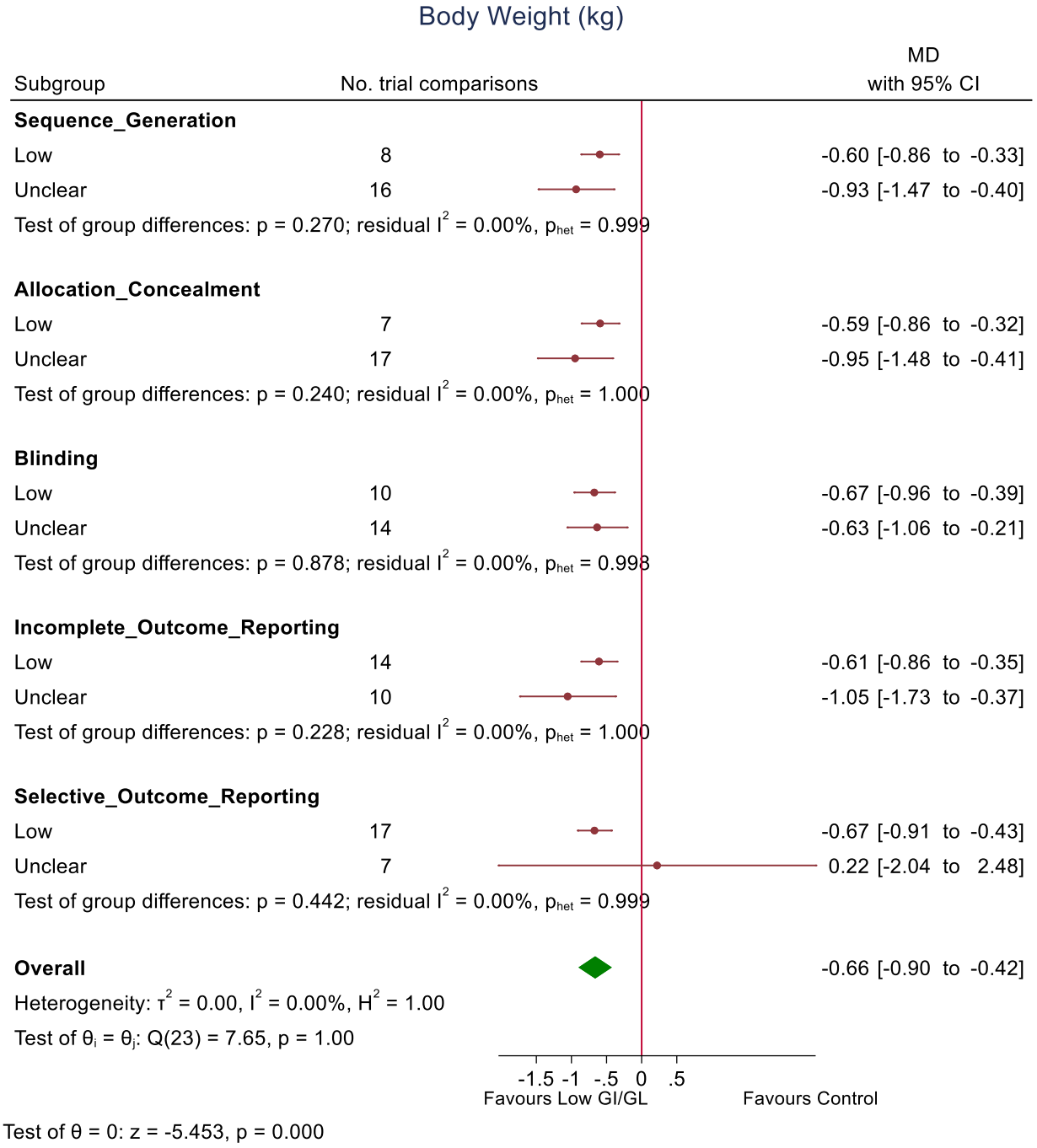
Test of θ = 0: z = -5.453, p = 0.000

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on body weight. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=2 trial comparisons missing data for absolute Test GI and Test GL, and 3 trial comparisons missing data for Diff in GI, and Diff in GL. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S46: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on body weight (kg) in diabetes

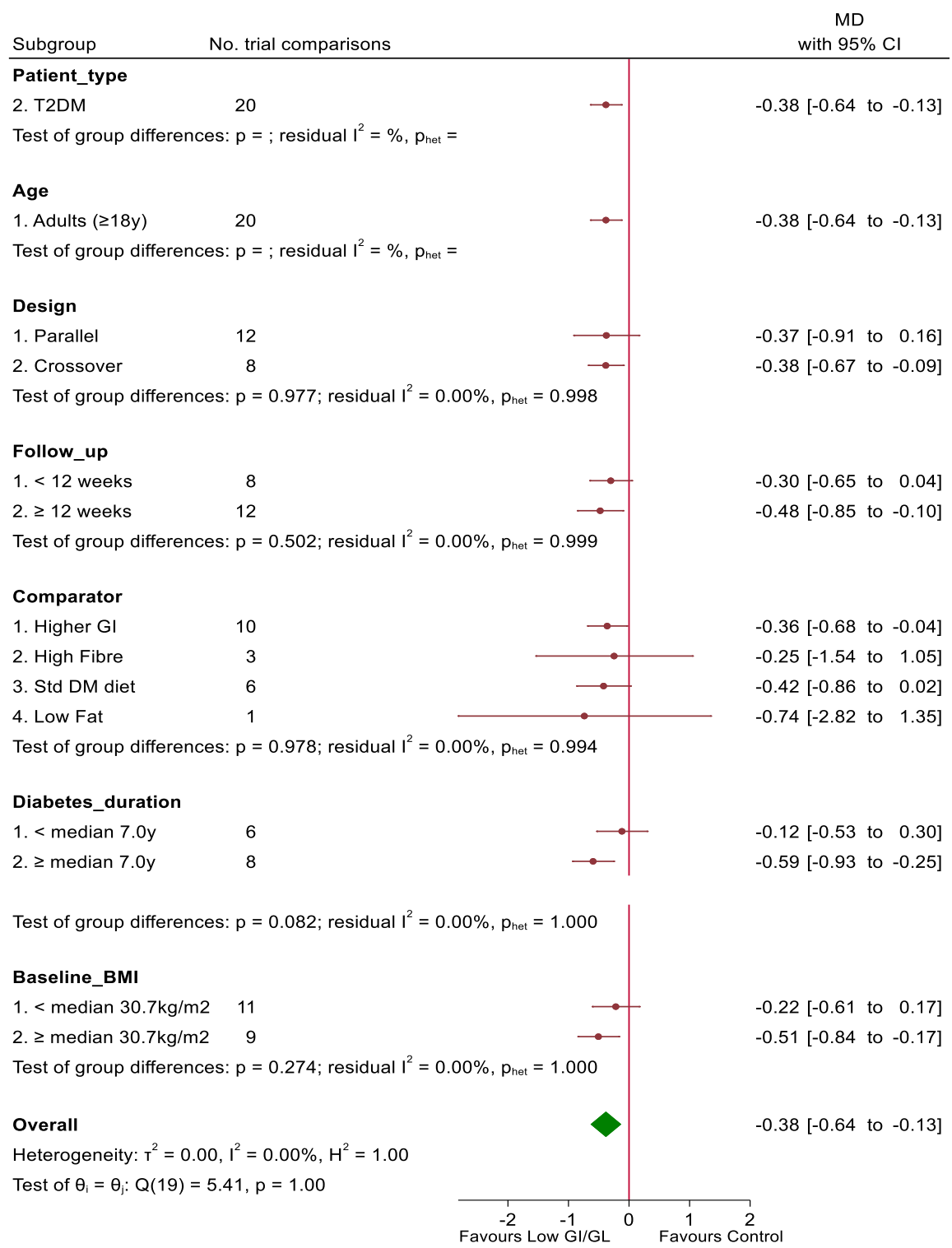


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on body weight. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model.

Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference

Supplemental Figure S47 (1 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on BMI (kg/m^2) in diabetes*



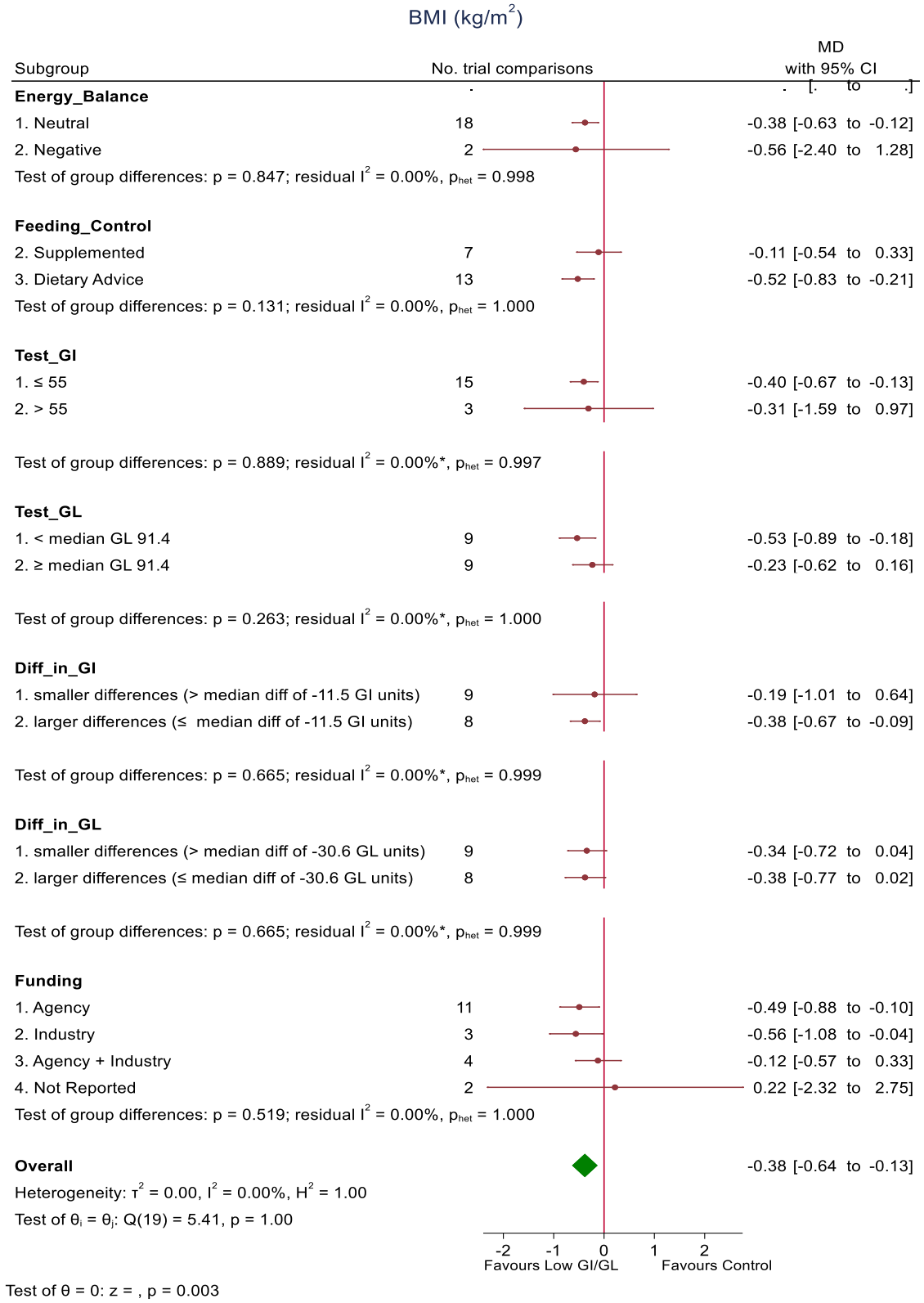
Test of θ = 0: z = -2.941, p = 0.003

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on BMI. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=6 trial comparisons were missing data for disease duration

BMI, body mass index; CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference; Std DM diet, standard diabetes diet; T2DM, type 2 diabetes; y, years

Supplemental Figure S47 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on BMI (kg/m^2) in diabetes*



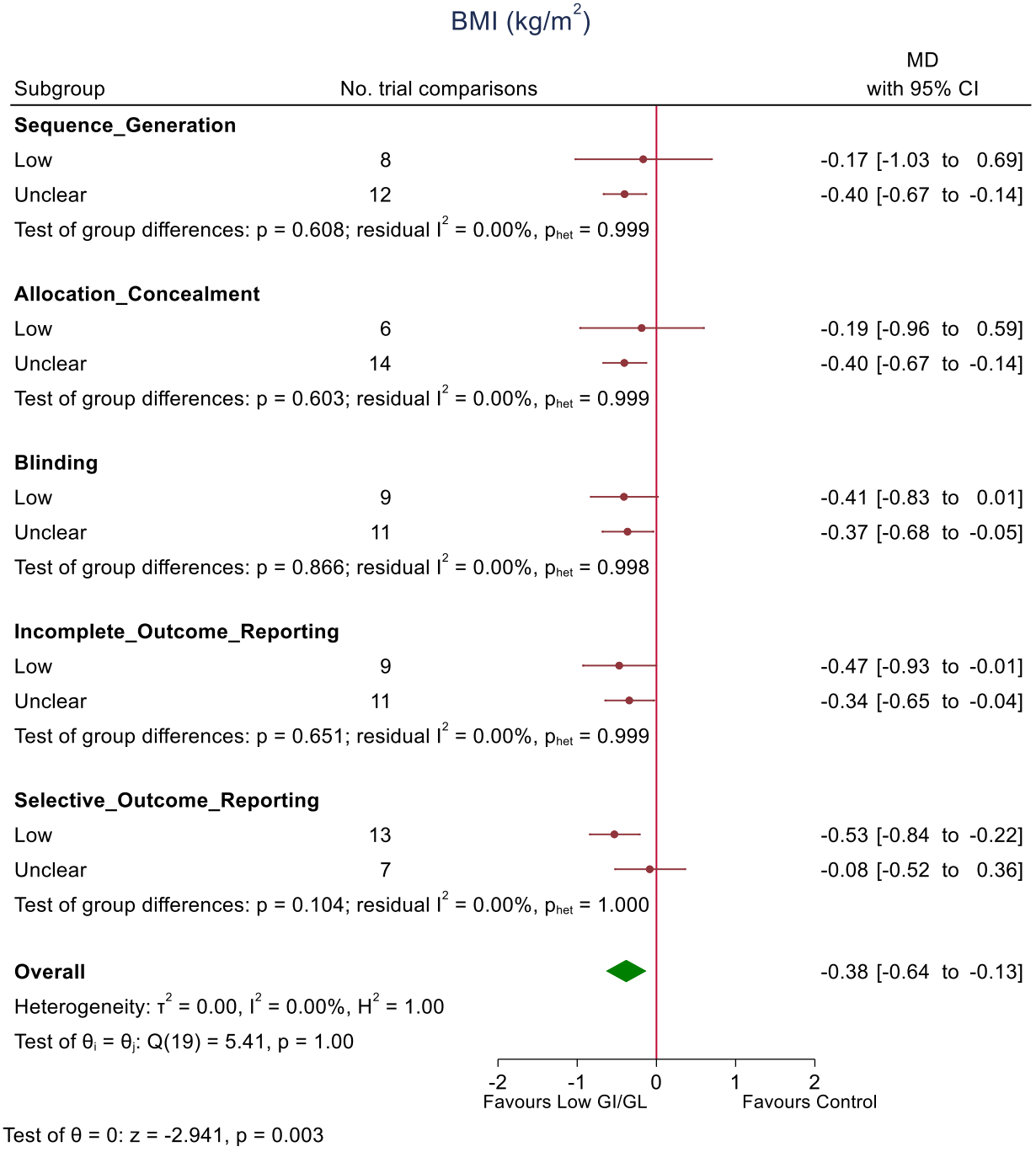
Test of θ = 0: z = , p = 0.003

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on BMI. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=2 trial comparisons missing data for absolute Test GI and Test GL, and 3 trial comparisons missing data for Diff in GI, and Diff in GL. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

BMI, body mass index; CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S48: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on BMI (kg/m²) in diabetes

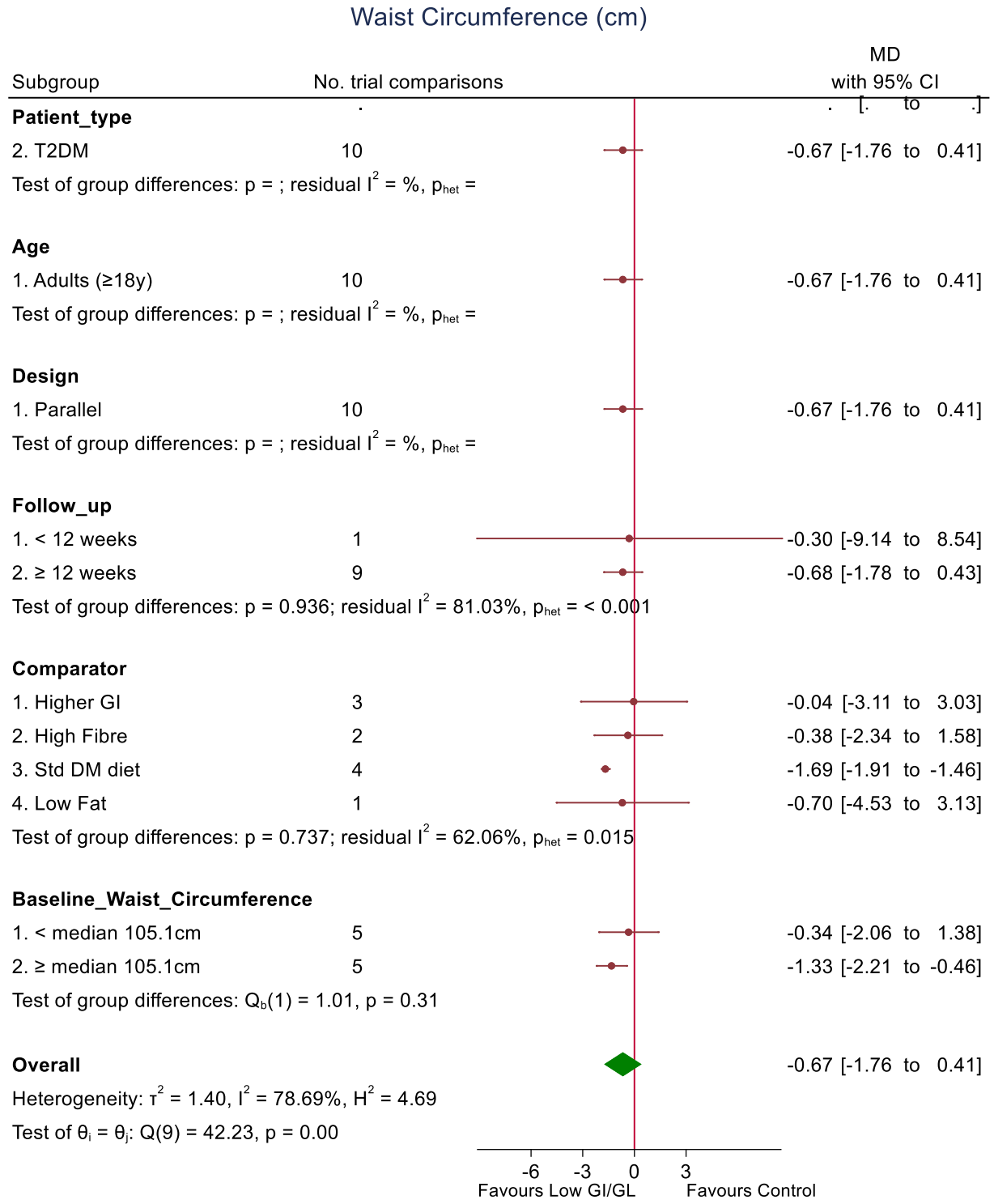


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on BMI. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study

heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.

BMI, body mass index; CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference

Supplemental Figure S49 (1 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on waist circumference (cm) in diabetes*



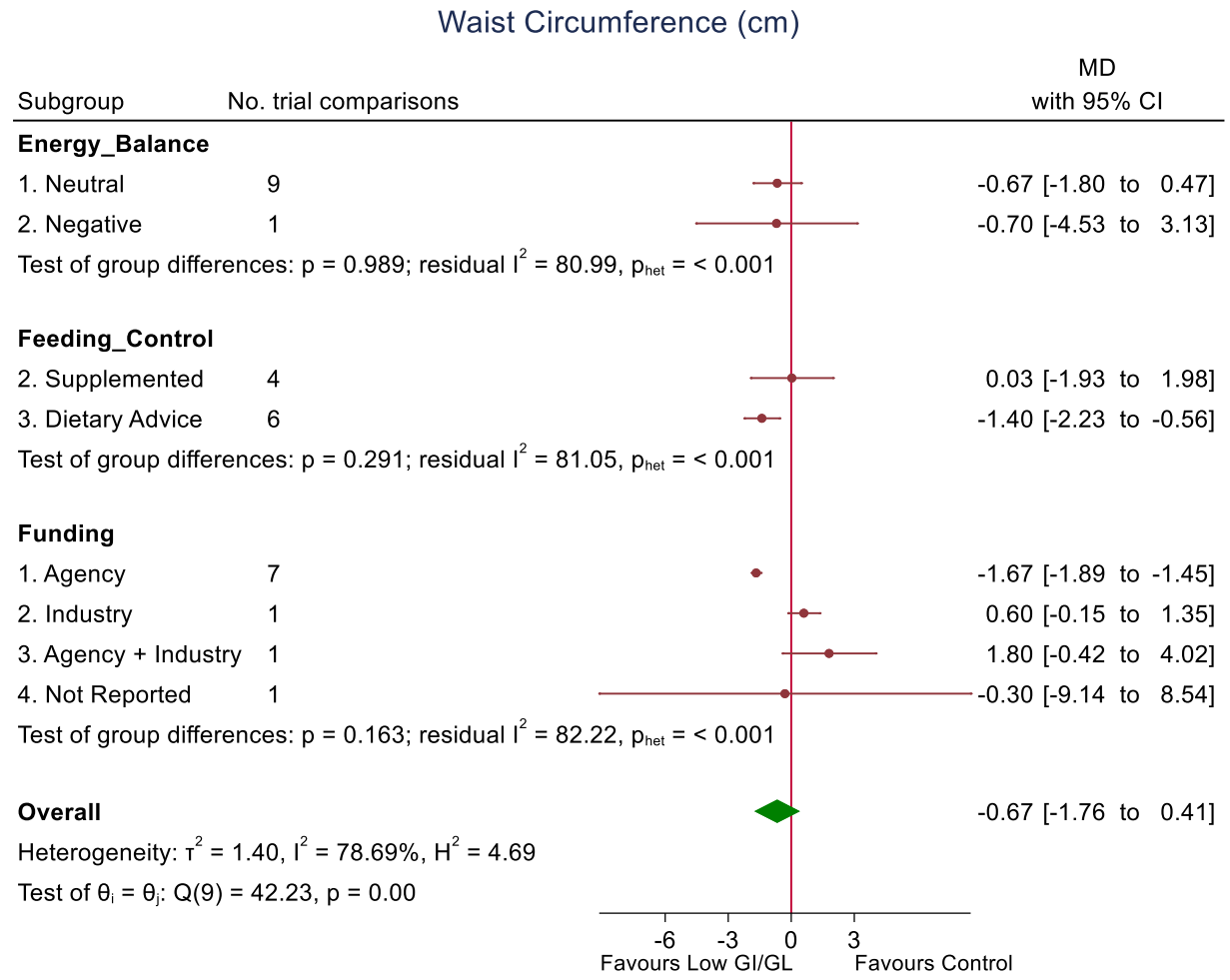
Test of θ = 0: z = -1.211, p = 0.226

The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

*N=3 trial comparisons were missing data for disease duration, thus subgroup analyses were not performed (<10 trial comparisons).

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference; Std DM diet, standard diabetes diet; T2DM, type 2 diabetes; y, years

Supplemental Figure S49 (2 of 2): Subgroup analyses for the effect of low-GI/GL dietary patterns on waist circumference (cm) in diabetes*

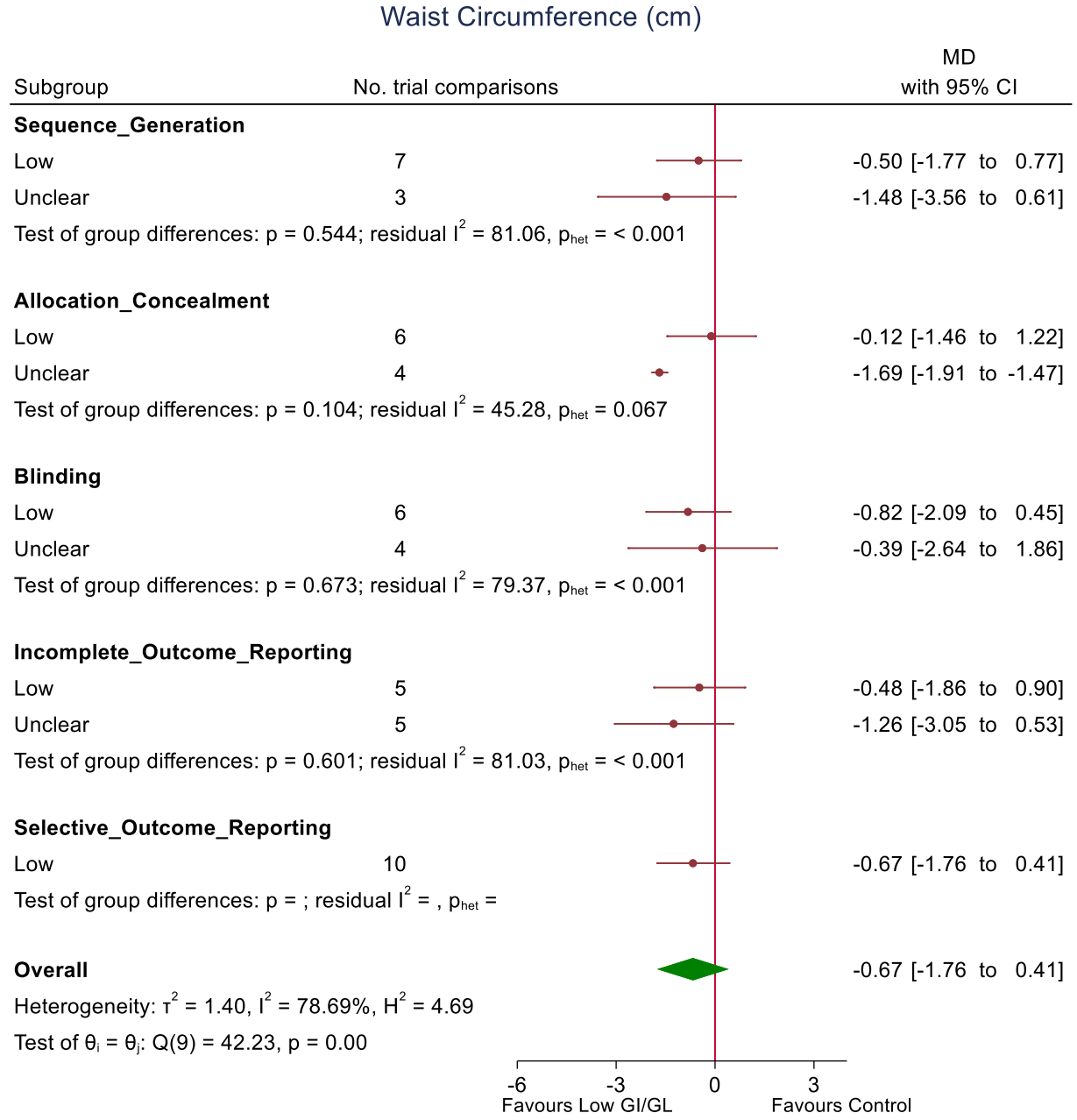


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

* Subgroup analyses were not conducted on either absolute Test GI, Test GL, Diff in GI or Diff in GL due to <10 trial comparisons (n=8 trials for each).

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; MD, mean difference; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S50: Risk of bias (using The Cochrane Collaboration Tool) subgroup analysis for the effect of low-GI/GL dietary patterns on waist circumference (cm) in diabetes



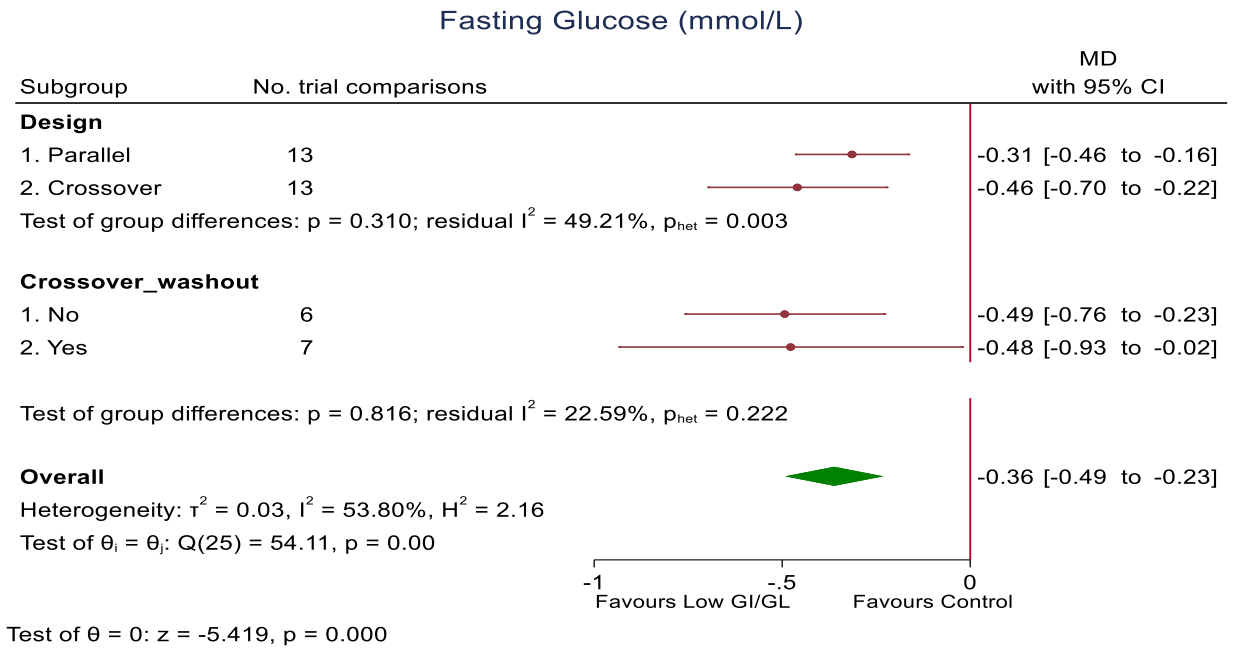
The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic,

with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup. Note: none of the trial comparisons had a High risk of bias rating and Blinding represented blinding of participants, personnel, and outcome assessors.

CI, confidence interval; GI, glycemic index; GL, glycemic load; MD, mean difference

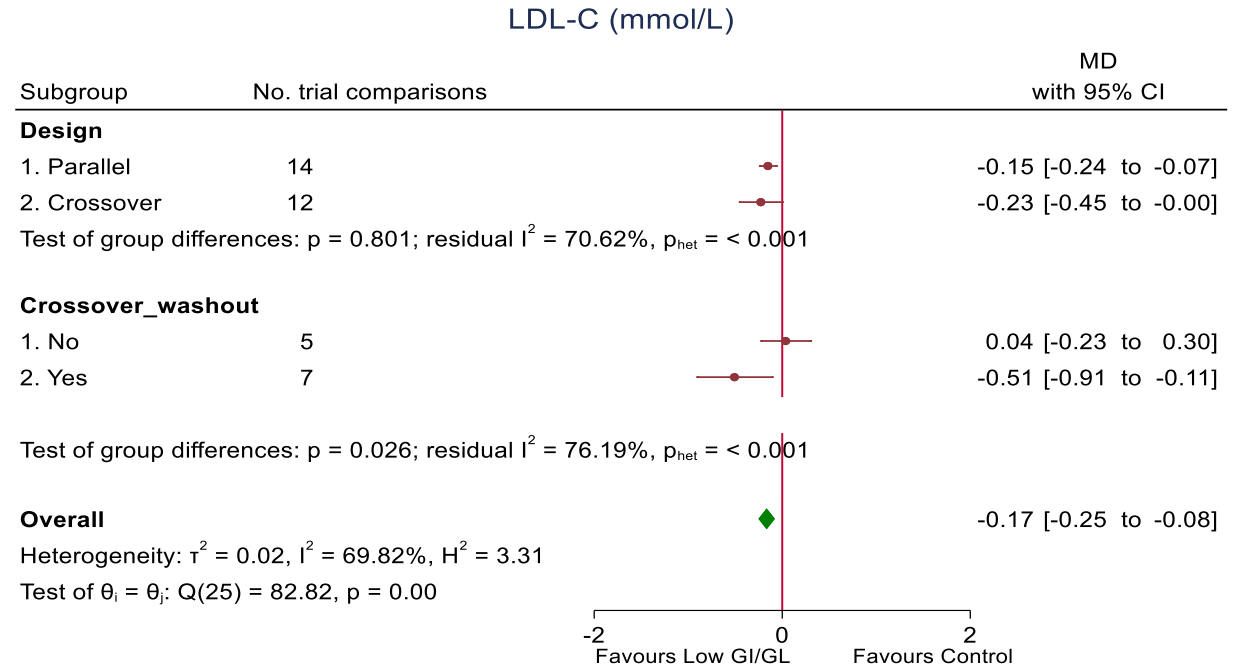
Supplemental Figure S51: Post-hoc subgroup analyses for the effect of low-GI/GL dietary patterns on cardiometabolic risk factors in diabetes by presence of a wash-out period in crossover trials[¥]

A



Test of θ = 0: z = -5.419, p = 0.000

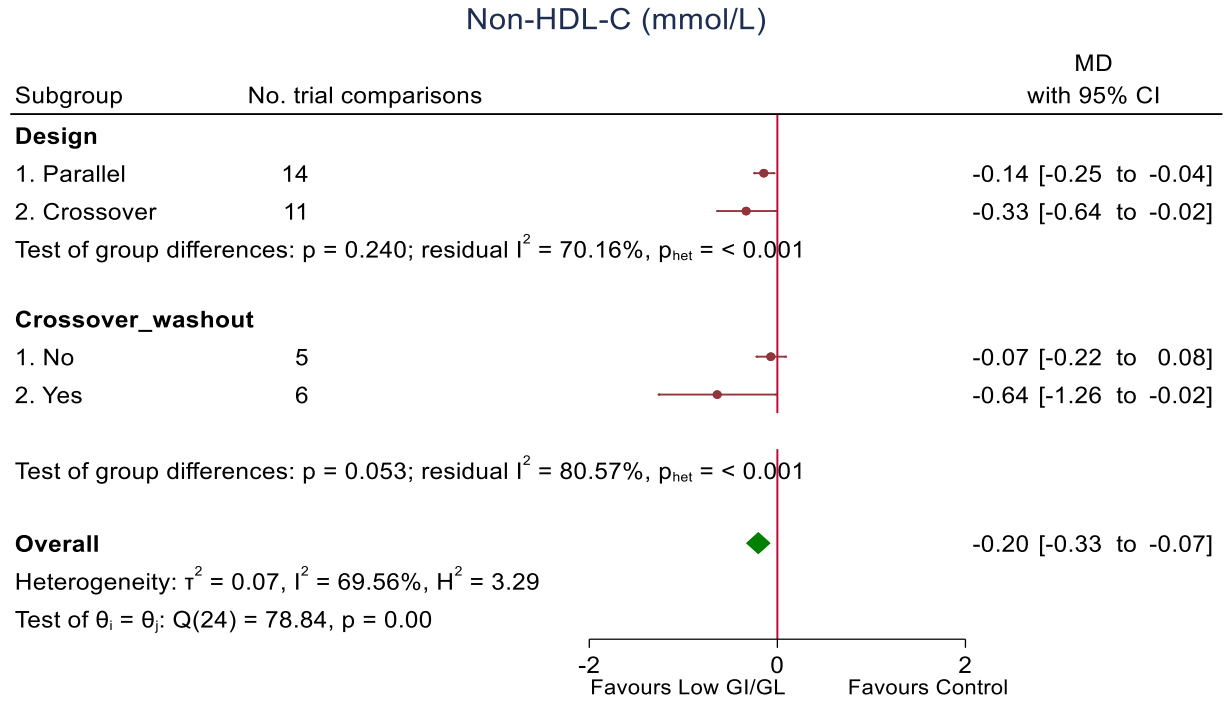
B



Test of θ = 0: z = -3.72, p = 0.00

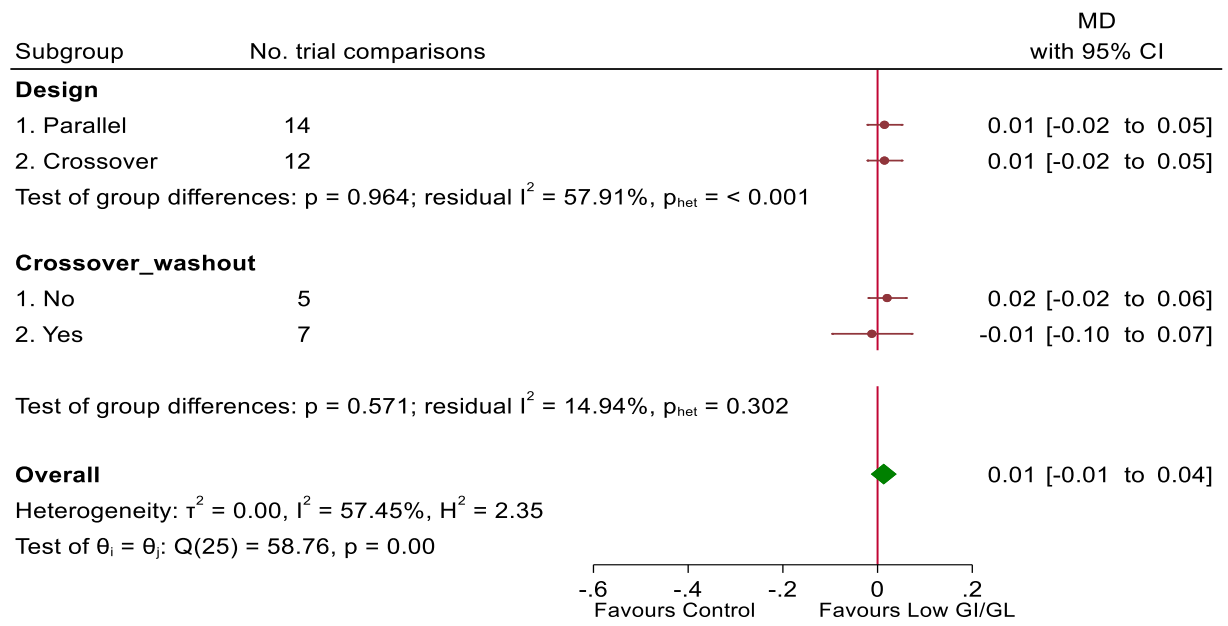
[a]Pairwise between-subgroup mean differences in LDL-C (95% CIs) for Crossover_washout were as follows: -0.53mmol/L (-1.00, -0.06) (1 vs. 2).

C



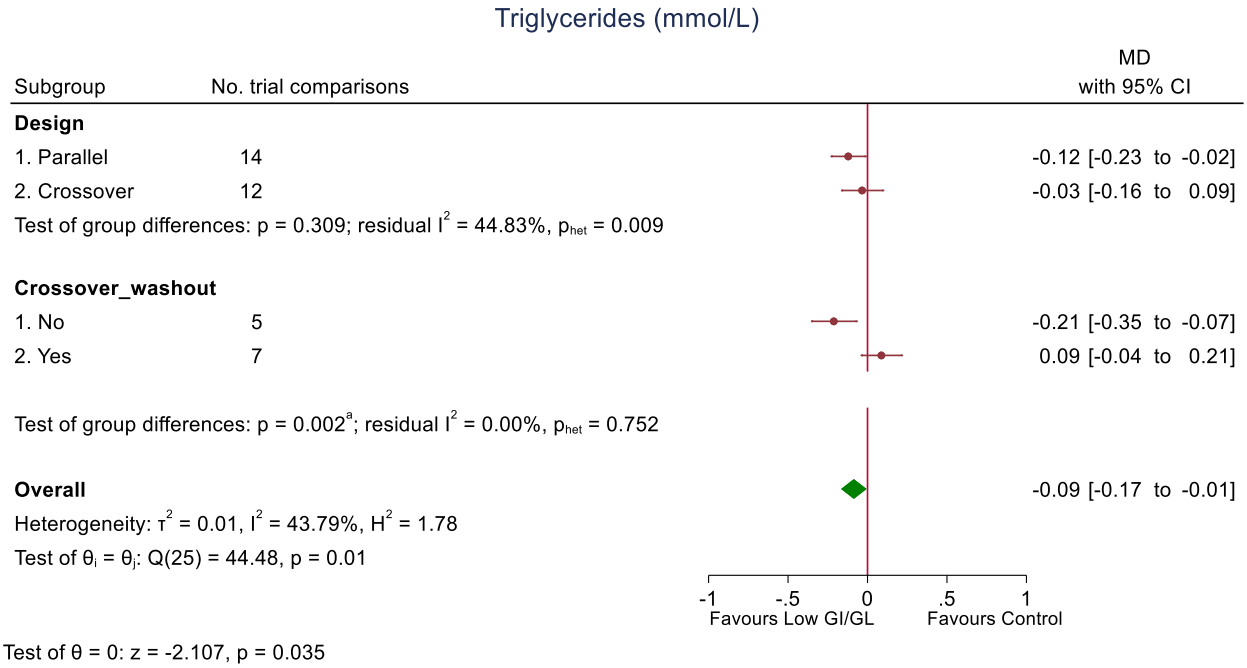
Test of θ = 0: z = -3.071, p = 0.002

D



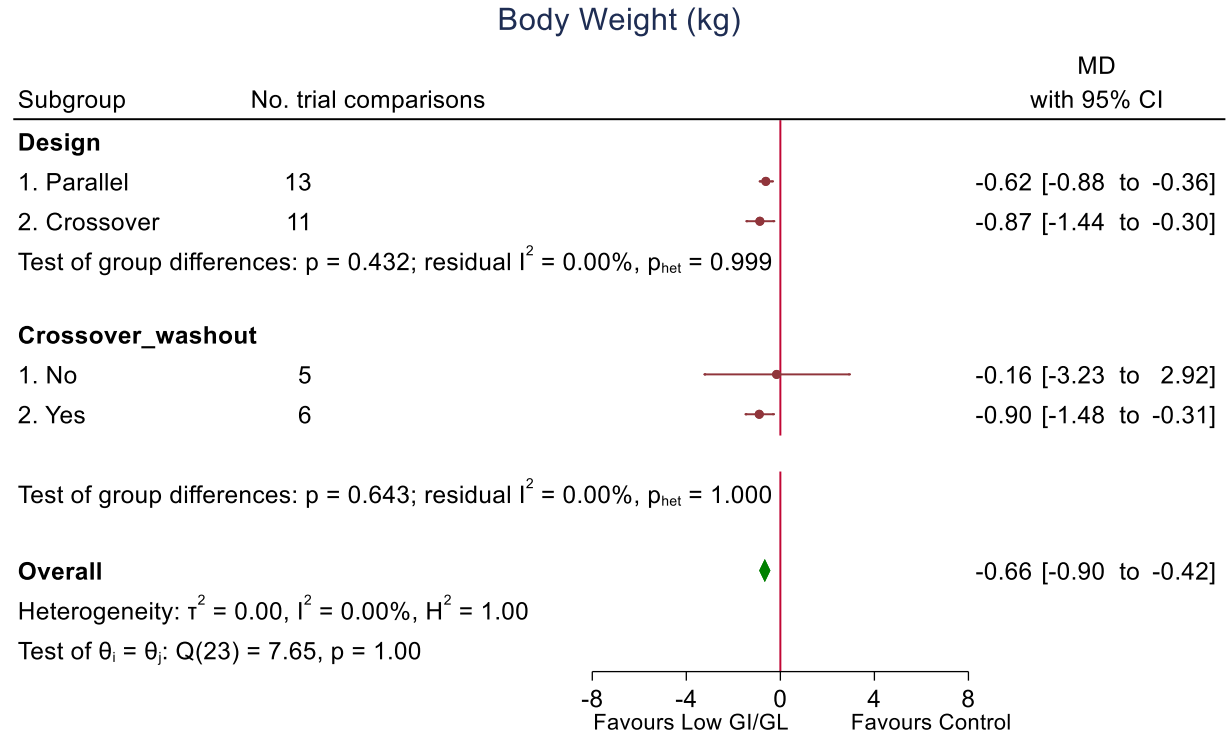
Test of θ = 0: z = 0.933, p = 0.351

E



[a]Pairwise between-subgroup mean differences in triglycerides (95% CIs) for Crossover_washout were as follows: 0.23mmol/L (0.11, 0.49) (1 vs. 2).

F

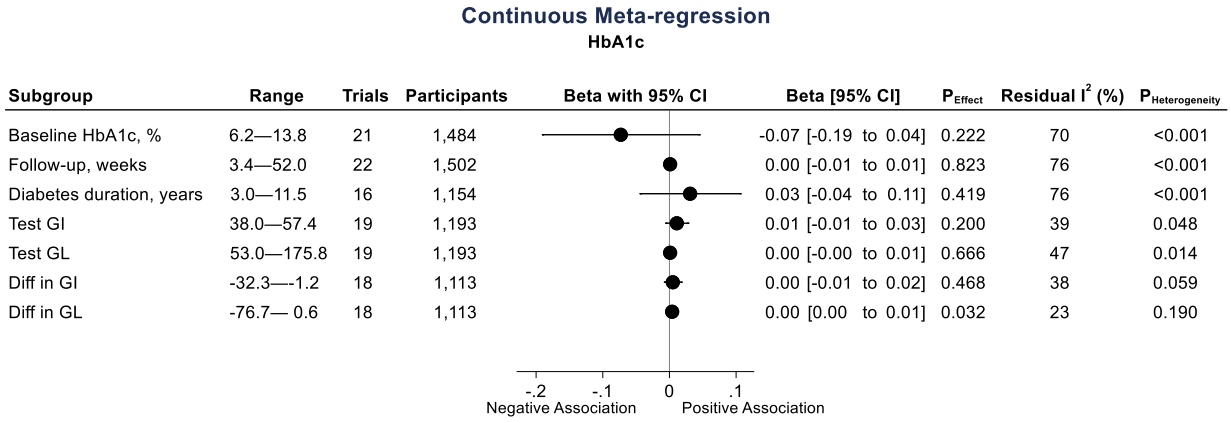


The green diamond represents the pooled estimate for the overall primary analysis of low-GI/GL diets on cardiometabolic outcomes: A, fasting glucose; B, LDL-C; C, non-HDL-C; D, HDL-C; E, Triglycerides; F, body weight. Within subgroup mean differences are the pooled effect estimates represented by a red circle. 95% CIs are represented by the line through the circle. Data are expressed as mean differences with 95% CIs using the generic inverse-variance method and random effects DerSimonian-Laird model. Inter-study heterogeneity was assessed using the Cochran Q statistic and quantified using the I^2 statistic, with significance set at $P<0.10$ and $I^2>50\%$ considered to be evidence of substantial heterogeneity. $P<0.05$ indicates that the effect size differed between levels of the subgroup.

[¥]Post-hoc subgroup analyses in crossover trials by presence of a washout were not conducted for HbA1c (n=8 trial comparisons which were of crossover design), fasting insulin (n=5), BMI (n=8) or waist circumference (n=0), nor apoB, SBP, DBP and CRP (<10 trial comparisons total).

BMI, body mass index; CI, confidence interval; CRP, c-reactive protein; DBP, diastolic blood pressure; GI, glycemic index; GL, glycemic load; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; MD, mean difference; non-HDL-C, non-high-density lipoprotein-cholesterol; SBP, systolic blood pressure

Supplemental Figure S52. Continuous meta-regression analysis for the effect of low-GI/GL dietary patterns on HbA1c (%) in diabetes*

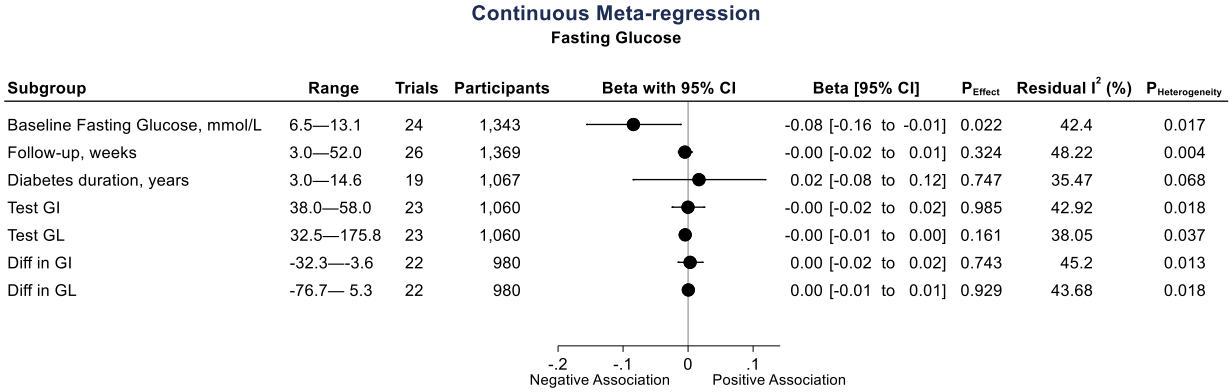


Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* N=1 trial comparison did not report baseline HbA1c value, 6 trial comparisons did not report baseline diabetes duration, 3 trial comparisons did not provide data for the absolute Test GI and GL of the diets and 4 trials did not provide data for the Diff in GI and GL between the diets.
Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; HbA1c, hemoglobin A1c; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S53. Continuous meta-regression analysis for the effect for the effect of low-GI/GL dietary patterns on fasting glucose (mmol/L) in diabetes*

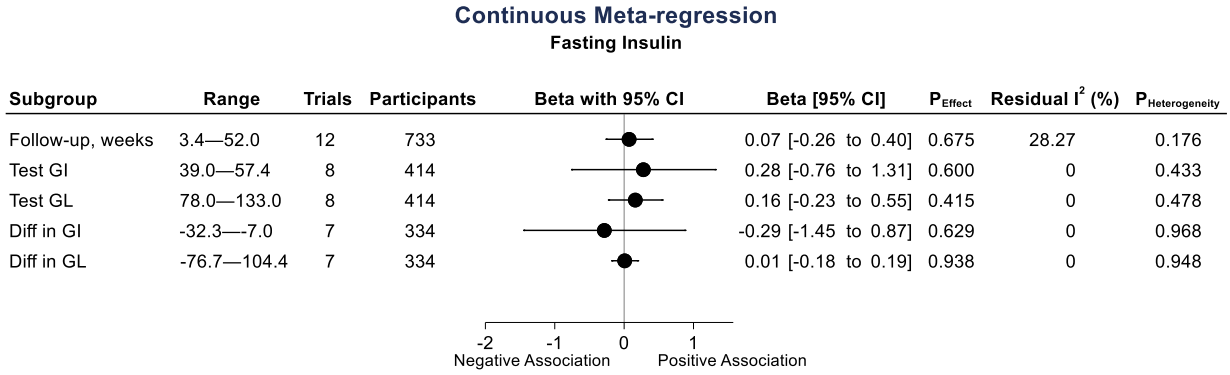


Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* N=2 trial comparisons did not report baseline fasting glucose value, 7 trial comparisons did not report baseline diabetes duration, 3 trial comparisons did not provide data for the absolute Test GI and GL of the diets and 4 trial comparisons did not provide data for the Diff in GI and GL between the diets. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S54. Continuous meta-regression analysis for the effect for the effect of low-GI/GL dietary patterns on fasting insulin (pmol/L) in diabetes*

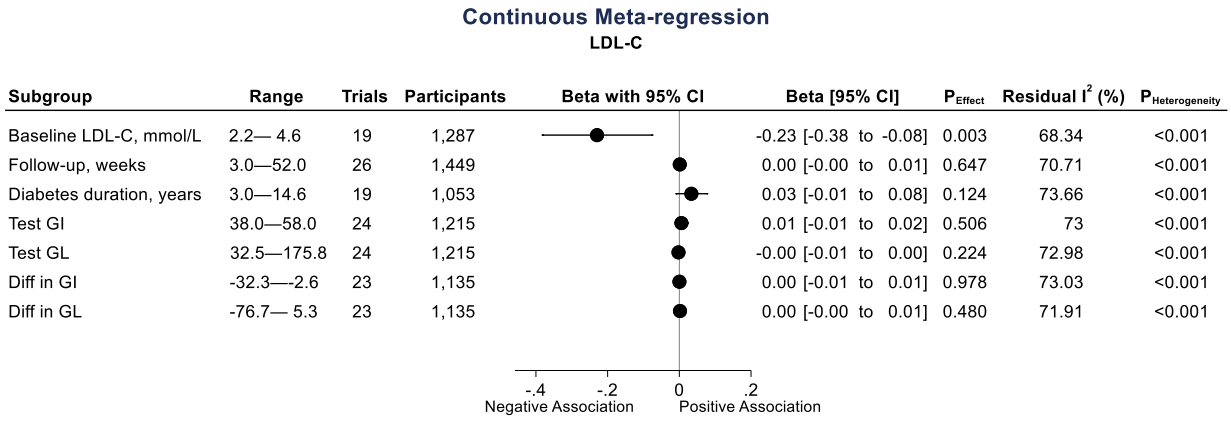


Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* Continuous subgroup analyses were not conducted on baseline insulin and baseline diabetes duration due to <10 trial comparisons (n=9 and 7, respectively). Four trial comparisons did not report the absolute Test GI and Test GL of the diets and 5 trial comparisons did not report data for the Diff in GI and Diff in GL between the diets. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S55. Continuous meta-regression analysis for the effect for the effect of low-GI/GL dietary patterns on LDL-C (mmol/L) in diabetes *

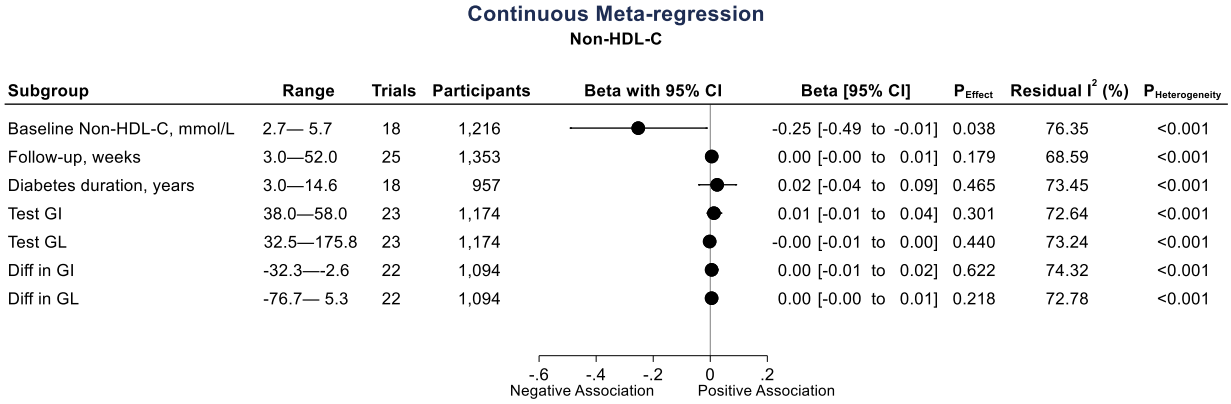


Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* N=7 trial comparisons did not report baseline LDL-C value, 7 trial comparisons did not report baseline diabetes duration, 3 trial comparisons did not provide data for the absolute Test GI and GL of the diets and 3 trial comparisons did not provide data for the Diff in GI and GL between the diets. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; LDL-C, low-density lipoprotein-cholesterol; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S56. Continuous meta-regression analysis for the effect for the effect of low-GI/GL dietary patterns on non-HDL-C (mmol/L) in diabetes*

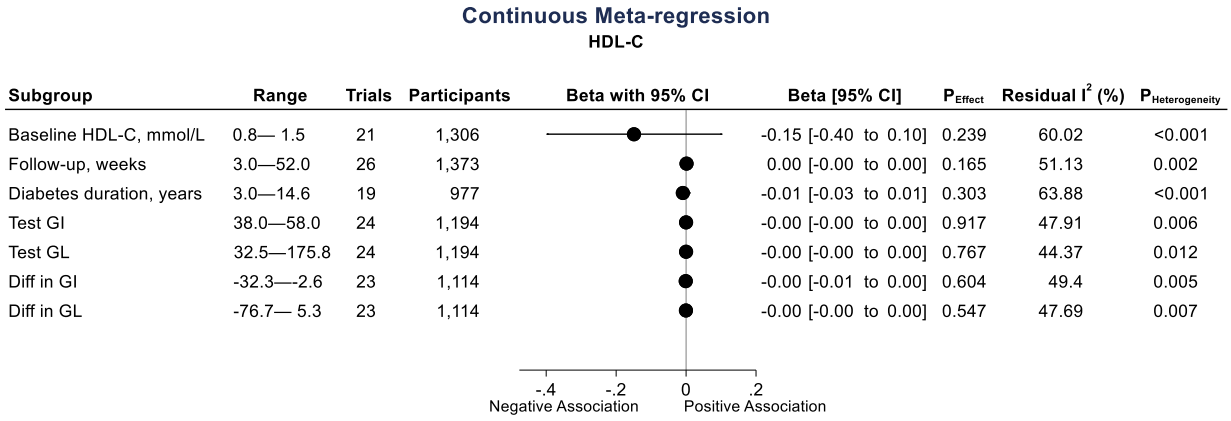


Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* N=7 trial comparisons did not report baseline Non-HDL-C value, 7 trial comparisons did not report baseline diabetes duration, 2 trial comparisons did not provide data for the absolute Test GI and GL of the diets and 3 trial comparisons did not provide data for the Diff in GI and GL between the diets. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; Non-HDL-C, non-high-density lipoprotein-cholesterol; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S57. Continuous meta-regression analysis for the effect for the effect of low-GI/GL dietary patterns on HDL-C (mmol/L) in diabetes*

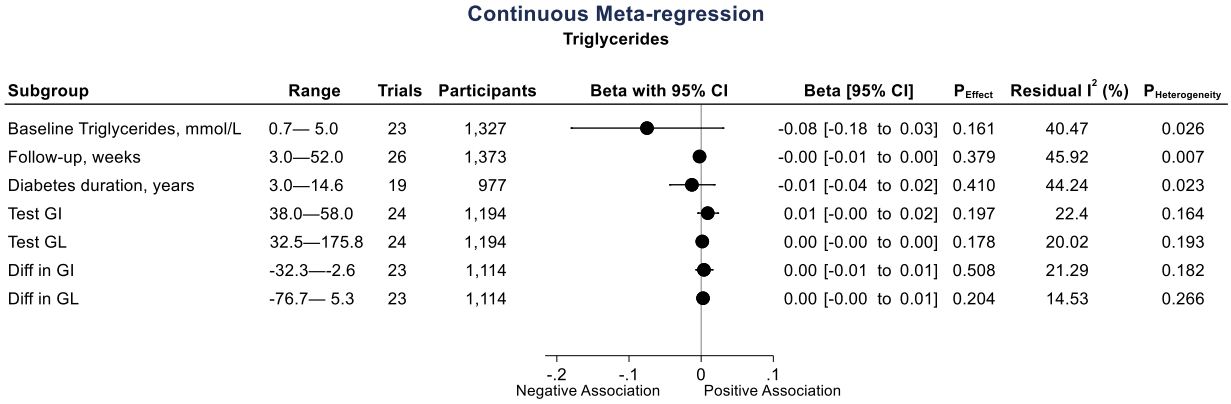


Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* N=5 trial comparisons did not report baseline HDL-C value, 7 trial comparisons did not report baseline diabetes duration, 2 trial comparisons did not provide data for the absolute Test GI and GL of the diets and 3 trial comparisons did not provide data for the Diff in GI and GL between diets. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; GI, glycemic index; GL, glycemic load; HDL-C, high-density lipoprotein-cholesterol; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S58. Continuous meta-regression analysis for the effect for the effect of low-GI/GL dietary patterns on triglycerides (mmol/L) in diabetes*

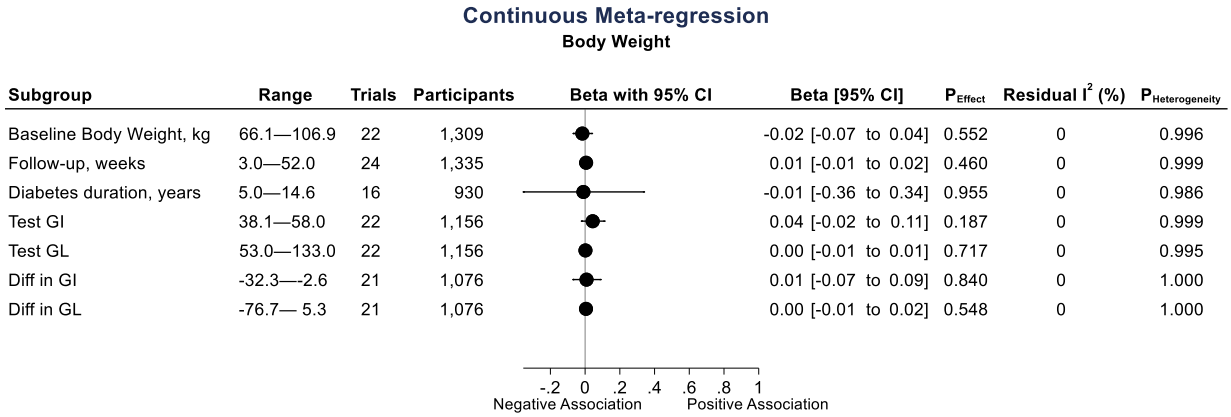


Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* N=3 trial comparisons did not report baseline triglyceride value, 7 trial comparisons did not report baseline diabetes duration, 2 trial comparisons did not provide data for the absolute Test GI and GL of the diets and 3 trial comparisons did not provide data for the Diff in GI and GL between diets. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; GI, glycemic index; GL, glycemic load; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S59. Continuous meta-regression analysis for the effect for the effect of low-GI/GL dietary patterns on body weight (kg) in diabetes*

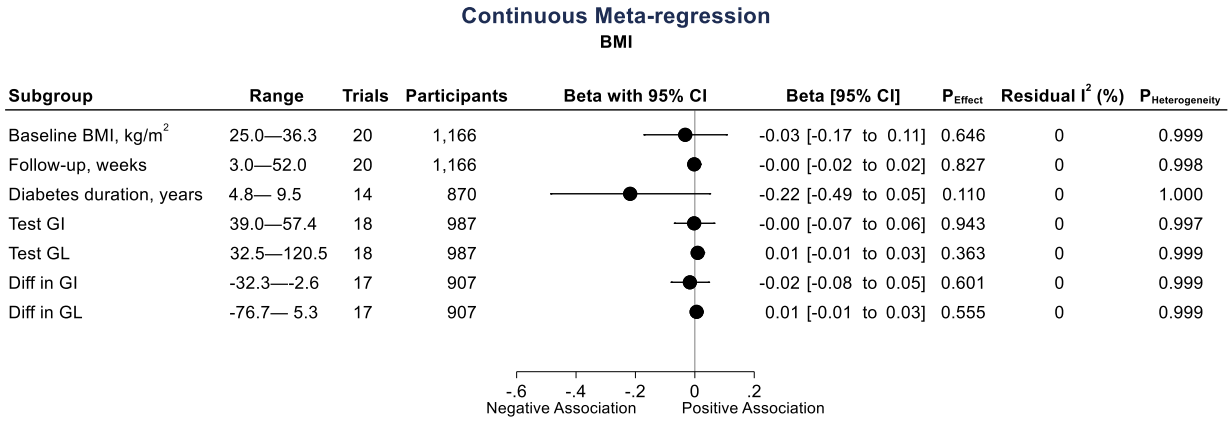


Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* N=2 trial comparisons did not report baseline body weight value, 8 trial comparisons did not report baseline diabetes duration, 2 trial comparisons did not provide data for the absolute Test GI and GL of the diets and 3 trial comparisons did not provide data for the Diff in GI and GL between the diets. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

CI, confidence interval; GI, glycemic index; GL, glycemic load; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S60. Continuous meta-regression analysis for the effect for the effect of low-GI/GL dietary patterns on BMI (kg/m²) in diabetes*

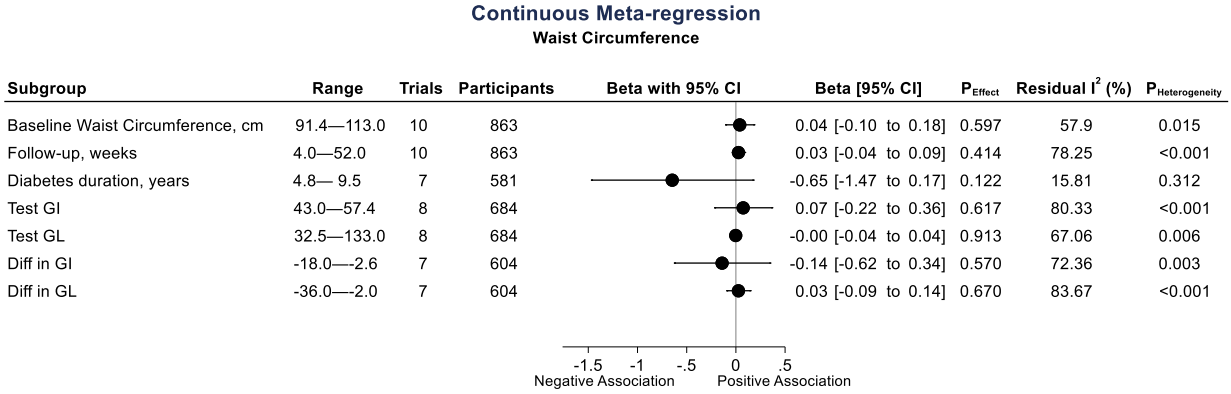


Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* N=6 trial comparisons did not report baseline diabetes duration, 2 trial comparisons did not provide data for the absolute Test GI and GL of the diets and 3 trial comparisons did not provide data for the Diff in GI and GL between the diets. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

BMI, body mass index; CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S61. Continuous meta-regression analysis for the effect for the effect of low-GI/GL dietary patterns on waist circumference (cm) in diabetes*



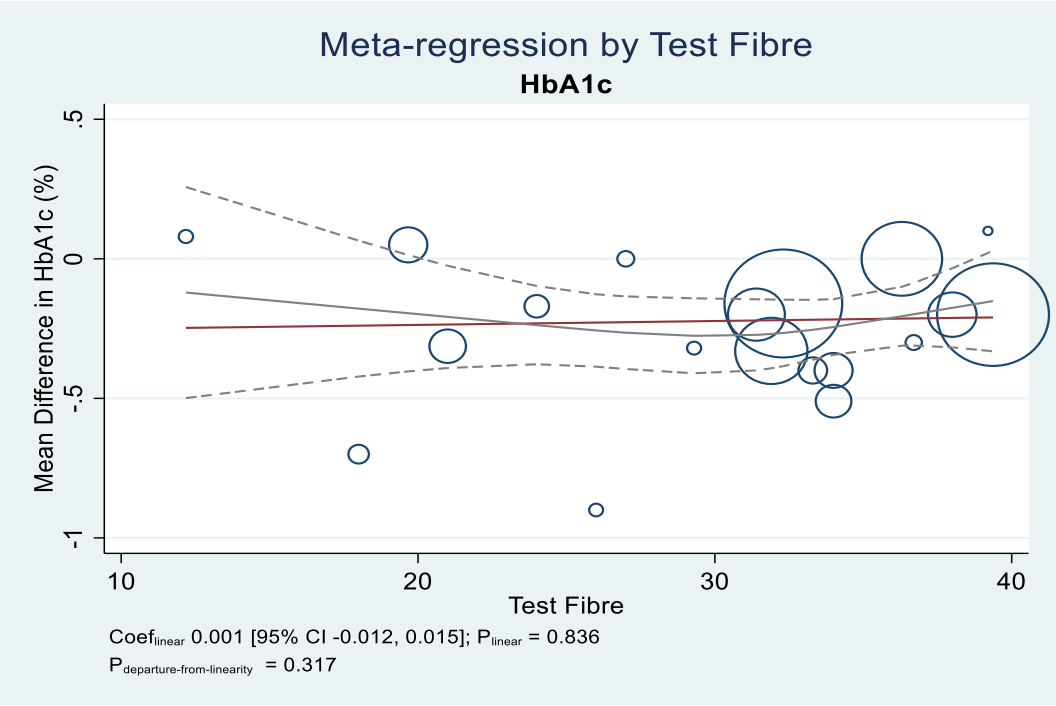
Data is presented as between group mean difference (95% CI) for a 1-unit change in the predictor variable. β –coefficients were estimated using continuous meta-regression analysis. A positive β -coefficient implies an increase in outcome on the low-GI/GL intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in outcome. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran Q statistic.

* N=3 trial comparisons did not report baseline diabetes duration, 2 trial comparisons did not report the absolute Test GI or GL of the diets and 3 trial comparisons did not report the Diff in GI and GL between diets. Note "Diff in" denotes difference in either GI or GL between the test and control groups (test – control) during the interventions.

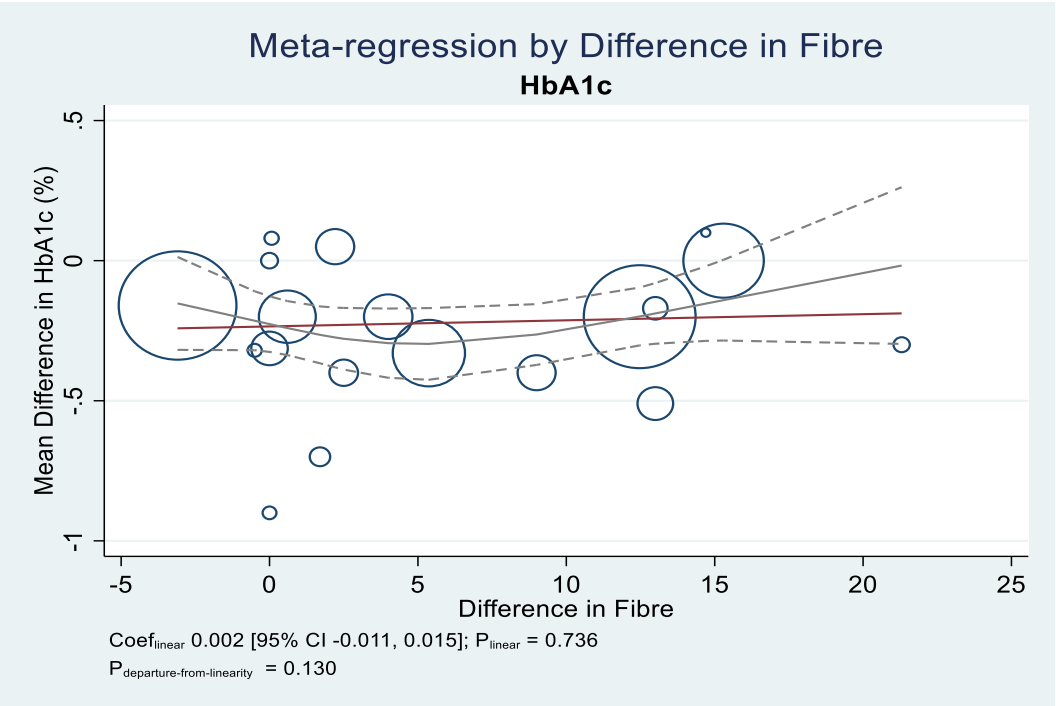
CI, confidence interval; Diff, difference; GI, glycemic index; GL, glycemic load; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S62: Post-hoc linear and non-linear meta-regression analyses for the effect of low-GI and GL by dietary fibre (as absolute test fibre and as difference in fibre)*

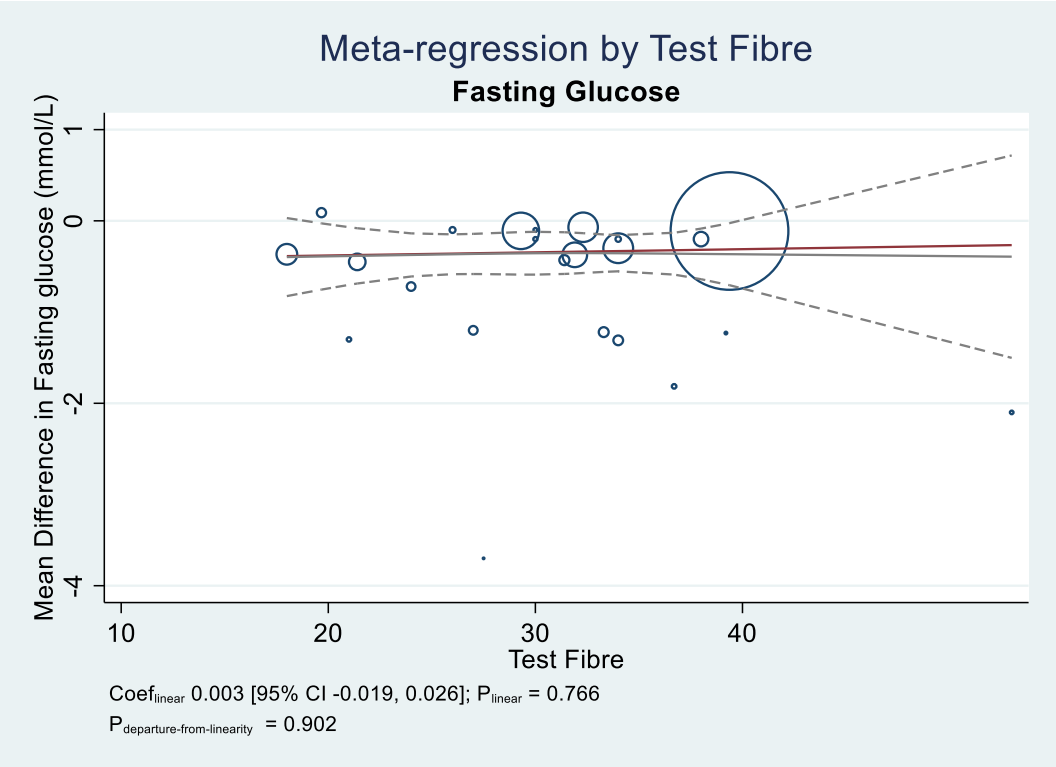
A



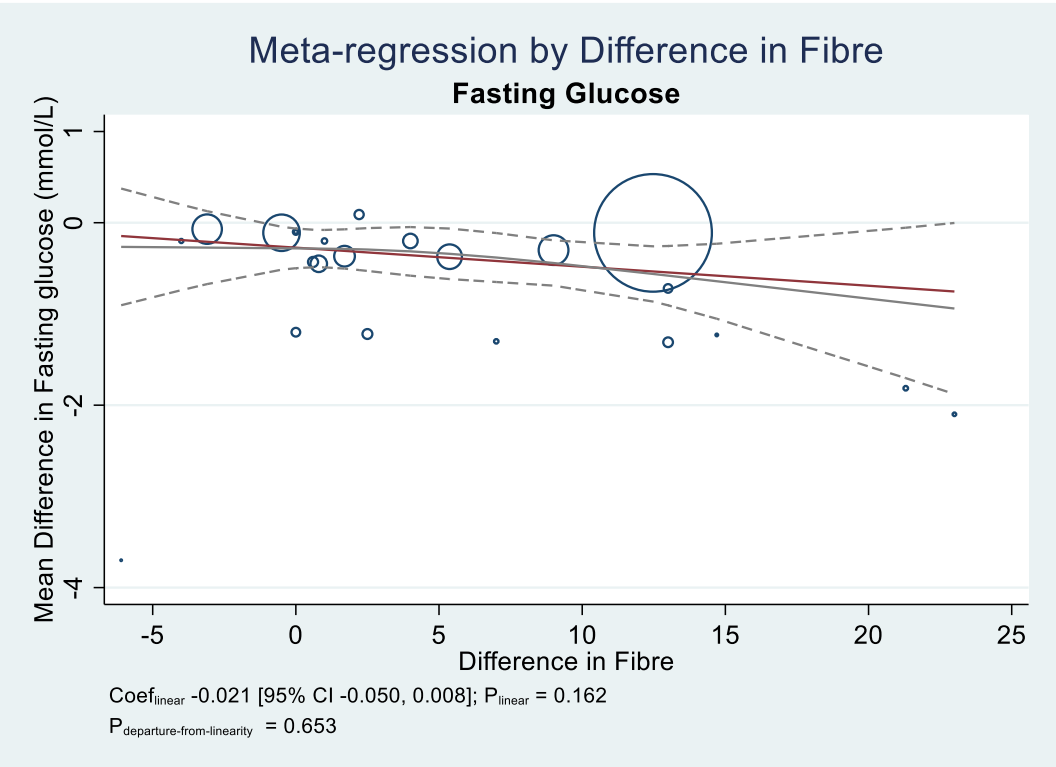
B



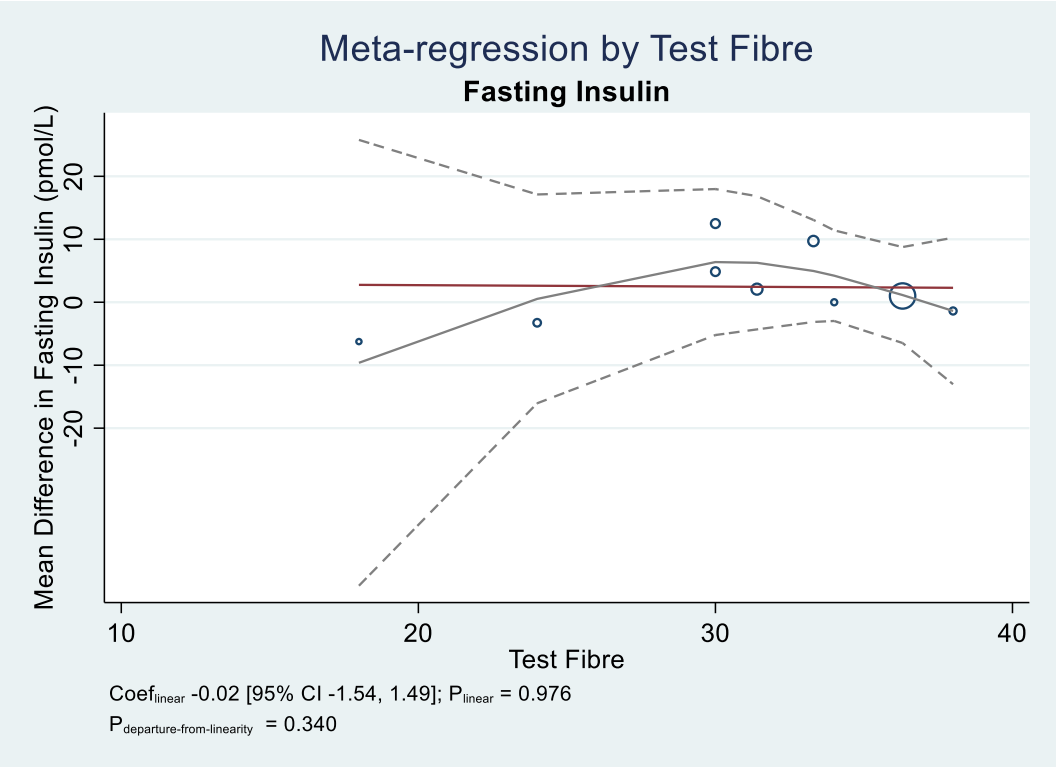
C


Meta-regression by Test Fibre
Fasting Glucose
Mean Difference in Fasting glucose (mmol/L)
1
0
-2
-4
10
20
30
40
Test Fibre
Coef$_{linear}$ 0.003 [95% CI -0.019, 0.026]; P$_{linear}$ = 0.766
P$_{departure\text{-}from\text{-}linearity}$ = 0.902

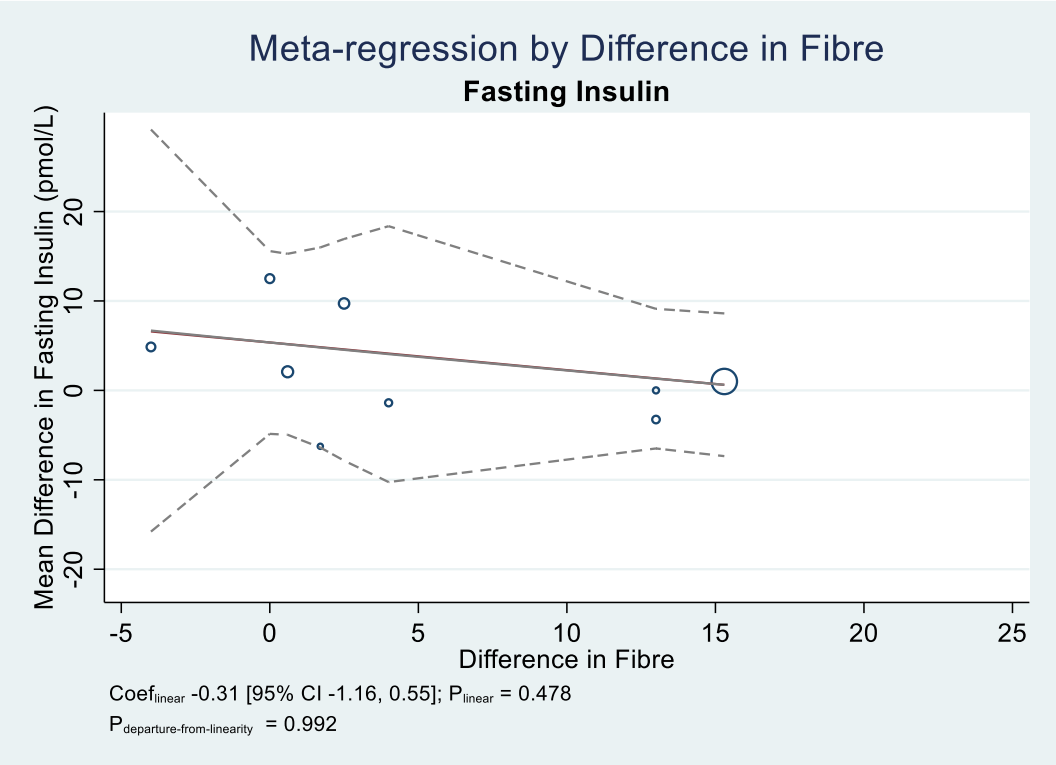
D


Meta-regression by Difference in Fibre
Fasting Glucose
Mean Difference in Fasting glucose (mmol/L)
1
0
-2
-4
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ -0.021 [95% CI -0.050, 0.008]; P$_{linear}$ = 0.162
P$_{departure\text{-}from\text{-}linearity}$ = 0.653

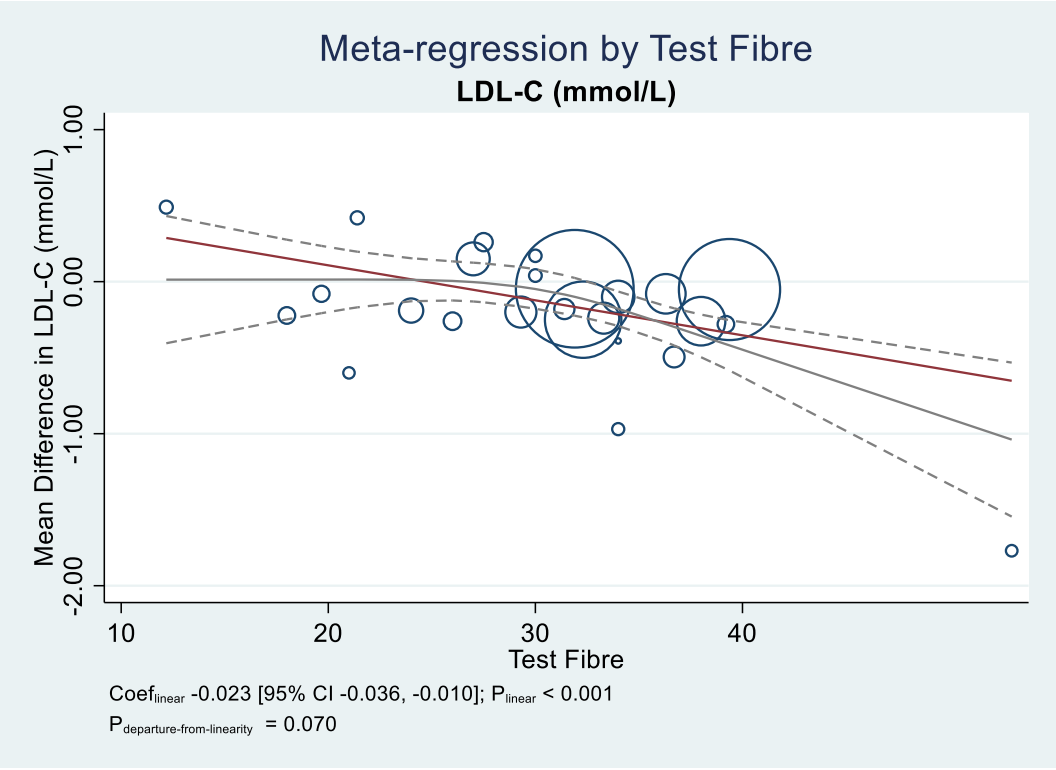
E


Meta-regression by Test Fibre
Fasting Insulin
Mean Difference in Fasting Insulin (pmol/L)
-20
-10
0
10
20
10
20
30
40
Test Fibre
Coef$_{linear}$ -0.02 [95% CI -1.54, 1.49]; P$_{linear}$ = 0.976
P$_{departure\text{-}from\text{-}linearity}$ = 0.340

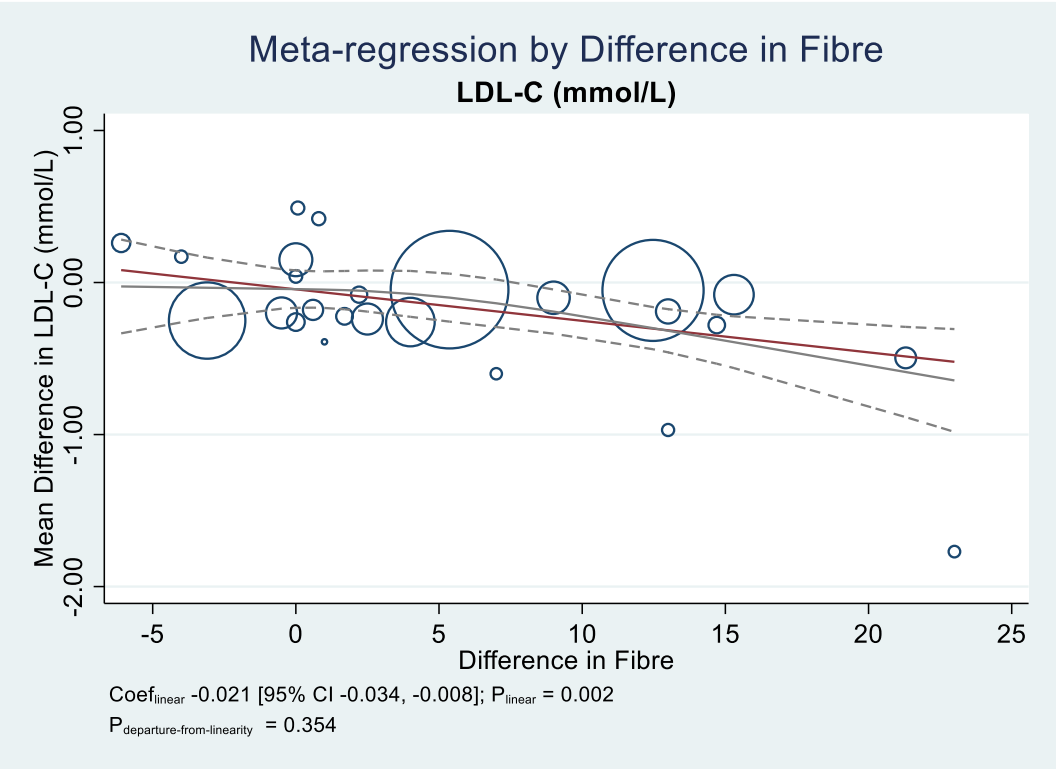
F


Meta-regression by Difference in Fibre
Fasting Insulin
Mean Difference in Fasting Insulin (pmol/L)
-20
-10
0
10
20
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ -0.31 [95% CI -1.16, 0.55]; P$_{linear}$ = 0.478
P$_{departure\text{-}from\text{-}linearity}$ = 0.992

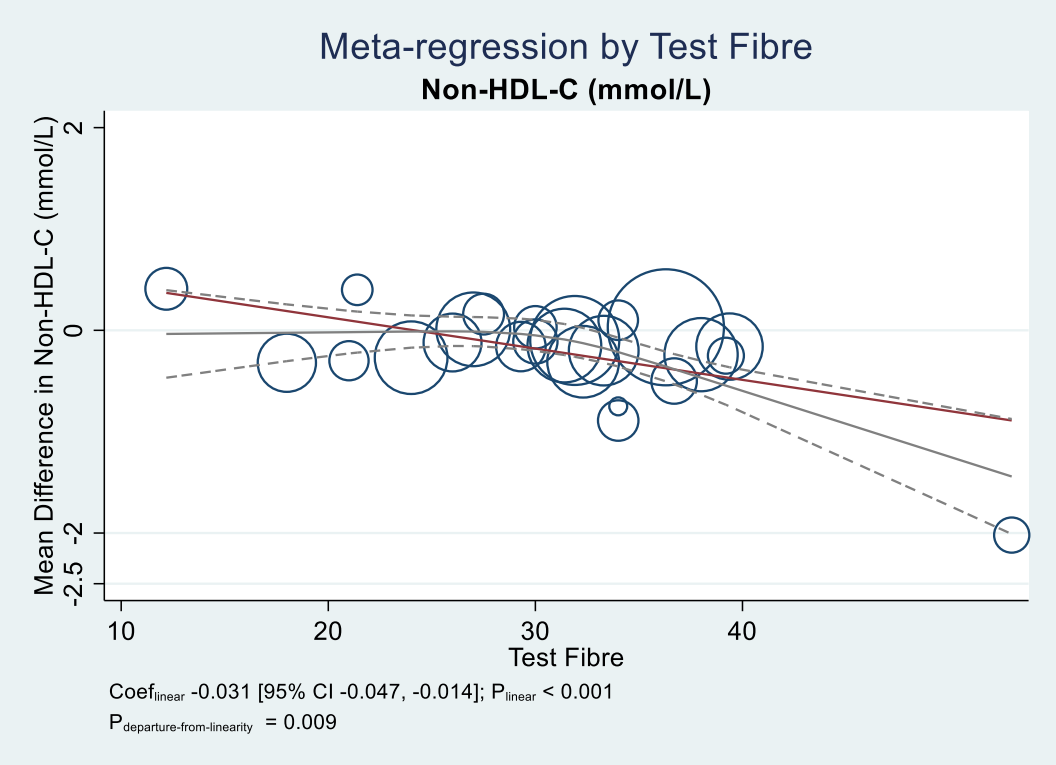
G


Meta-regression by Test Fibre
LDL-C (mmol/L)
Mean Difference in LDL-C (mmol/L)
1.00
0.00
-1.00
-2.00
10
20
30
40
Test Fibre
Coef$_{linear}$ -0.023 [95% CI -0.036, -0.010]; P$_{linear}$ < 0.001
P$_{departure\text{-}from\text{-}linearity}$ = 0.070

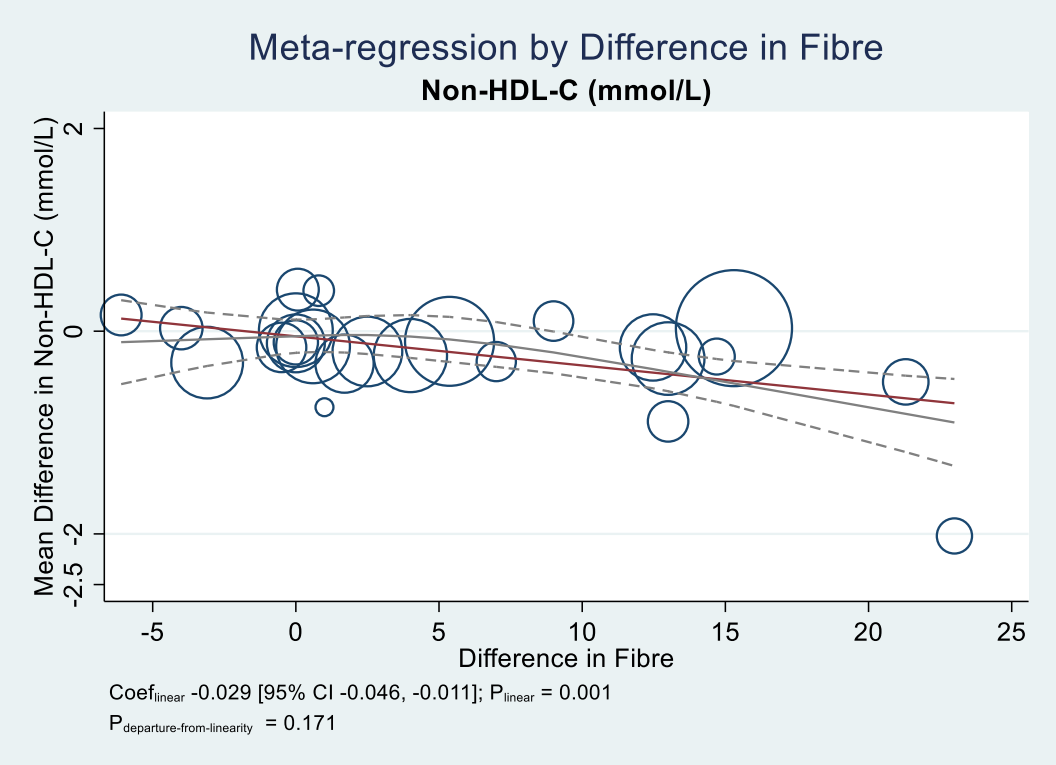
H


Meta-regression by Difference in Fibre
LDL-C (mmol/L)
Mean Difference in LDL-C (mmol/L)
1.00
0.00
-1.00
-2.00
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ -0.021 [95% CI -0.034, -0.008]; P$_{linear}$ = 0.002
P$_{departure\text{-}from\text{-}linearity}$ = 0.354

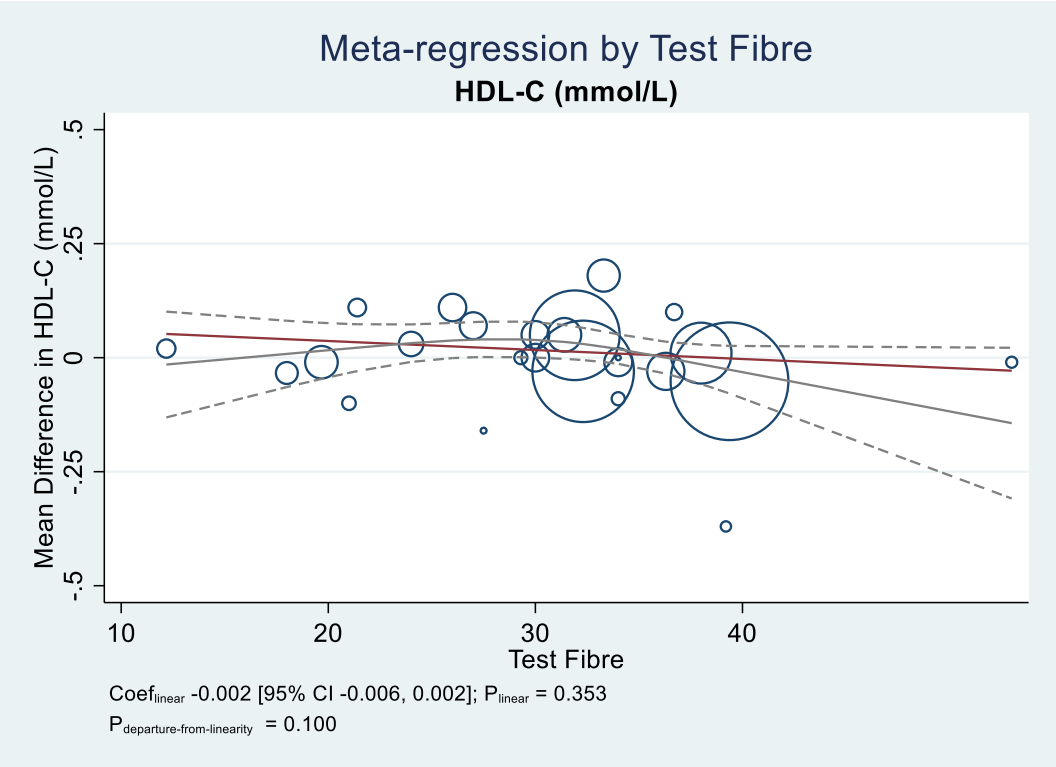
I


Meta-regression by Test Fibre
Non-HDL-C (mmol/L)
Mean Difference in Non-HDL-C (mmol/L)
2
0
-2
-2.5
10
20
30
40
Test Fibre
Coef$_{linear}$ -0.031 [95% CI -0.047, -0.014]; P$_{linear}$ < 0.001
P$_{departure-from-linearity}$ = 0.009

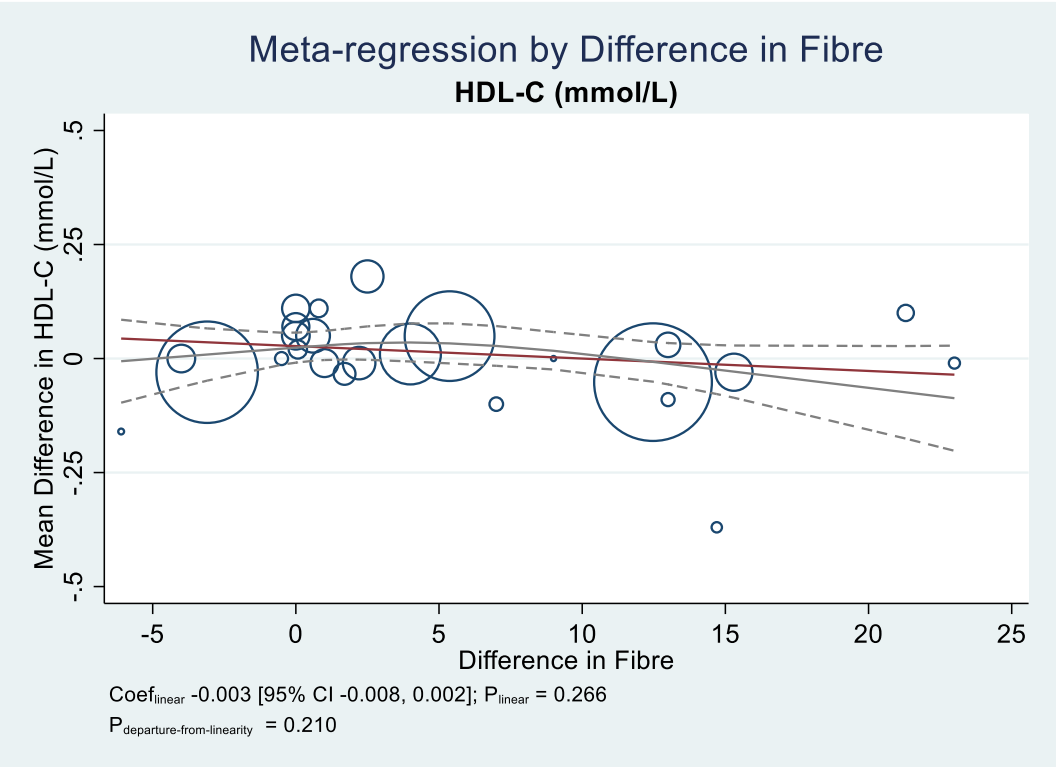
J


Meta-regression by Difference in Fibre
Non-HDL-C (mmol/L)
Mean Difference in Non-HDL-C (mmol/L)
2
0
-2
-2.5
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ -0.029 [95% CI -0.046, -0.011]; P$_{linear}$ = 0.001
P$_{departure-from-linearity}$ = 0.171

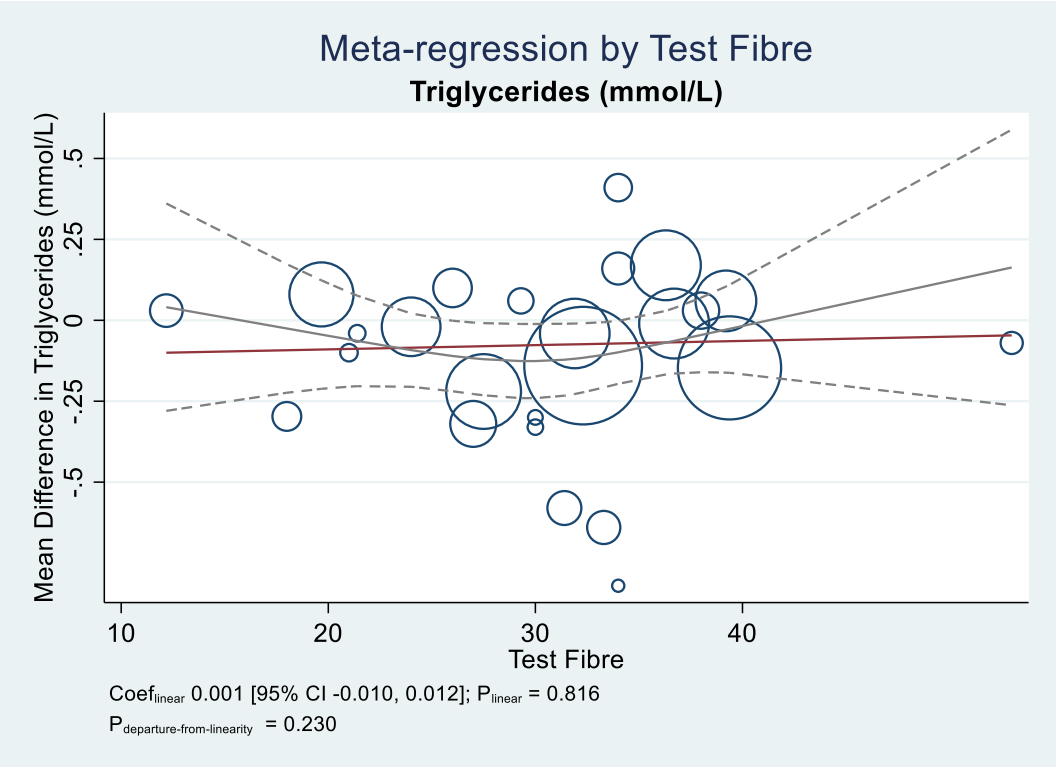
K


Meta-regression by Test Fibre
HDL-C (mmol/L)
Mean Difference in HDL-C (mmol/L)
-.5
-.25
0
.25
.5
10
20
30
40
Test Fibre
Coef$_{linear}$ -0.002 [95% CI -0.006, 0.002]; P$_{linear}$ = 0.353
P$_{departure\text{-}from\text{-}linearity}$ = 0.100

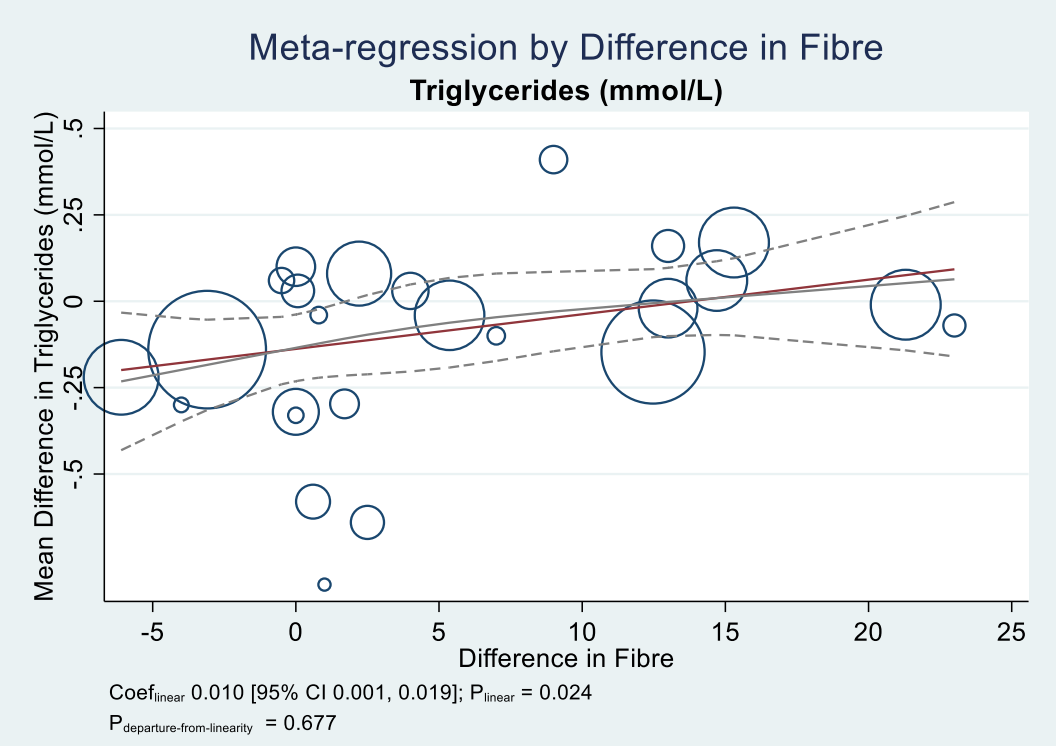
L


Meta-regression by Difference in Fibre
HDL-C (mmol/L)
Mean Difference in HDL-C (mmol/L)
-.5
-.25
0
.25
.5
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ -0.003 [95% CI -0.008, 0.002]; P$_{linear}$ = 0.266
P$_{departure\text{-}from\text{-}linearity}$ = 0.210

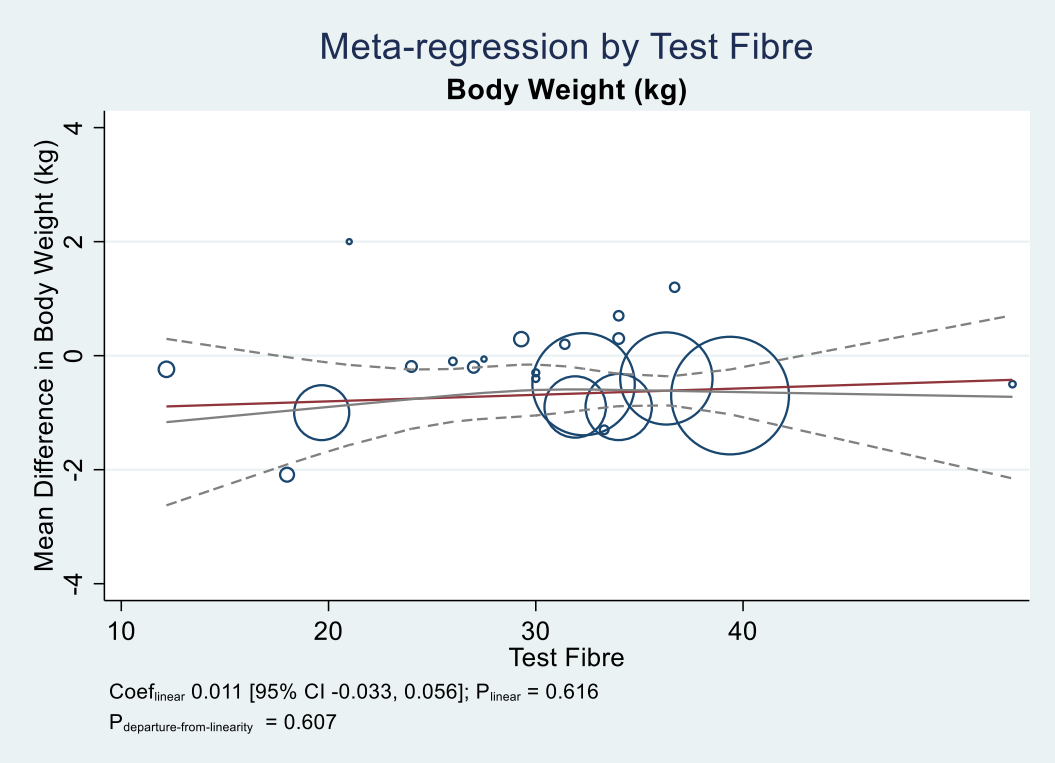
M


Meta-regression by Test Fibre
Triglycerides (mmol/L)
Mean Difference in Triglycerides (mmol/L)
-.5
-.25
0
.25
.5
10
20
30
40
Test Fibre
Coef$_{linear}$ 0.001 [95% CI -0.010, 0.012]; P$_{linear}$ = 0.816
P$_{departure\text{-}from\text{-}linearity}$ = 0.230

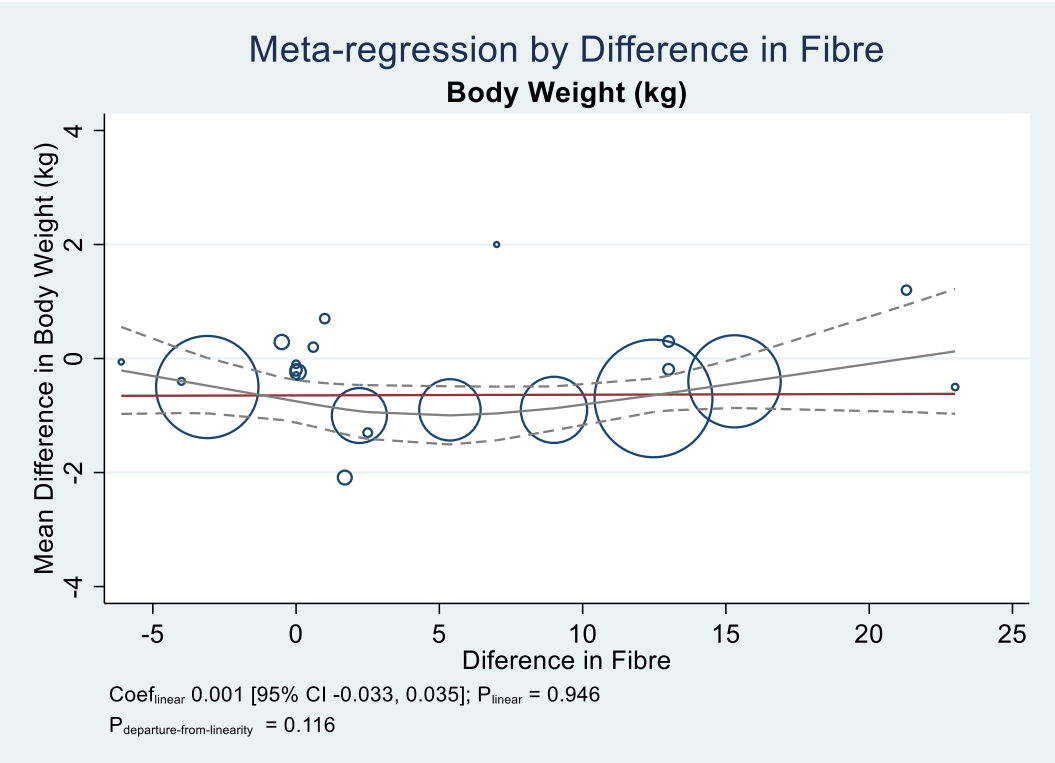
N


Meta-regression by Difference in Fibre
Triglycerides (mmol/L)
Mean Difference in Triglycerides (mmol/L)
-.5
-.25
0
.25
.5
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ 0.010 [95% CI 0.001, 0.019]; P$_{linear}$ = 0.024
P$_{departure\text{-}from\text{-}linearity}$ = 0.677

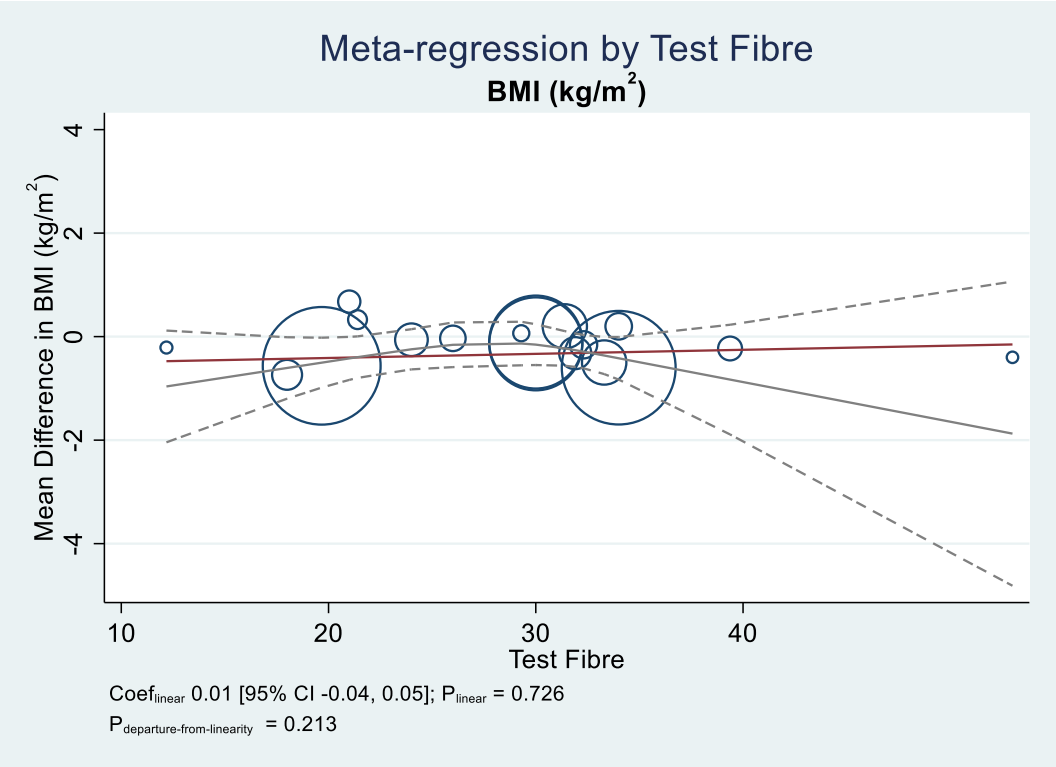
O


Meta-regression by Test Fibre
Body Weight (kg)
Mean Difference in Body Weight (kg)
-4
-2
0
2
4
10
20
30
40
Test Fibre
Coef linear 0.011 [95% CI -0.033, 0.056]; P linear = 0.616
P departure-from-linearity = 0.607

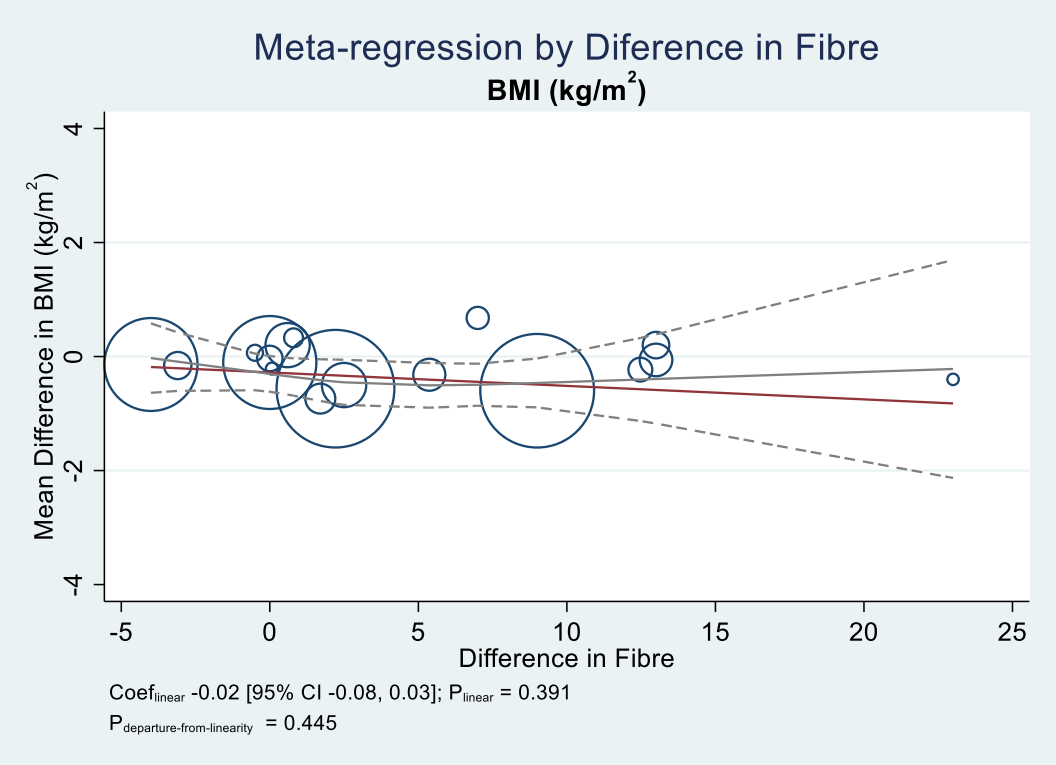
P


Meta-regression by Difference in Fibre
Body Weight (kg)
Mean Difference in Body Weight (kg)
-4
-2
0
2
4
-5
0
5
10
15
20
25
Diference in Fibre
Coef linear 0.001 [95% CI -0.033, 0.035]; P linear = 0.946
P departure-from-linearity = 0.116

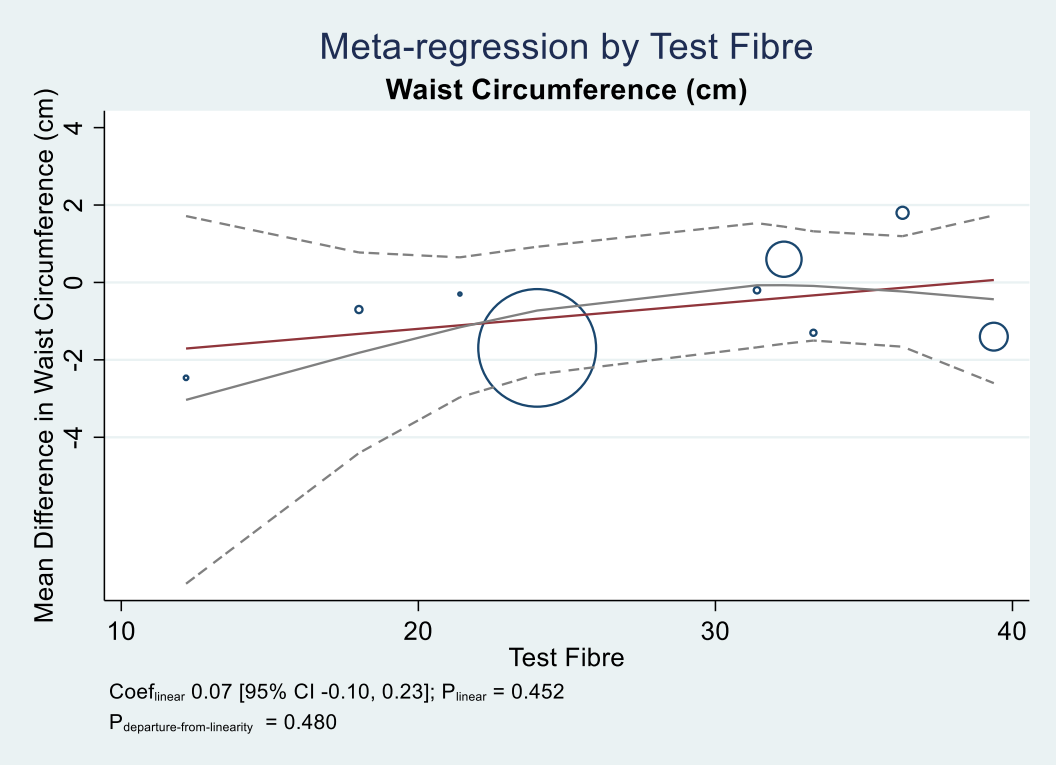
Q


Meta-regression by Test Fibre
BMI (kg/m^2)
Mean Difference in BMI (kg/m^2)
4
2
0
-2
-4
10
20
30
40
Test Fibre
Coef$_{linear}$ 0.01 [95% CI -0.04, 0.05]; P$_{linear}$ = 0.726
P$_{departure\text{-}from\text{-}linearity}$ = 0.213

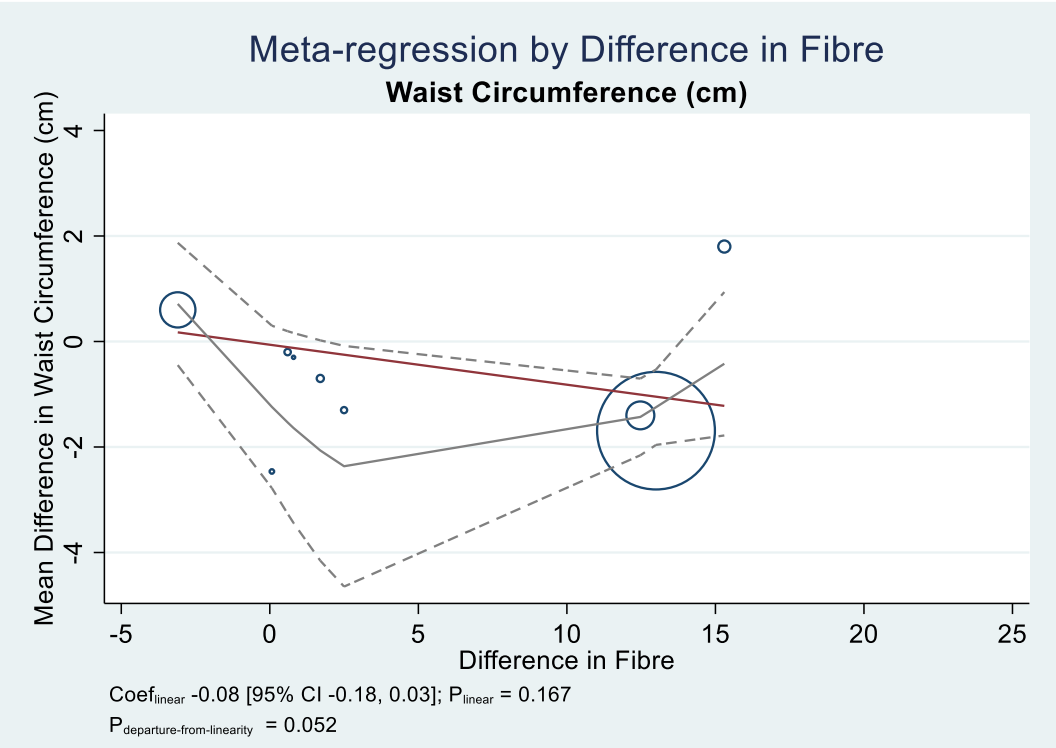
R


Meta-regression by Diference in Fibre
BMI (kg/m^2)
Mean Difference in BMI (kg/m^2)
4
2
0
-2
-4
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ -0.02 [95% CI -0.08, 0.03]; P$_{linear}$ = 0.391
P$_{departure\text{-}from\text{-}linearity}$ = 0.445

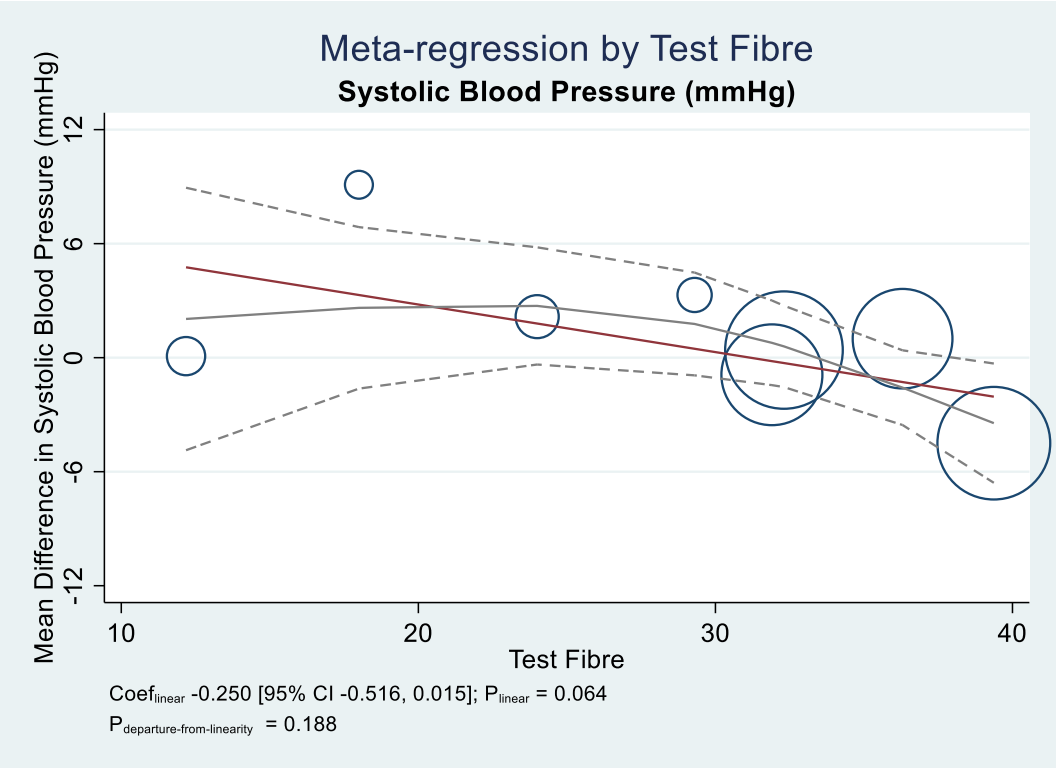
S


Meta-regression by Test Fibre
Waist Circumference (cm)
Mean Difference in Waist Circumference (cm)
-4
-2
0
2
4
10
20
30
40
Test Fibre
Coef$_{linear}$ 0.07 [95% CI -0.10, 0.23]; P$_{linear}$ = 0.452
P$_{departure-from-linearity}$ = 0.480

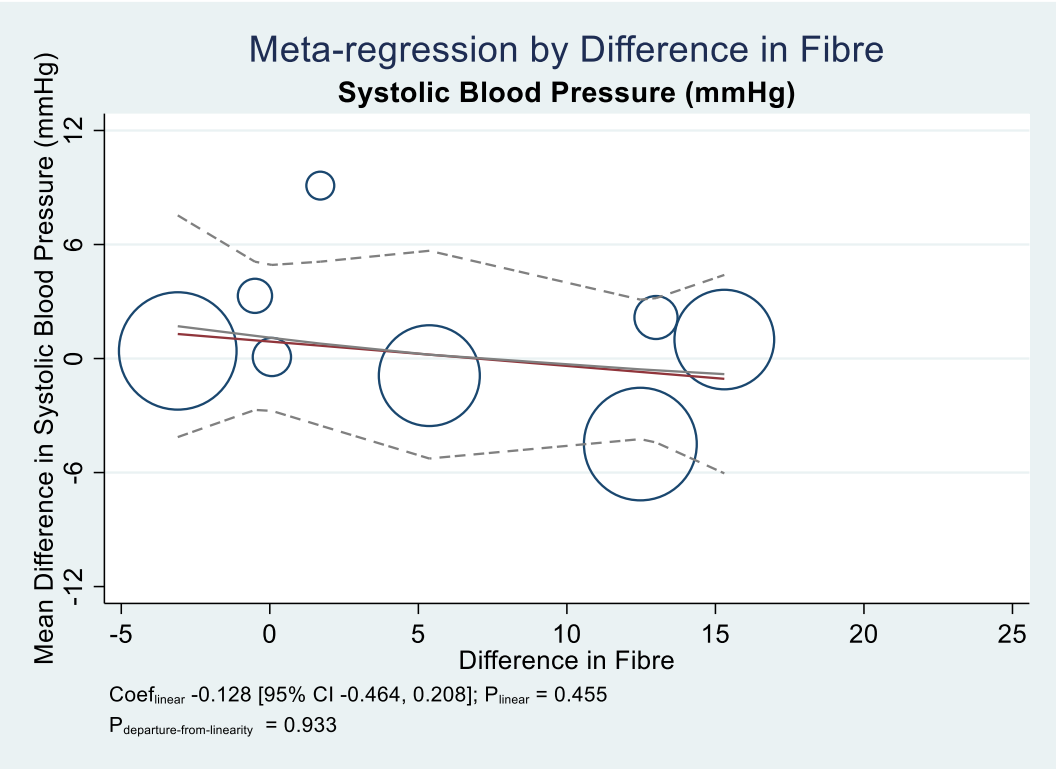
T


Meta-regression by Difference in Fibre
Waist Circumference (cm)
Mean Difference in Waist Circumference (cm)
-4
-2
0
2
4
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ -0.08 [95% CI -0.18, 0.03]; P$_{linear}$ = 0.167
P$_{departure-from-linearity}$ = 0.052

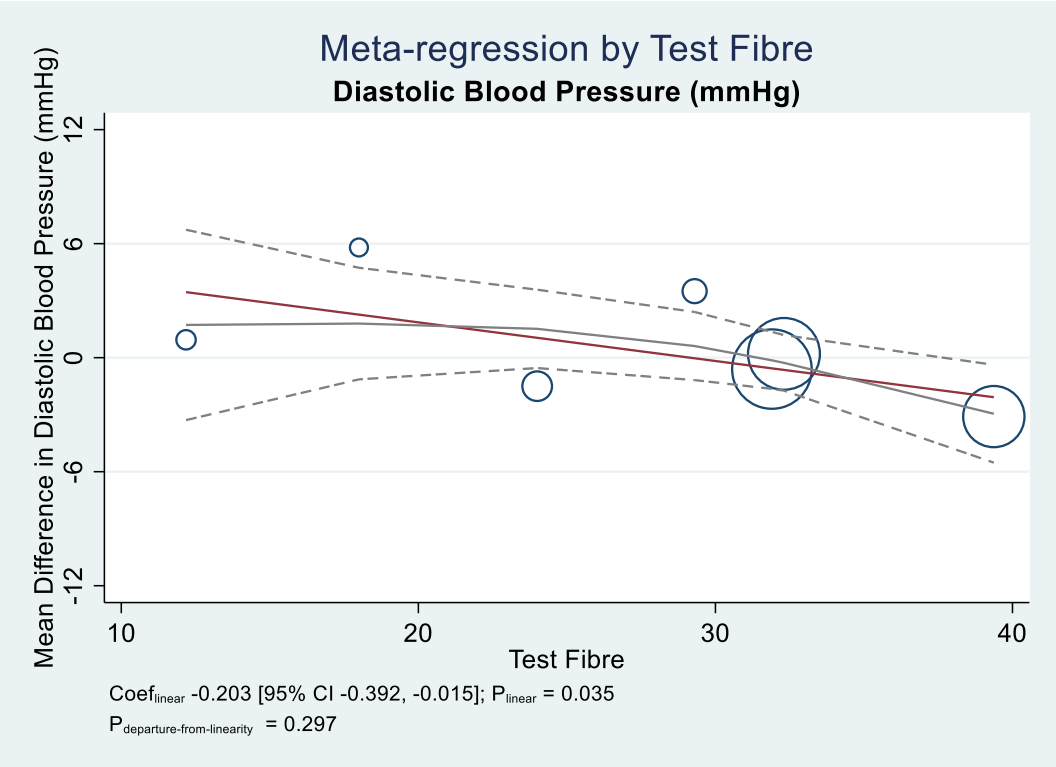
U


Meta-regression by Test Fibre
Systolic Blood Pressure (mmHg)
Mean Difference in Systolic Blood Pressure (mmHg)
12
6
0
-6
-12
10
20
30
40
Test Fibre
Coef$_{linear}$ -0.250 [95% CI -0.516, 0.015]; P$_{linear}$ = 0.064
P$_{departure\text{-}from\text{-}linearity}$ = 0.188

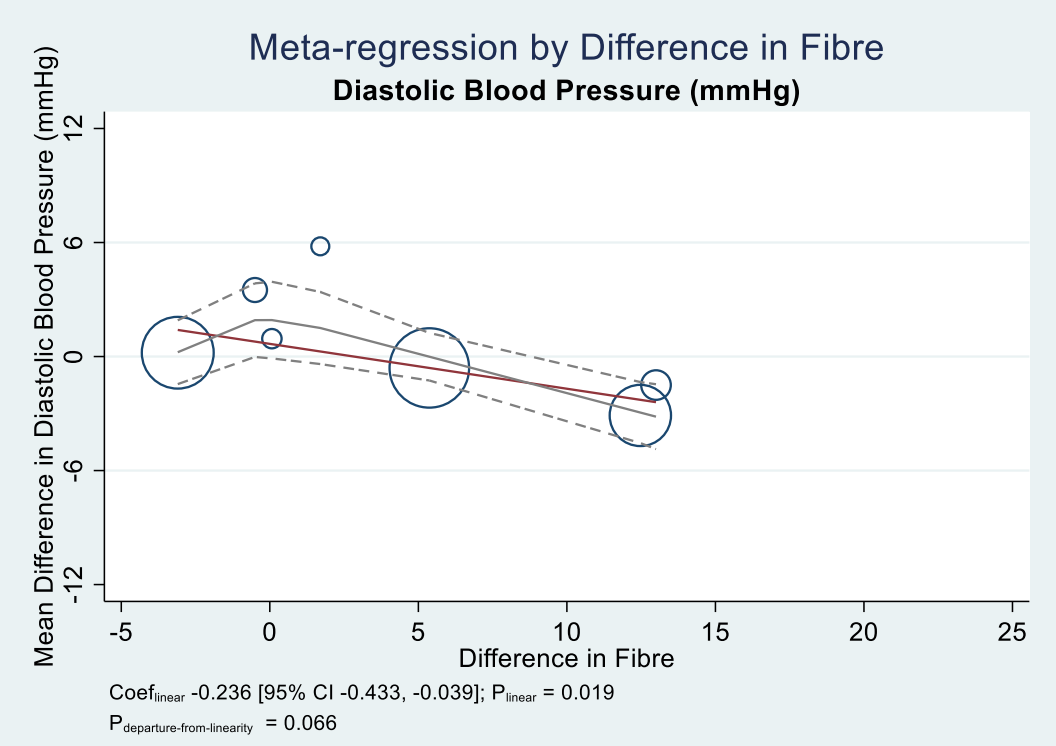
V


Meta-regression by Difference in Fibre
Systolic Blood Pressure (mmHg)
Mean Difference in Systolic Blood Pressure (mmHg)
12
6
0
-6
-12
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ -0.128 [95% CI -0.464, 0.208]; P$_{linear}$ = 0.455
P$_{departure\text{-}from\text{-}linearity}$ = 0.933

W


Meta-regression by Test Fibre
Diastolic Blood Pressure (mmHg)
Mean Difference in Diastolic Blood Pressure (mmHg)
-12
-6
0
6
12
10
20
30
40
Test Fibre
Coef$_{linear}$ -0.203 [95% CI -0.392, -0.015]; P$_{linear}$ = 0.035
P$_{departure\text{-}from\text{-}linearity}$ = 0.297

X


Meta-regression by Difference in Fibre
Diastolic Blood Pressure (mmHg)
Mean Difference in Diastolic Blood Pressure (mmHg)
-12
-6
0
6
12
-5
0
5
10
15
20
25
Difference in Fibre
Coef$_{linear}$ -0.236 [95% CI -0.433, -0.039]; P$_{linear}$ = 0.019
P$_{departure\text{-}from\text{-}linearity}$ = 0.066

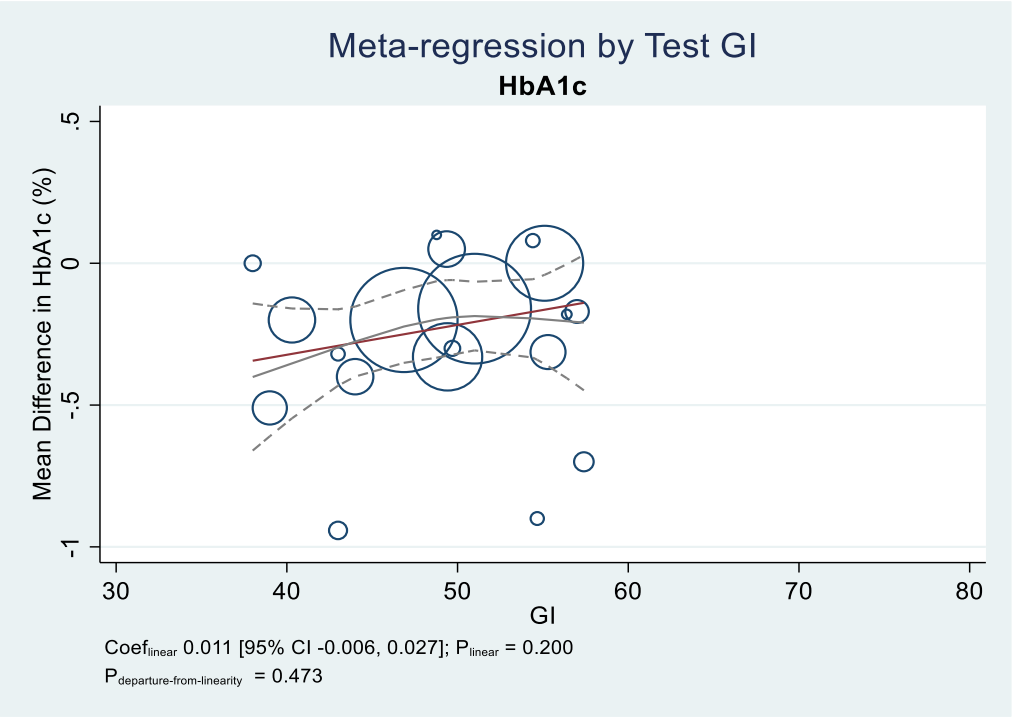
Individual studies are represented by the circles, with their weight in the overall analysis represented by the size of the circles. The straight red line represents the estimate linear dose response and the grey line the non-linear dose response for the GI of the diet and the dashed lines represent the upper and lower 95% confidence intervals. Linear and non-linear continuous subgroup analyses are presented for: **A**, Test Fibre and HbA1c; **B**, Difference in Fibre and HbA1c; **C**, Test Fibre and fasting glucose; **D**, Difference in Fibre and fasting glucose; **E**, Test Fibre and fasting insulin; **F**, Difference in Fibre and fasting insulin; **G**, Test Fibre and LDL-C; **H**, Difference in fibre and LDL-C; **I**, Test Fibre and non-HDL-C; **J**, Difference in Fibre and non-HDL-C; **K**, Test Fibre and HDL-C; **L**, Difference in Fibre and HDL-C; **M**, Test Fibre and triglycerides; **N**, Difference in Fibre and triglycerides; **O**, Test Fibre and body weight; **P**, Difference in Fibre and body weight; **Q**, Test Fibre and BMI; **R**, Difference in Fibre and BMI; **S**, Test Fibre and waist circumference; **T**, Difference in Fibre and waist circumference; **U**, Test Fibre and SBP; **V**, Difference in Fibre and SBP; **W**, Test Fibre and DBP; **X**, Difference in Fibre and DBP.

*Dose response analyses for fibre were not conducted for apoB and CRP due to <6 trial comparisons (n=5)

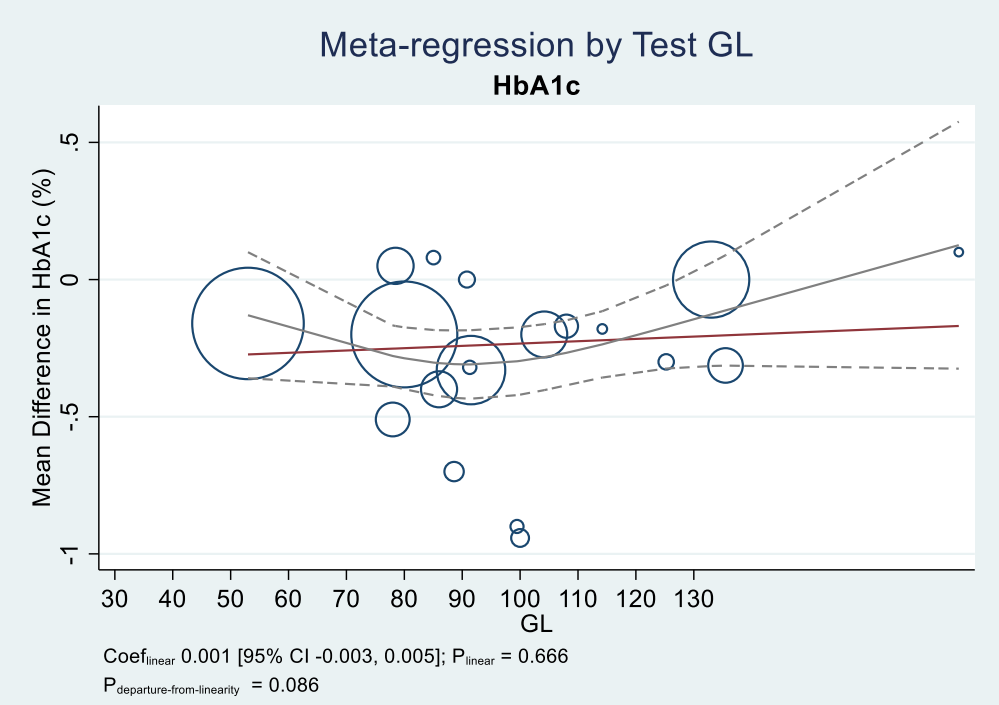
ApoB, apolipoprotein B; CI, confidence interval; Coef, coefficient; HDL-C, high-density lipoprotein-cholesterol; GI, glycemic index; GL, glycemic load; Non-HDL-C, non-high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; Test Fibre, the prescribed or in-trial achieved absolute dietary fibre on the low-GI/GL diets; Diff in Fibre, difference in fibre between the low-GI/GL diets and control diets (test-control)

Supplemental Figure S63: Linear and non-linear meta-regression analyses for the effect of low-GI and GL intervention dose on glycemic control in diabetes

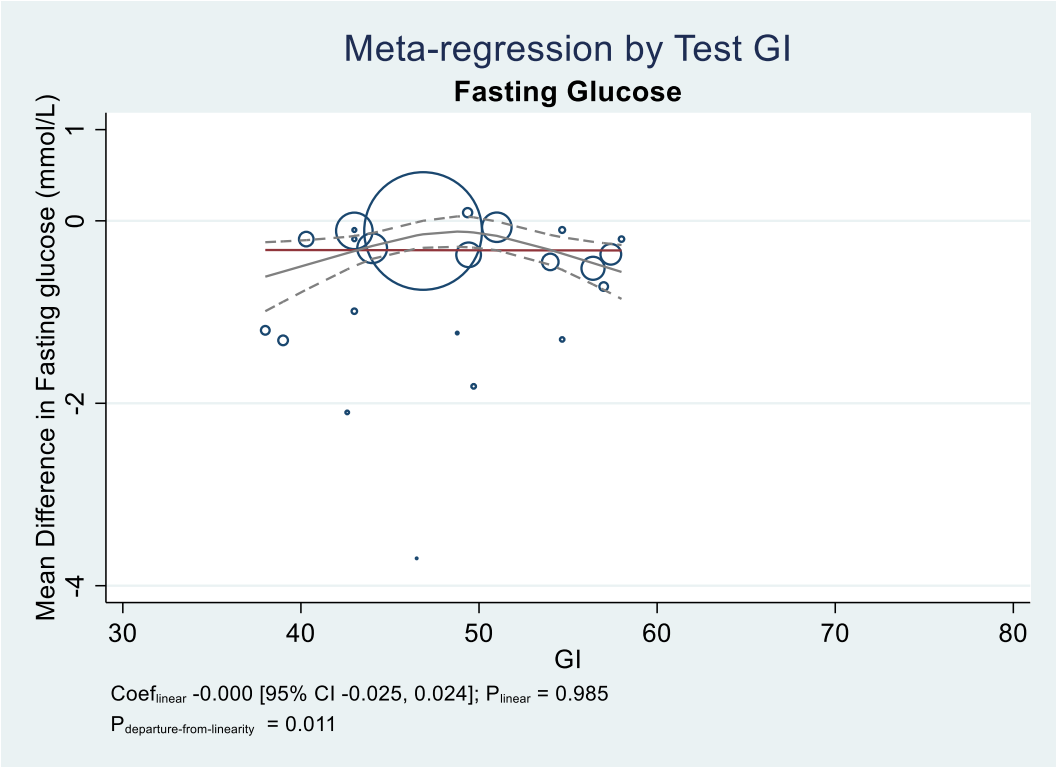
A



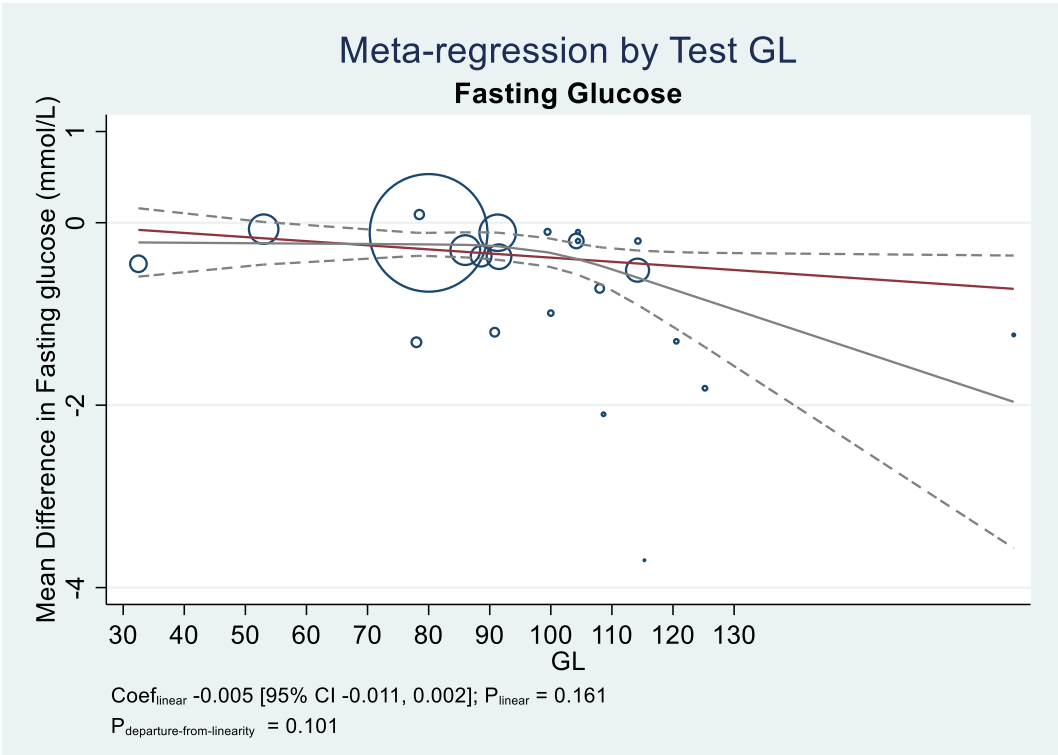
B



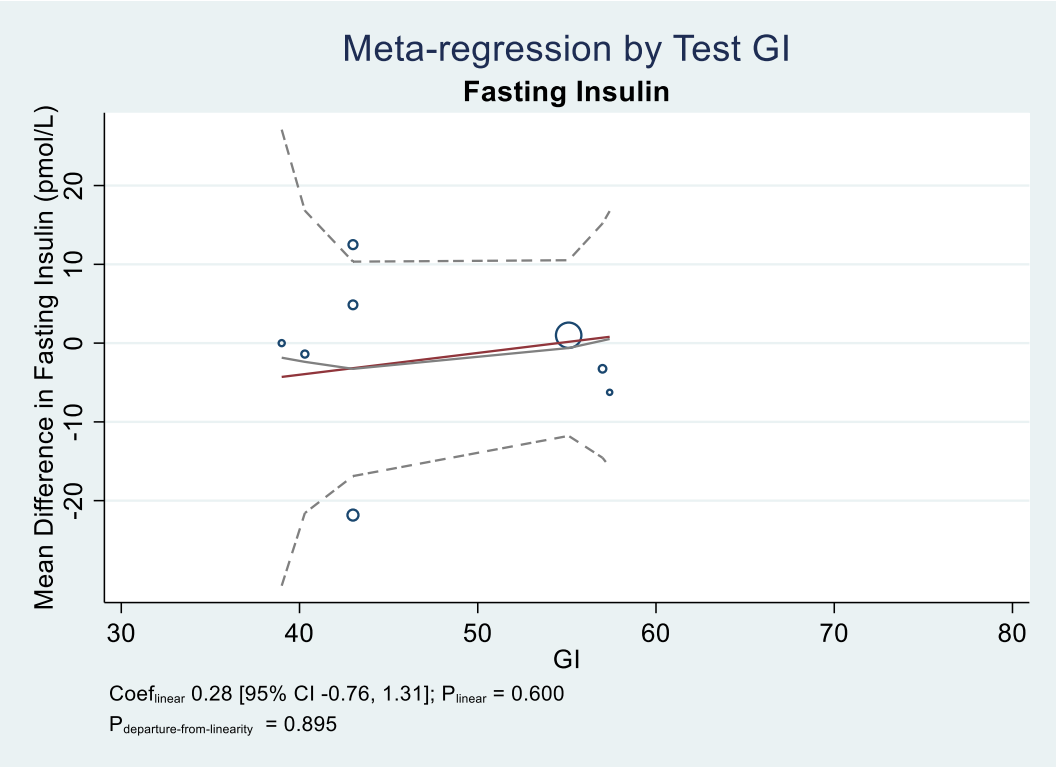
C


Meta-regression by Test GI
Fasting Glucose
Mean Difference in Fasting glucose (mmol/L)
1
0
-2
-4
30
40
50
60
70
80
GI
Coef$_{linear}$ -0.000 [95% CI -0.025, 0.024]; P$_{linear}$ = 0.985
P$_{departure\text{-}from\text{-}linearity}$ = 0.011

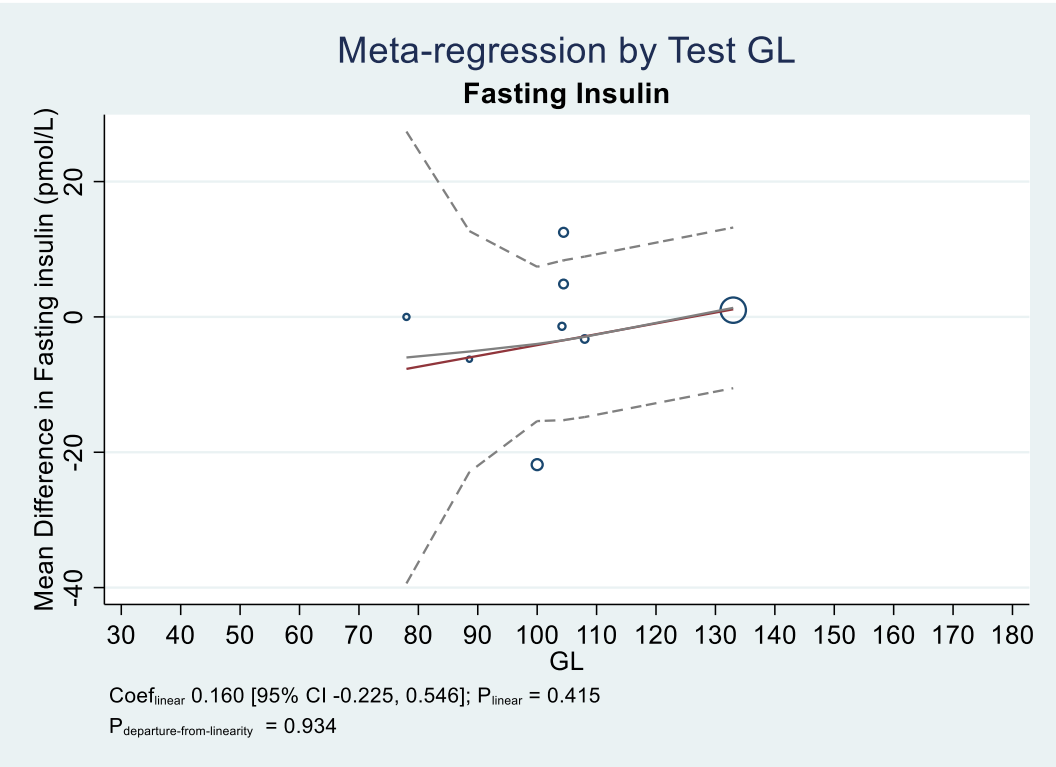
D


Meta-regression by Test GL
Fasting Glucose
Mean Difference in Fasting glucose (mmol/L)
1
0
-2
-4
30
40
50
60
70
80
90
100
110
120
130
GL
Coef$_{linear}$ -0.005 [95% CI -0.011, 0.002]; P$_{linear}$ = 0.161
P$_{departure\text{-}from\text{-}linearity}$ = 0.101

E


Meta-regression by Test GI
Fasting Insulin
Mean Difference in Fasting Insulin (pmol/L)
-20
-10
0
10
20
30
40
50
60
70
80
GI
Coef$_{linear}$ 0.28 [95% CI -0.76, 1.31]; P$_{linear}$ = 0.600
P$_{departure\text{-}from\text{-}linearity}$ = 0.895

F

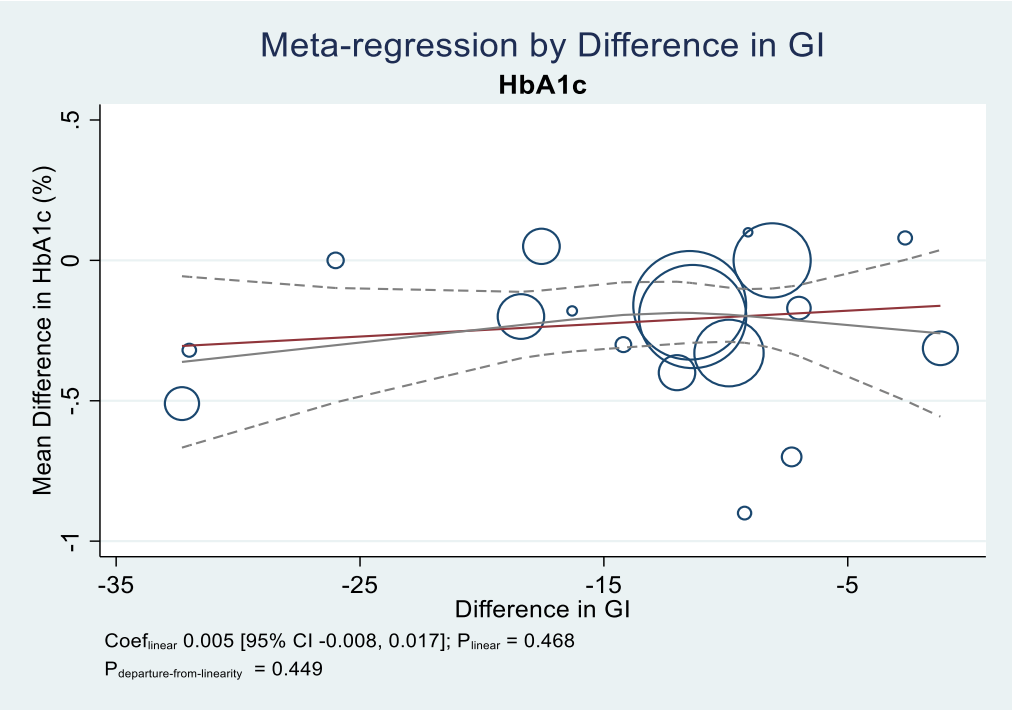

Meta-regression by Test GL
Fasting Insulin
Mean Difference in Fasting insulin (pmol/L)
-40
-20
0
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
GL
Coef$_{linear}$ 0.160 [95% CI -0.225, 0.546]; P$_{linear}$ = 0.415
P$_{departure\text{-}from\text{-}linearity}$ = 0.934

Individual studies are represented by the circles, with their weight in the overall analysis represented by the size of the circles. The straight red line represents the estimate linear dose response and the grey line the non-linear dose response for the GI of the diet and the dashed lines represent the upper and lower 95% confidence intervals. Linear and non-linear dose response analyses are presented for: **A**, Test GI and HbA1c; **B**, Test GL and HbA1c; **C**, Test GI and fasting glucose; **D**, Test GL and fasting glucose; **E**, Test GI and fasting insulin; **F**, Test GL and fasting insulin.

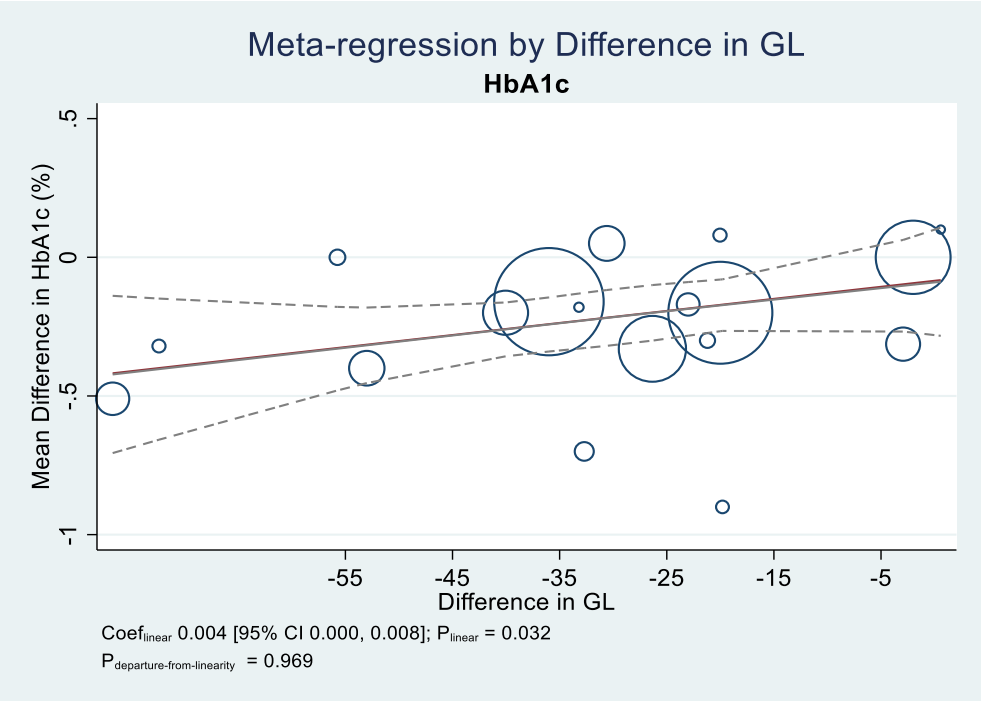
CI, confidence intervals; Coef, coefficient; GI, glycemic index; GL, Glycemic load; HbA1c, hemoglobin A1c; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S64: Linear and non-linear meta-regression analyses for the effect of low-GI/GL dietary patterns by difference in GI or GL between the intervention and control groups on glycemic control in diabetes

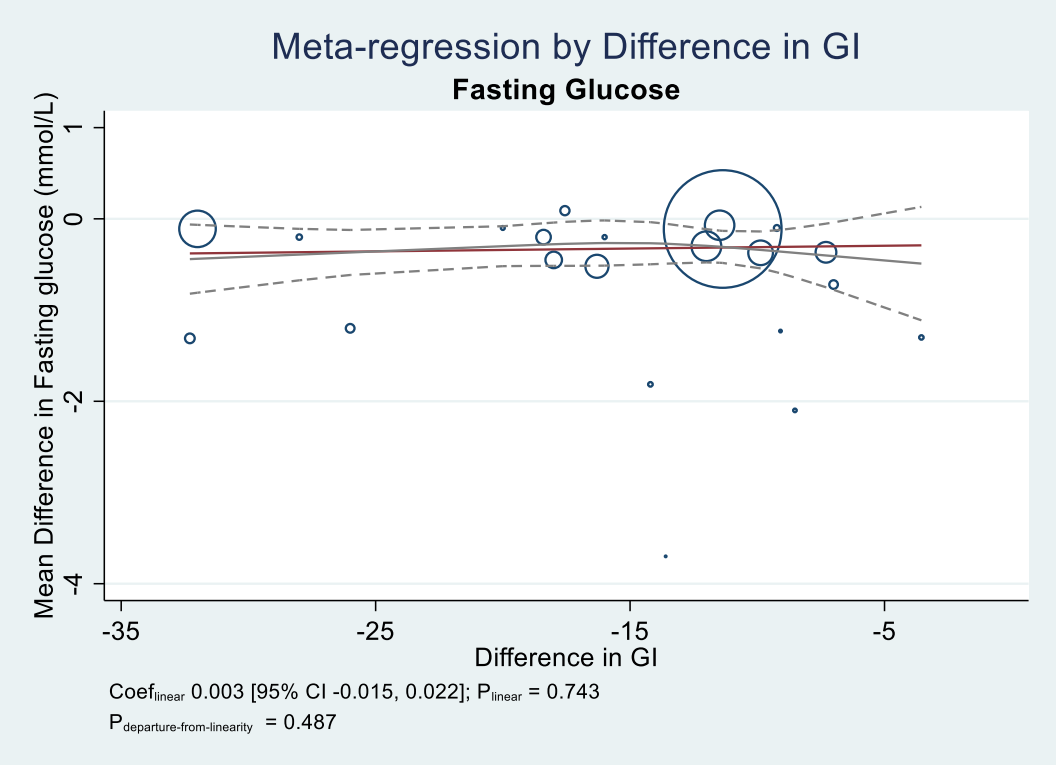
A



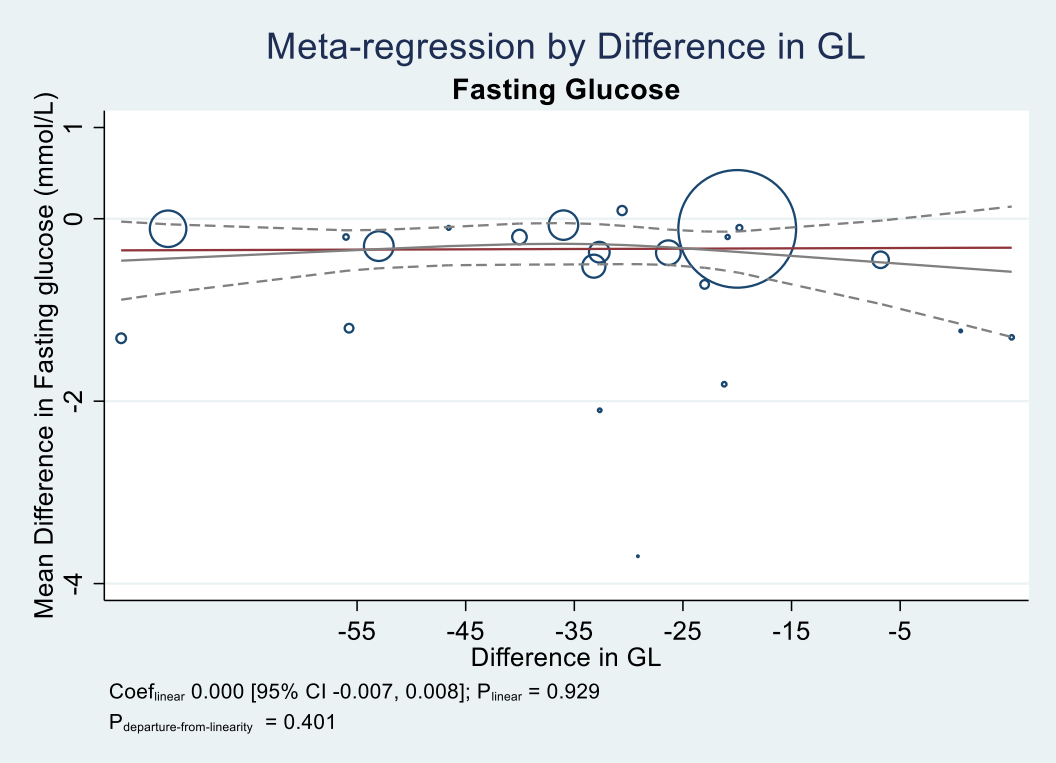
B



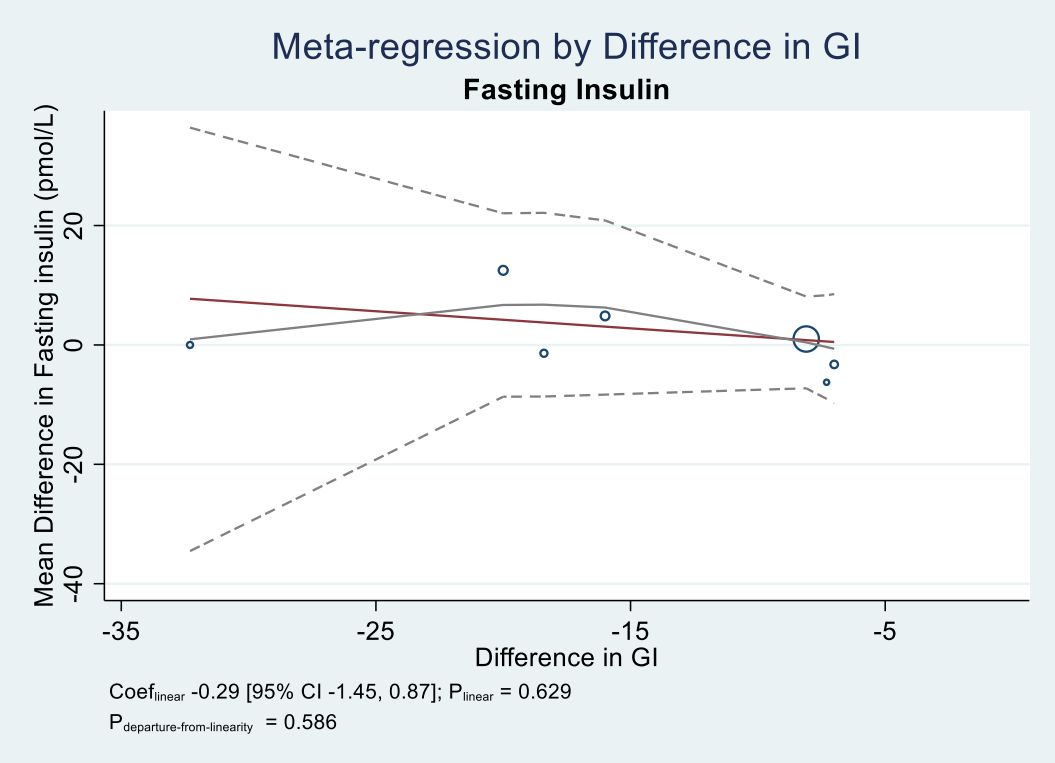
C


Meta-regression by Difference in GI
Fasting Glucose
Mean Difference in Fasting glucose (mmol/L)
1
0
-2
-4
-35
-25
-15
-5
Difference in GI
Coef$_{linear}$ 0.003 [95% CI -0.015, 0.022]; P$_{linear}$ = 0.743
P$_{departure\text{-}from\text{-}linearity}$ = 0.487

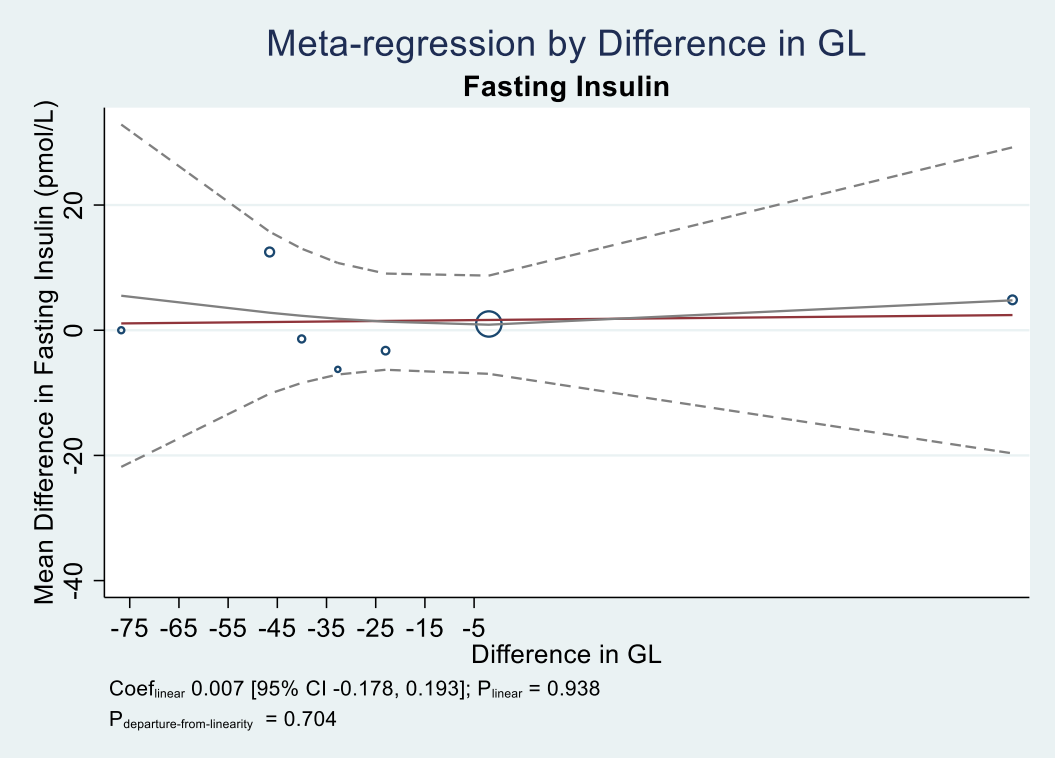
D


Meta-regression by Difference in GL
Fasting Glucose
Mean Difference in Fasting glucose (mmol/L)
1
0
-2
-4
-55
-45
-35
-25
-15
-5
Difference in GL
Coef$_{linear}$ 0.000 [95% CI -0.007, 0.008]; P$_{linear}$ = 0.929
P$_{departure\text{-}from\text{-}linearity}$ = 0.401

E


Meta-regression by Difference in GI
Fasting Insulin
Mean Difference in Fasting insulin (pmol/L)
-40
-20
0
20
-35
-25
-15
-5
Difference in GI
Coef linear -0.29 [95% CI -1.45, 0.87]; P linear = 0.629
P departure-from-linearity = 0.586

F

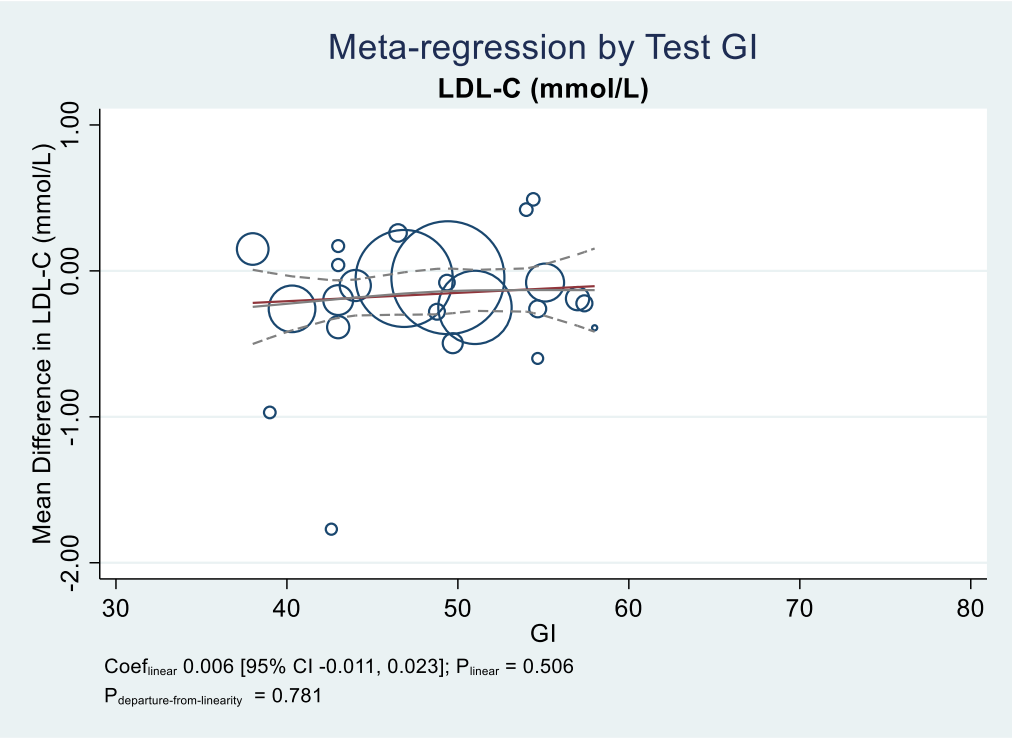

Meta-regression by Difference in GL
Fasting Insulin
Mean Difference in Fasting Insulin (pmol/L)
-40
-20
0
20
-75 -65 -55 -45 -35 -25 -15 -5
Difference in GL
Coef linear 0.007 [95% CI -0.178, 0.193]; P linear = 0.938
P departure-from-linearity = 0.704

Individual studies are represented by the circles, with their weight in the overall analysis represented by the size of the circles. The straight red line represents the estimate linear dose response and the grey line the non-linear dose response for the GI of the diet and the dashed lines represent the upper and lower 95% confidence intervals. Linear and non-linear dose response analyses are presented for: **A**, Difference in GI and HbA1c; **B**, Difference in GL and HbA1c; **C**, Difference in GI and fasting glucose; **D**, Difference in GL and fasting glucose; **E**, Difference in GI and fasting insulin; **F**, Difference in GL and fasting insulin. Note "Difference in" denotes difference in either GI or GL between the low-GI/GL and control diets (test – control) during the interventions, so that negative numbers denote the magnitude of reductions in GI/GL.

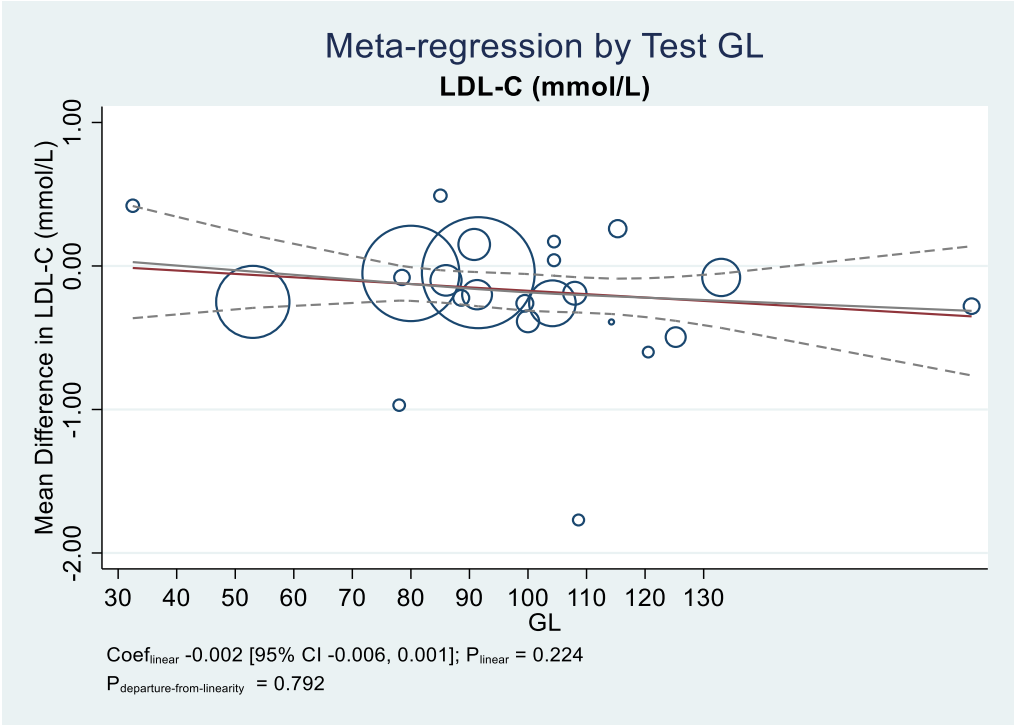
CI, confidence intervals; Coef, coefficient; Diff, difference; GI, glycemic index; GL, Glycemic load; HbA1c, hemoglobin A1c

Supplemental Figure S65: Linear and non-linear meta-regression analyses for the effect of low-GI and GL intervention dose on blood lipids in diabetes

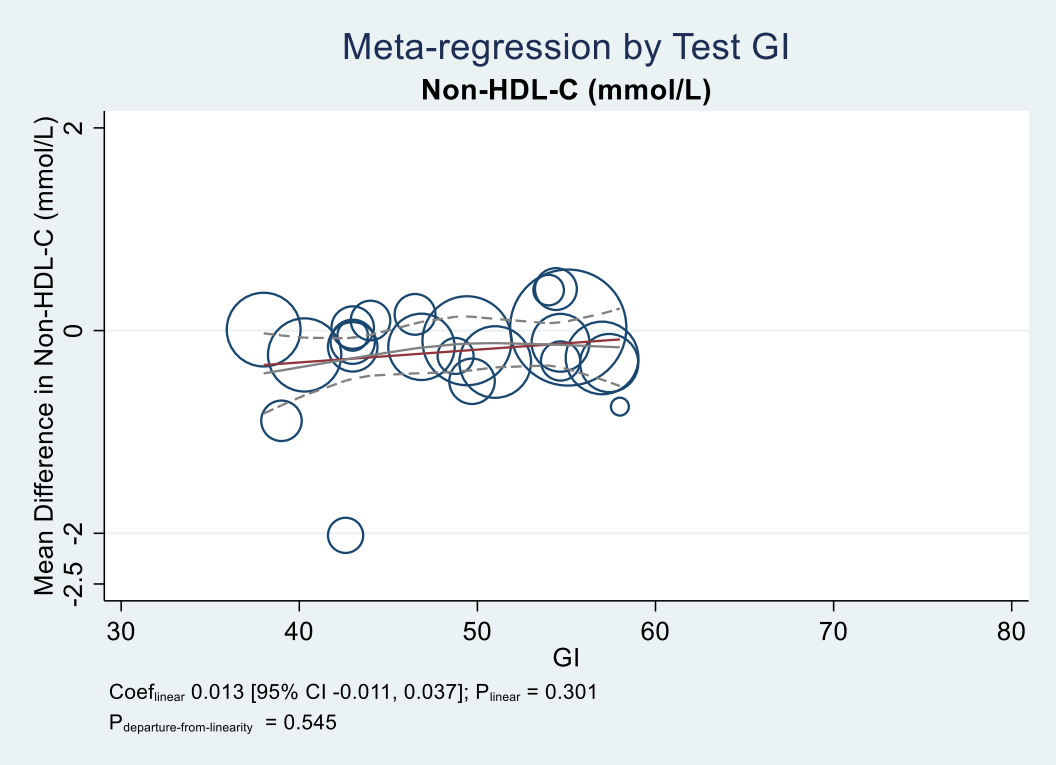
A



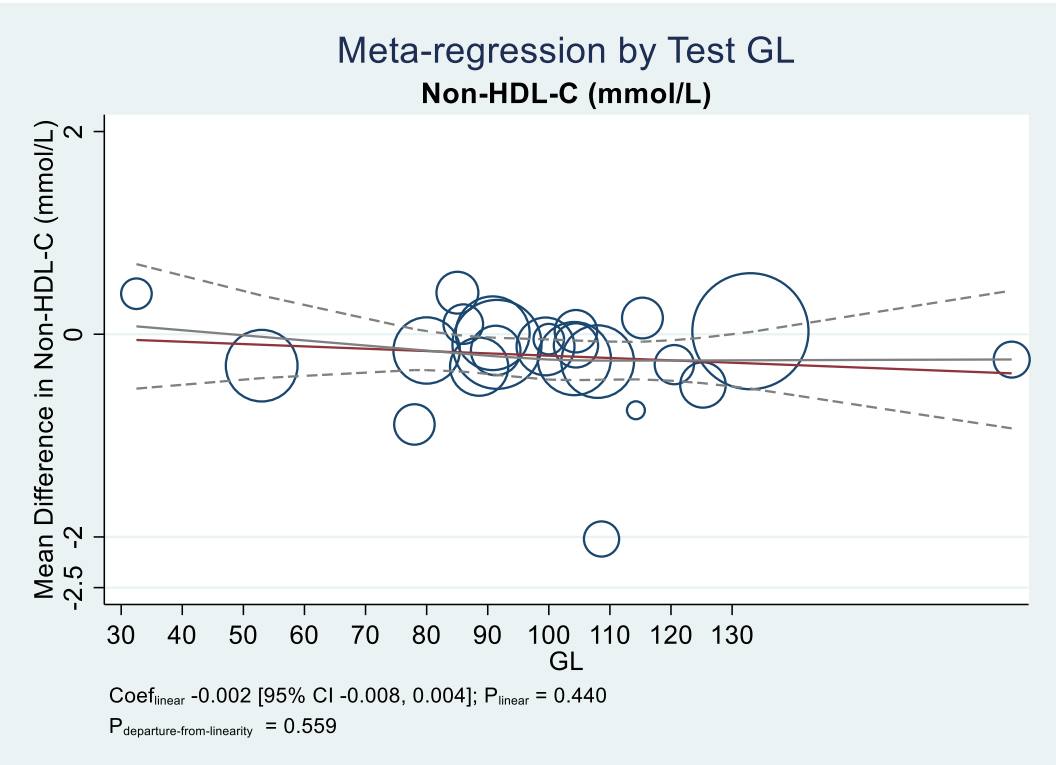
B



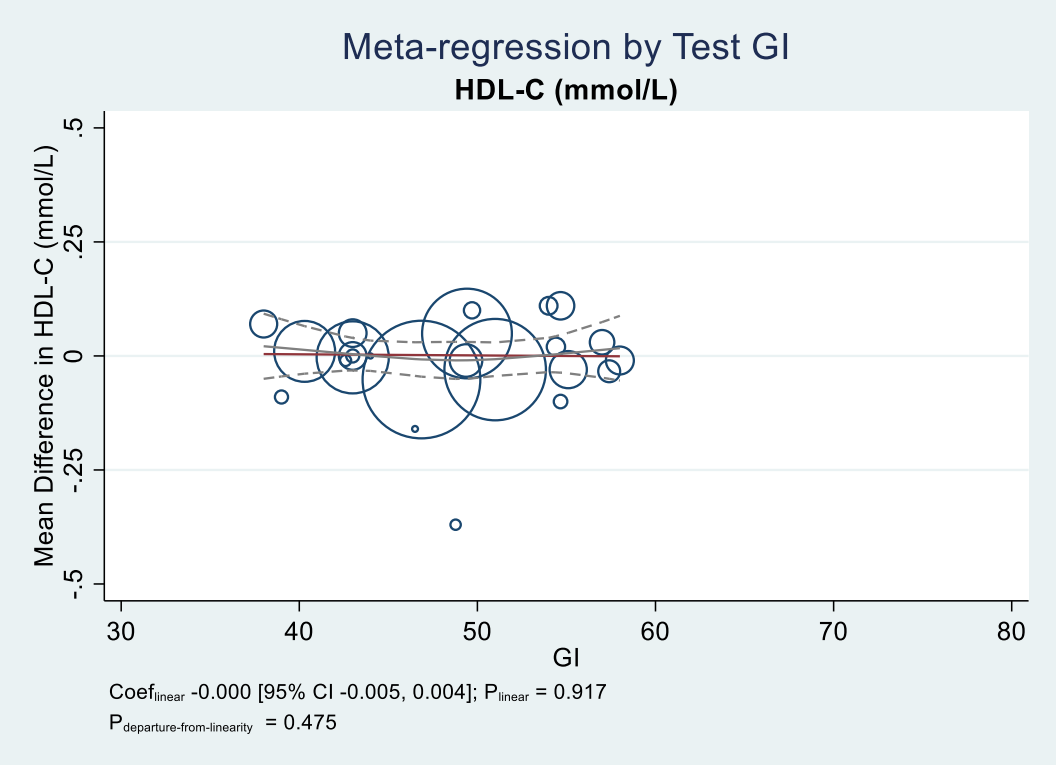
C


Meta-regression by Test GI
Non-HDL-C (mmol/L)
Mean Difference in Non-HDL-C (mmol/L)
2
0
-2
-2.5
30
40
50
60
70
80
GI
Coef$_{linear}$ 0.013 [95% CI -0.011, 0.037]; P$_{linear}$ = 0.301
P$_{departure\text{-}from\text{-}linearity}$ = 0.545

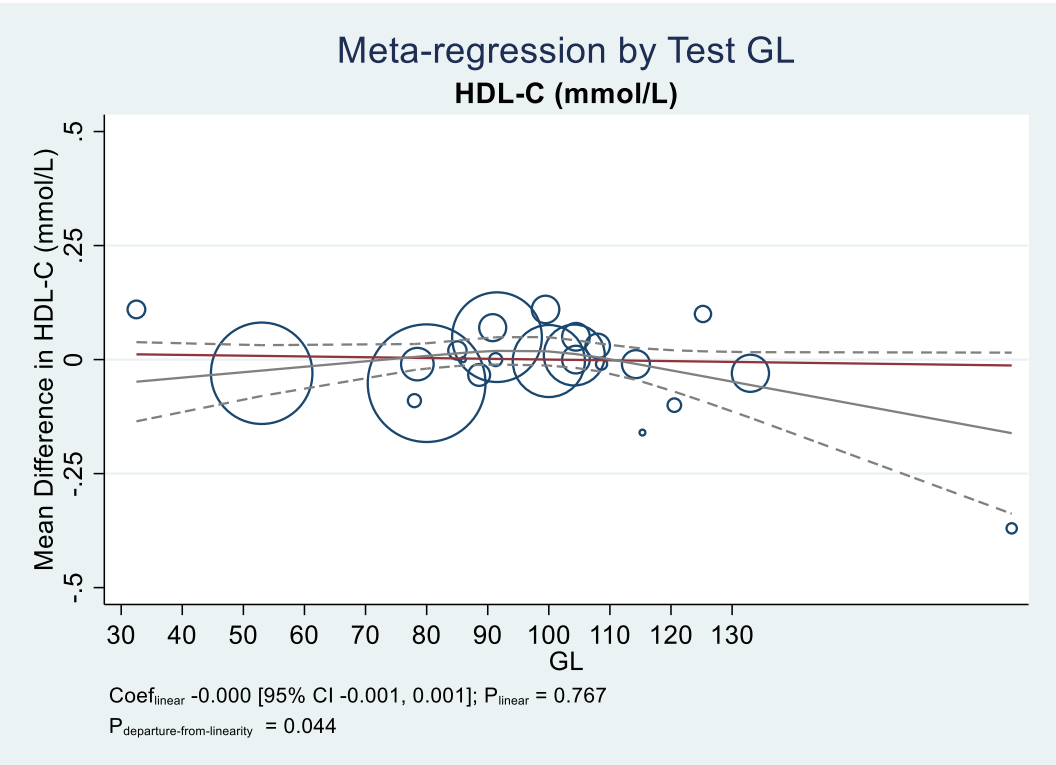
D


Meta-regression by Test GL
Non-HDL-C (mmol/L)
Mean Difference in Non-HDL-C (mmol/L)
2
0
-2
-2.5
30
40
50
60
70
80
90
100
110
120
130
GL
Coef$_{linear}$ -0.002 [95% CI -0.008, 0.004]; P$_{linear}$ = 0.440
P$_{departure\text{-}from\text{-}linearity}$ = 0.559

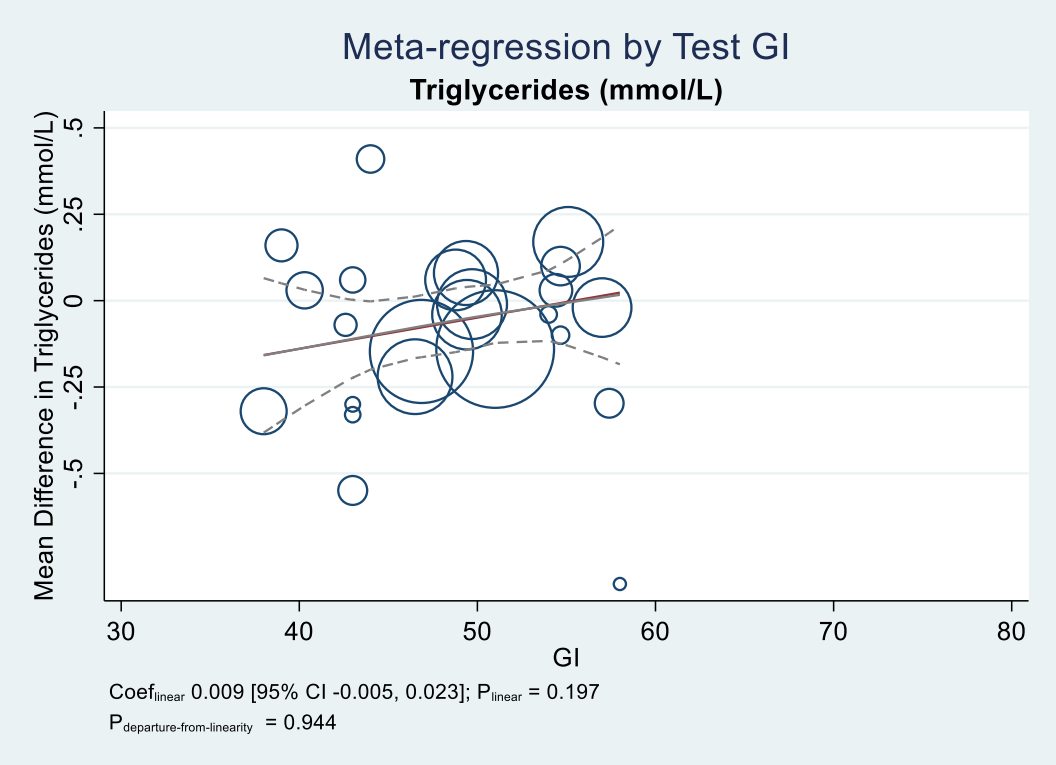
E


Meta-regression by Test GI
HDL-C (mmol/L)
Mean Difference in HDL-C (mmol/L)
-.5
-.25
0
.25
.5
30
40
50
60
70
80
GI
Coef linear -0.000 [95% CI -0.005, 0.004]; P linear = 0.917
P departure-from-linearity = 0.475

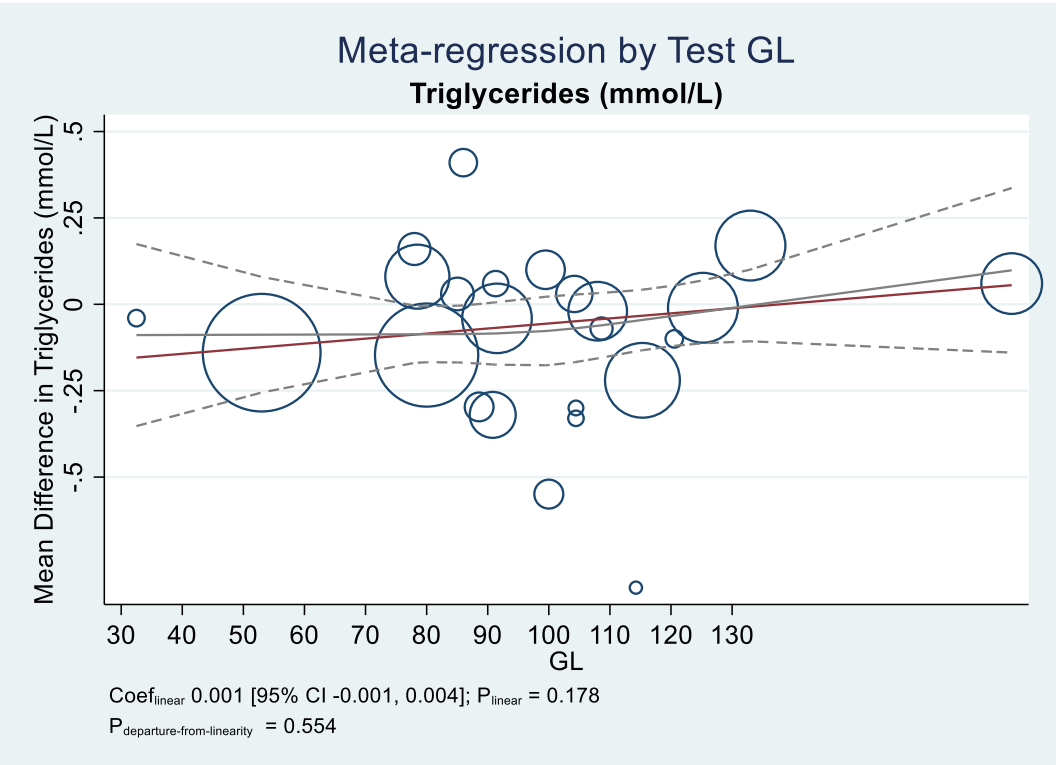
F


Meta-regression by Test GL
HDL-C (mmol/L)
Mean Difference in HDL-C (mmol/L)
-.5
-.25
0
.25
.5
30
40
50
60
70
80
90
100
110
120
130
GL
Coef linear -0.000 [95% CI -0.001, 0.001]; P linear = 0.767
P departure-from-linearity = 0.044

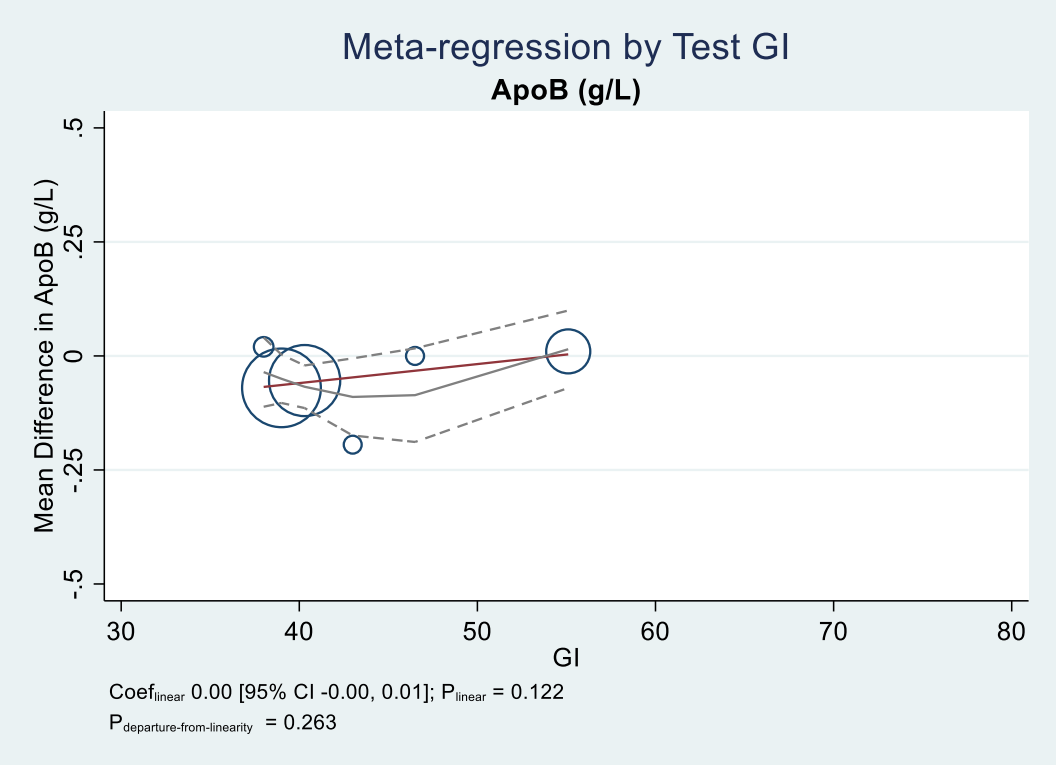
G


Meta-regression by Test GI
Triglycerides (mmol/L)
Mean Difference in Triglycerides (mmol/L)
-.5
-.25
0
.25
.5
30
40
50
60
70
80
GI
Coef$_{linear}$ 0.009 [95% CI -0.005, 0.023]; P$_{linear}$ = 0.197
P$_{departure\text{-}from\text{-}linearity}$ = 0.944

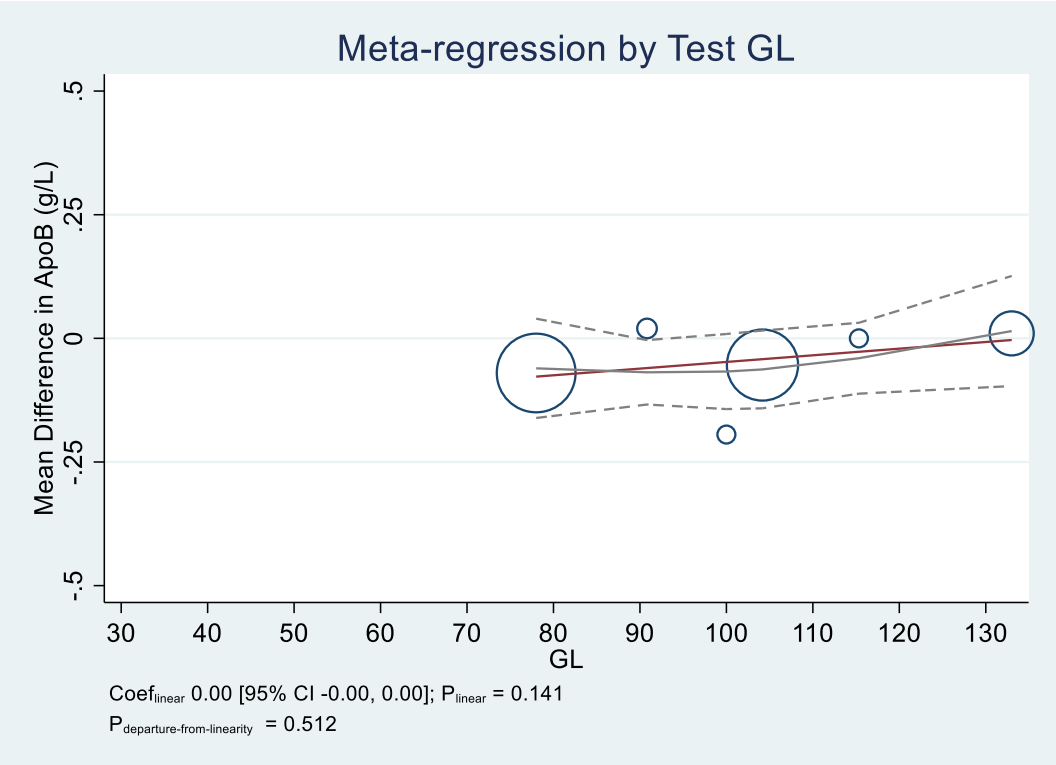
H


Meta-regression by Test GL
Triglycerides (mmol/L)
Mean Difference in Triglycerides (mmol/L)
-.5
-.25
0
.25
.5
30
40
50
60
70
80
90
100
110
120
130
GL
Coef$_{linear}$ 0.001 [95% CI -0.001, 0.004]; P$_{linear}$ = 0.178
P$_{departure\text{-}from\text{-}linearity}$ = 0.554

I


Meta-regression by Test GI
ApoB (g/L)
Mean Difference in ApoB (g/L)
.5
.25
0
-.25
-.5
30
40
50
60
70
80
GI
Coef linear 0.00 [95% CI -0.00, 0.01]; P linear = 0.122
P departure-from-linearity = 0.263

J

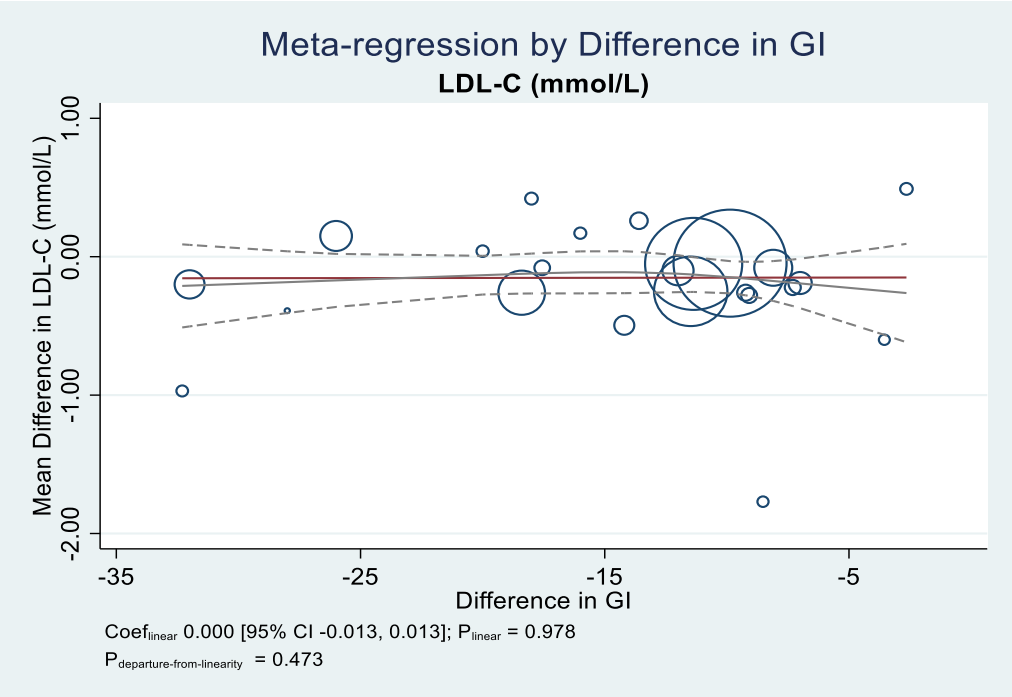

Meta-regression by Test GL
Mean Difference in ApoB (g/L)
.5
.25
0
-.25
-.5
30
40
50
60
70
80
90
100
110
120
130
GL
Coef linear 0.00 [95% CI -0.00, 0.00]; P linear = 0.141
P departure-from-linearity = 0.512

Individual studies are represented by the circles, with their weight in the overall analysis represented by the size of the circles. The straight red line represents the estimate linear dose response and the grey line the non-linear dose response for the GI of the diet and the dashed lines represent the upper and lower 95% confidence intervals. Linear and non-linear dose response analyses are presented for: **A**, Test GI and LDL-C; **B**, Test GL and LDL-C; **C**, Test GI and non-HDL-C; **D**, Test GL and non-HDL-C; **E**, Test GI and HDL-C; **F**, Test GL and HDL-C; **G**, Test GI and triglycerides; **H**, Test GL and triglycerides; **I**, Test GI and apoB; **J**, Test GL and apoB.

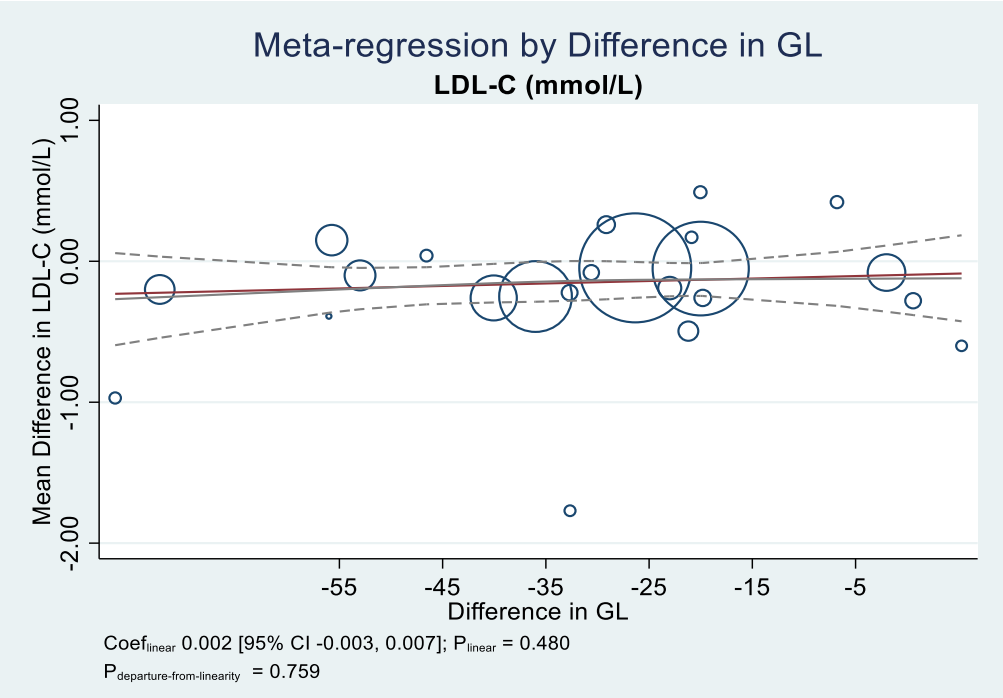
ApoB, apolipoprotein B; CI, confidence interval; Coef, coefficient; HDL-C, high-density lipoprotein-cholesterol; GI, glycemic index; GL, glycemic load; Non-HDL-C, non-high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S66: Linear and non-linear meta-regression analyses for the effect of low-GI/GL dietary patterns by difference in GI or GL between the intervention and control groups on blood lipids in diabetes*

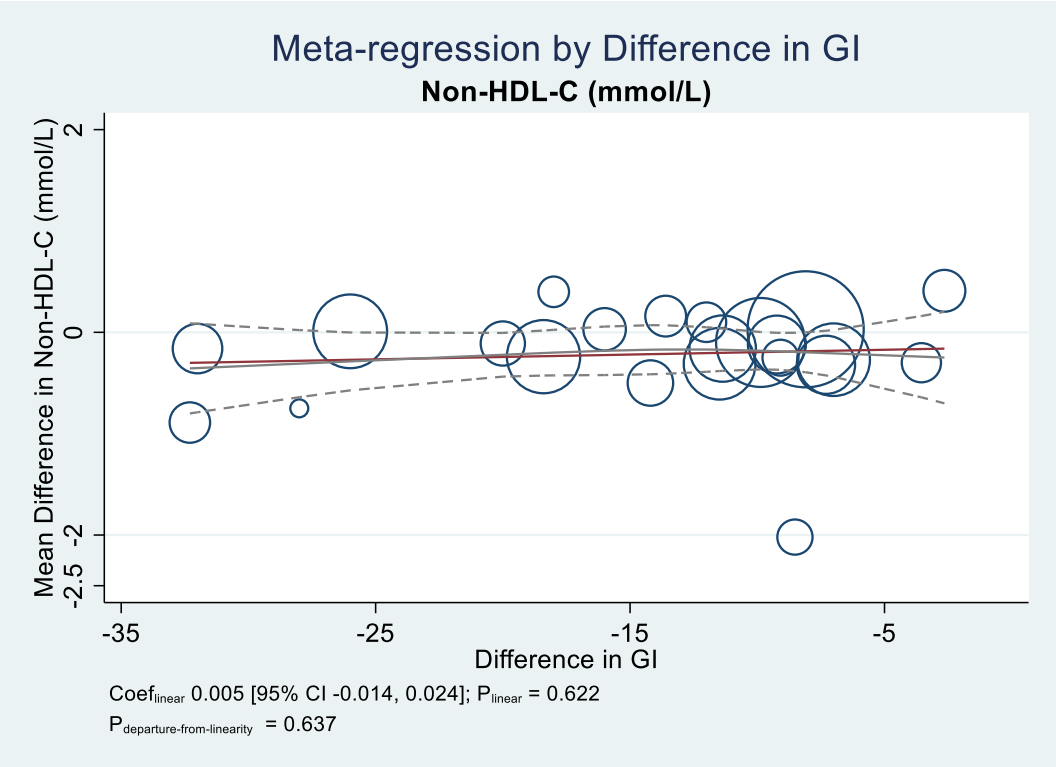
A



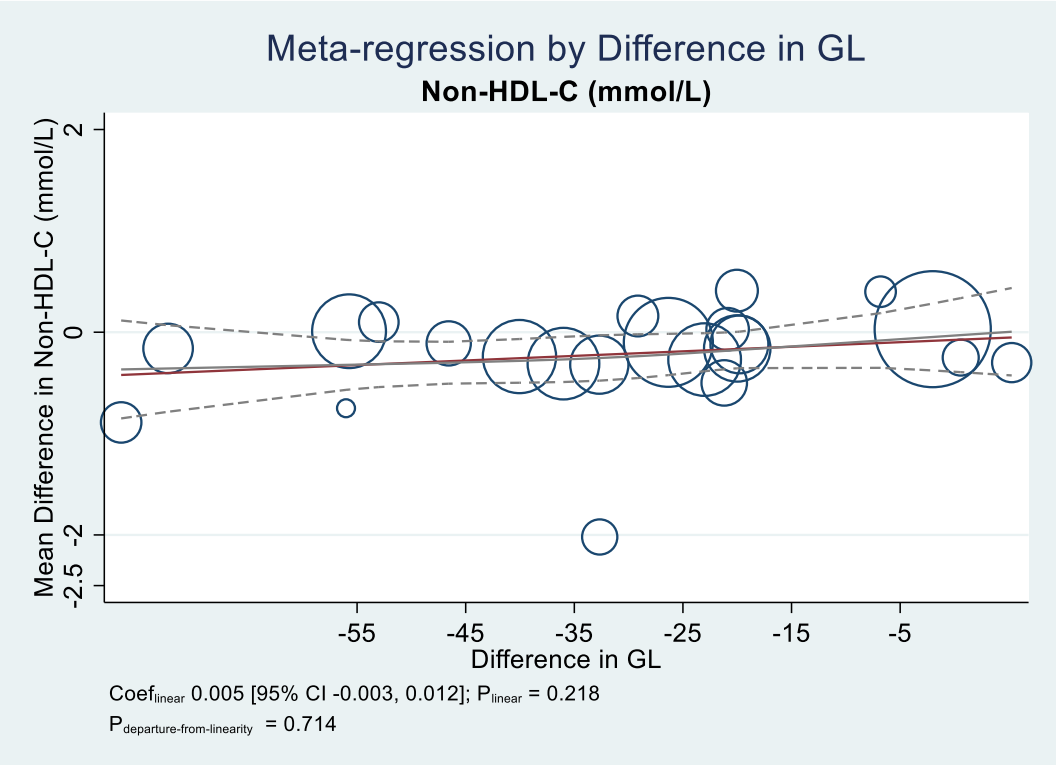
B



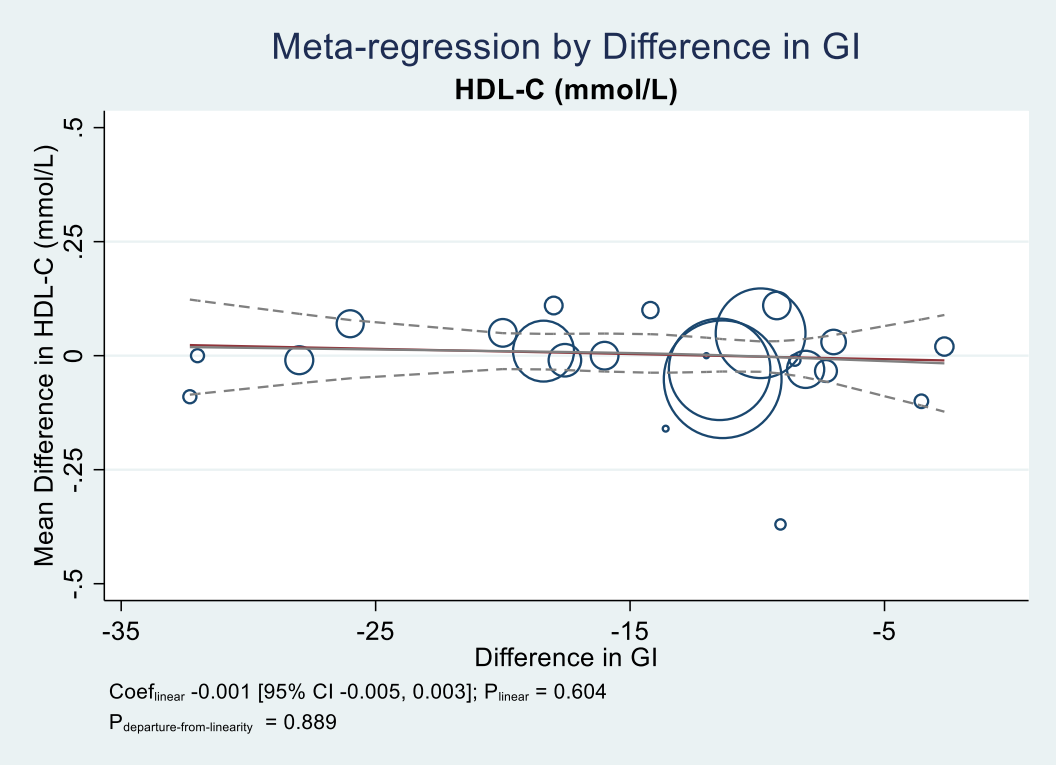
C


Meta-regression by Difference in GI
Non-HDL-C (mmol/L)
Mean Difference in Non-HDL-C (mmol/L)
2
0
-2
-2.5
-35
-25
-15
-5
Difference in GI
Coef$_{linear}$ 0.005 [95% CI -0.014, 0.024]; P$_{linear}$ = 0.622
P$_{departure\text{-}from\text{-}linearity}$ = 0.637

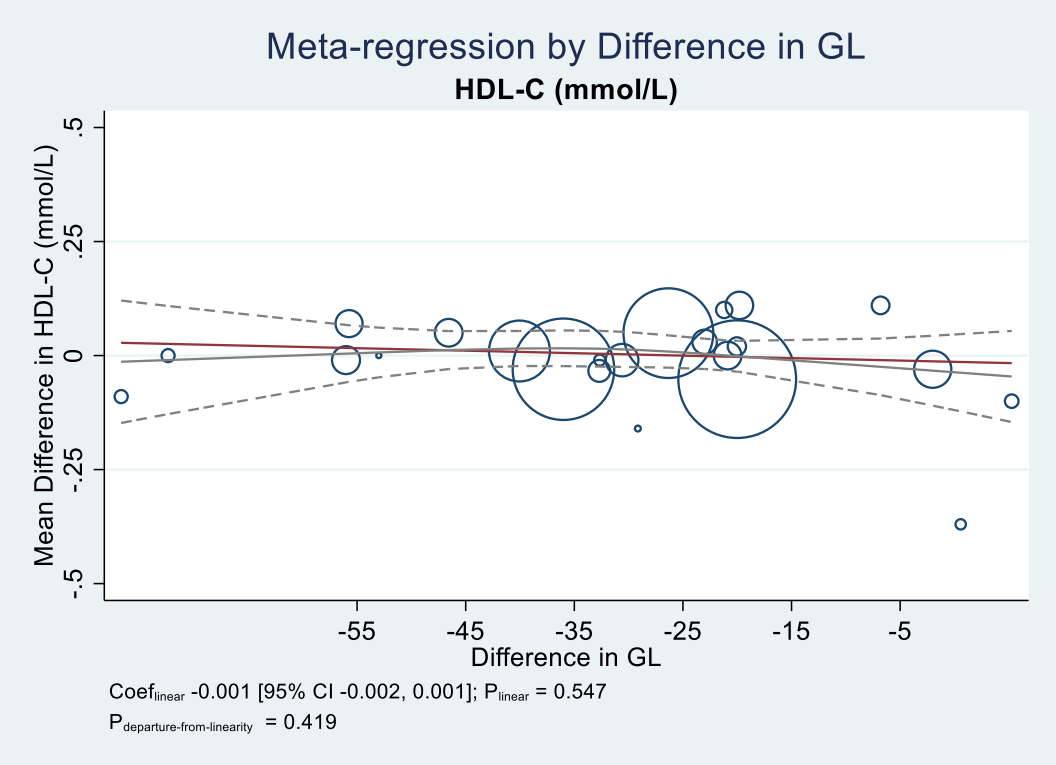
D


Meta-regression by Difference in GL
Non-HDL-C (mmol/L)
Mean Difference in Non-HDL-C (mmol/L)
2
0
-2
-2.5
-55
-45
-35
-25
-15
-5
Difference in GL
Coef$_{linear}$ 0.005 [95% CI -0.003, 0.012]; P$_{linear}$ = 0.218
P$_{departure\text{-}from\text{-}linearity}$ = 0.714

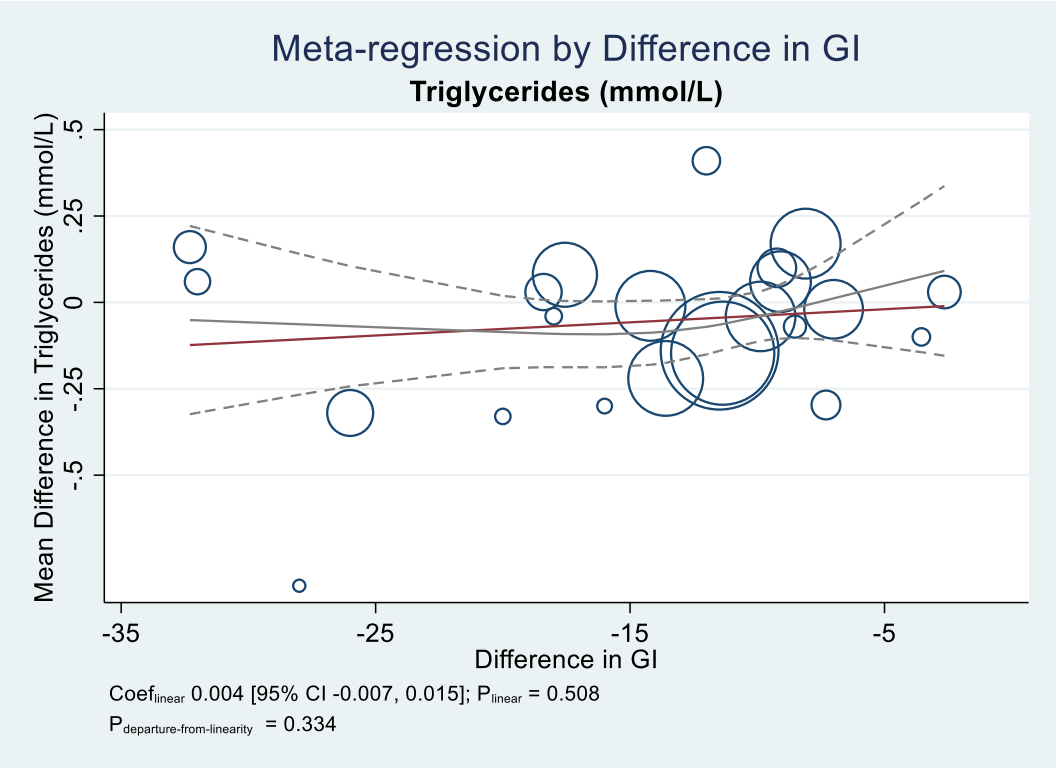
E


Meta-regression by Difference in GI
HDL-C (mmol/L)
Mean Difference in HDL-C (mmol/L)
.5
.25
0
-.25
-.5
-35
-25
-15
-5
Difference in GI
Coef$_{linear}$ -0.001 [95% CI -0.005, 0.003]; P$_{linear}$ = 0.604
P$_{departure\text{-}from\text{-}linearity}$ = 0.889

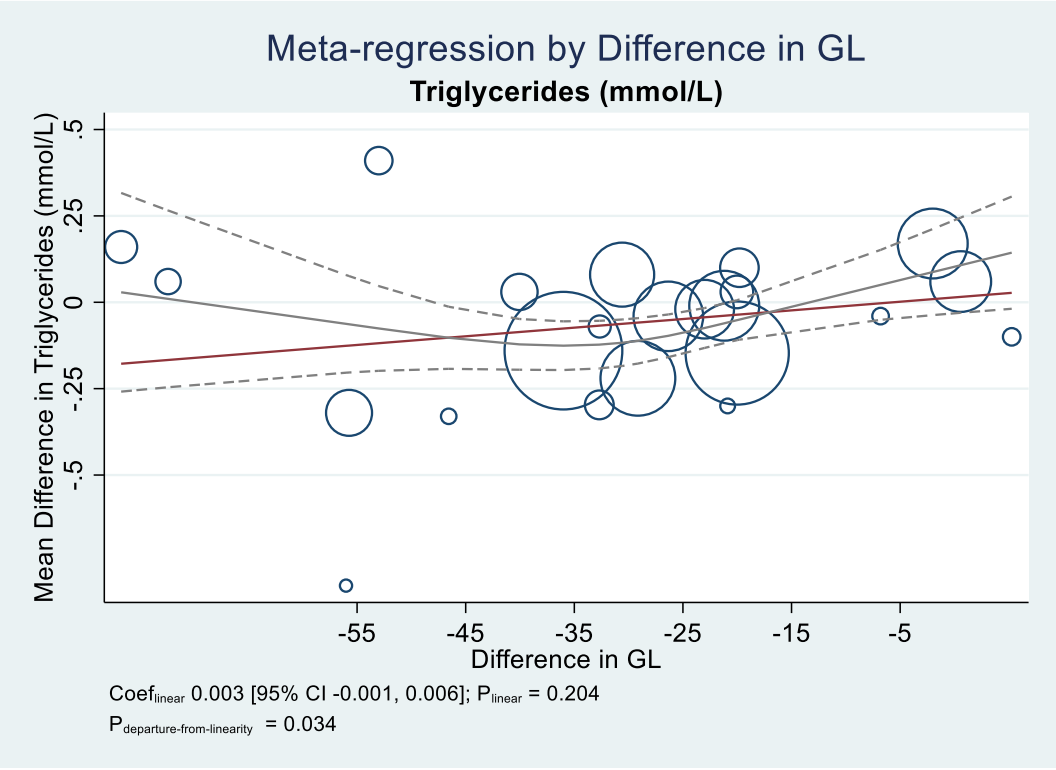
F


Meta-regression by Difference in GL
HDL-C (mmol/L)
Mean Difference in HDL-C (mmol/L)
.5
.25
0
-.25
-.5
-55
-45
-35
-25
-15
-5
Difference in GL
Coef$_{linear}$ -0.001 [95% CI -0.002, 0.001]; P$_{linear}$ = 0.547
P$_{departure\text{-}from\text{-}linearity}$ = 0.419

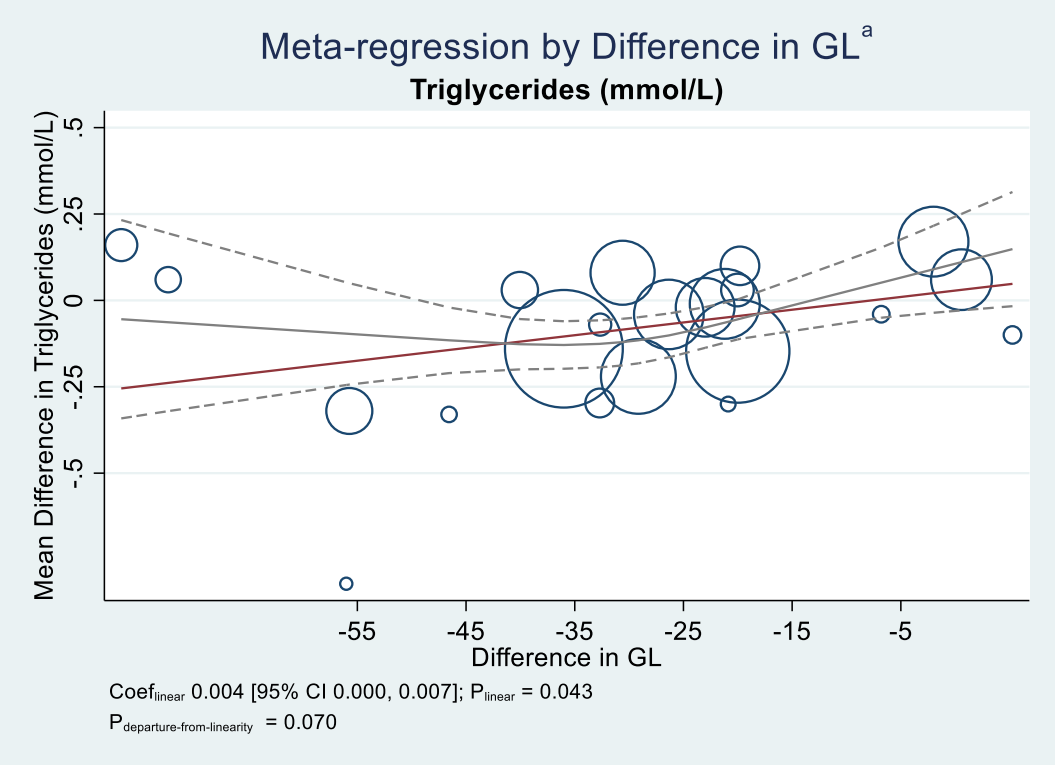
G


Meta-regression by Difference in GI
Triglycerides (mmol/L)
Mean Difference in Triglycerides (mmol/L)
.5
.25
0
-.25
-.5
-35
-25
-15
-5
Difference in GI
Coef$_{linear}$ 0.004 [95% CI -0.007, 0.015]; P$_{linear}$ = 0.508
P$_{departure\text{-}from\text{-}linearity}$ = 0.334

H


Meta-regression by Difference in GL
Triglycerides (mmol/L)
Mean Difference in Triglycerides (mmol/L)
.5
.25
0
-.25
-.5
-55
-45
-35
-25
-15
-5
Difference in GL
Coef$_{linear}$ 0.003 [95% CI -0.001, 0.006]; P$_{linear}$ = 0.204
P$_{departure\text{-}from\text{-}linearity}$ = 0.034

I



a meta-regression of difference in GL with the removal of a single outlier of effect (Jimenez-cruz et al. 2003)

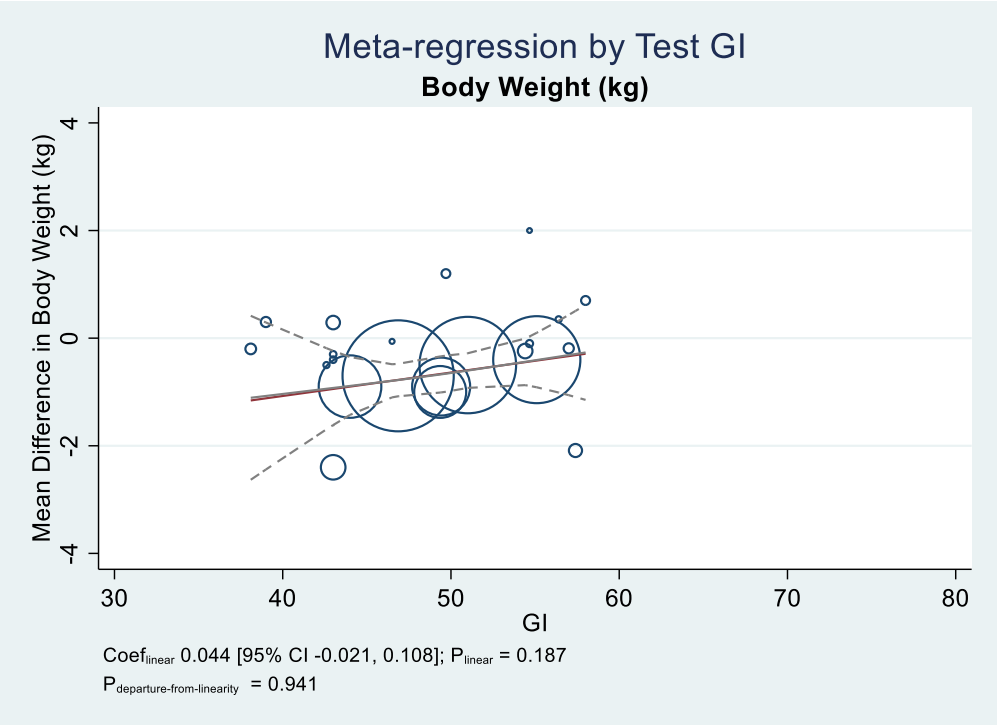
Individual studies are represented by the circles, with their weight in the overall analysis represented by the size of the circles. The straight red line represents the estimate linear dose response and the grey line the non-linear dose response for the GI of the diet and the dashed lines represent the upper and lower 95% confidence intervals. Linear and non-linear dose response analyses are presented for: **A**, Difference in GI and LDL-C; **B**, Difference in GL and LDL-C; **C**, Difference in GI and non-HDL-C; **D**, Difference in GL and non-HDL-C; **E**, Difference in GI and HDL-C; **F**, Difference in GL and HDL-C; **G**, Difference in GI and triglycerides; **H**, Difference in GL and triglycerides; **I**, sensitivity analysis of Difference in GL and triglycerides after removal of an outlier. Note "Difference in" denotes difference in either GI or GL between the low-GI/GL and control diets (test – control) during the interventions, so that negative numbers denote the magnitude of reductions in GI/GL.

*Dose response analyses were not conducted on either difference in GI or GL for apoB due to <6 trial comparisons (n=5)

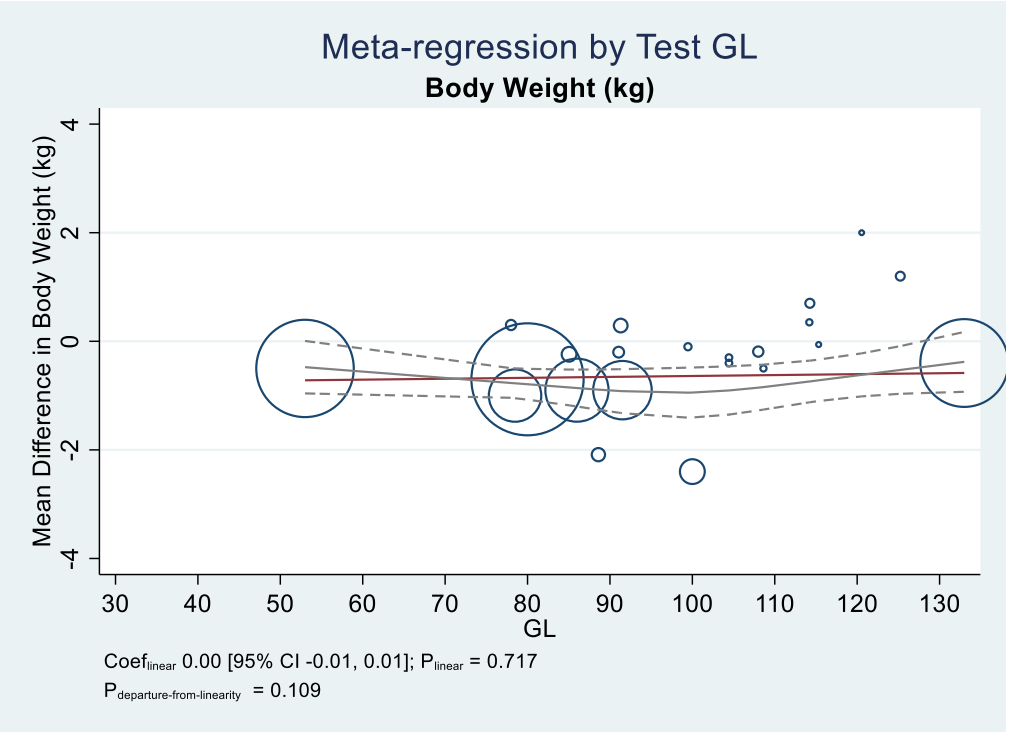
ApoB, apolipoprotein B; CI, confidence interval; Coef, coefficient; HDL-C, high-density lipoprotein-cholesterol; GI, glycemic index; GL, glycemic load; Non-HDL-C, non-high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol

Supplemental Figure S67: Linear and non-linear meta-regression analyses for the effect of low-GI and GL intervention dose on adiposity in diabetes

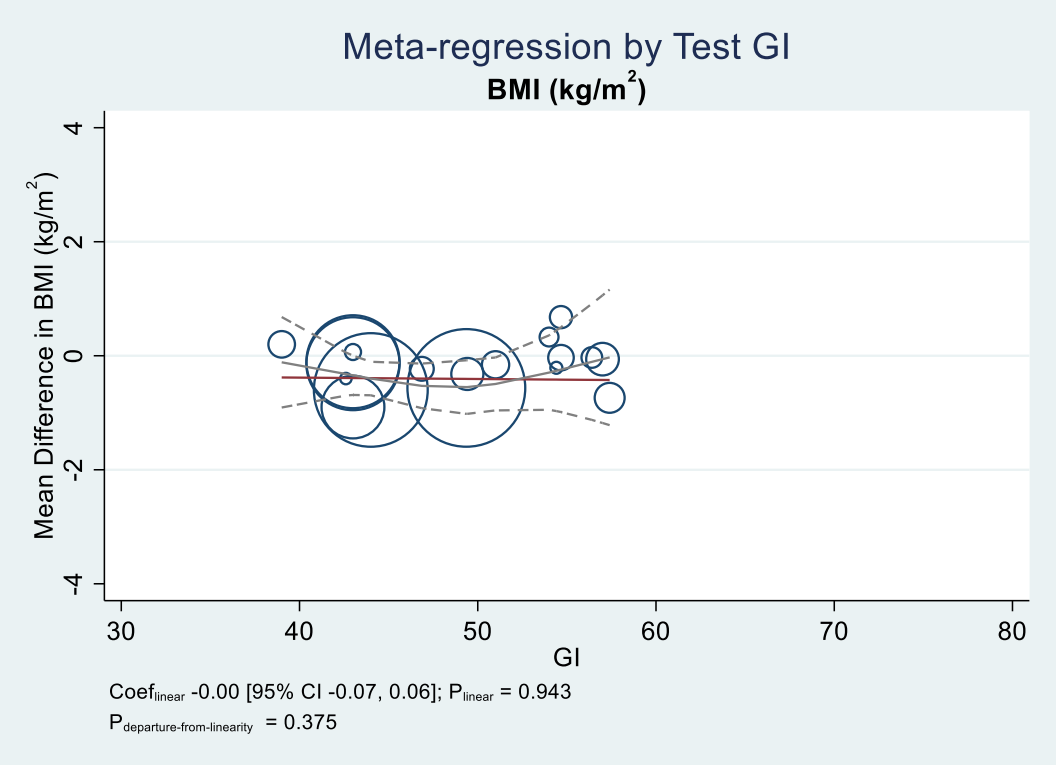
A



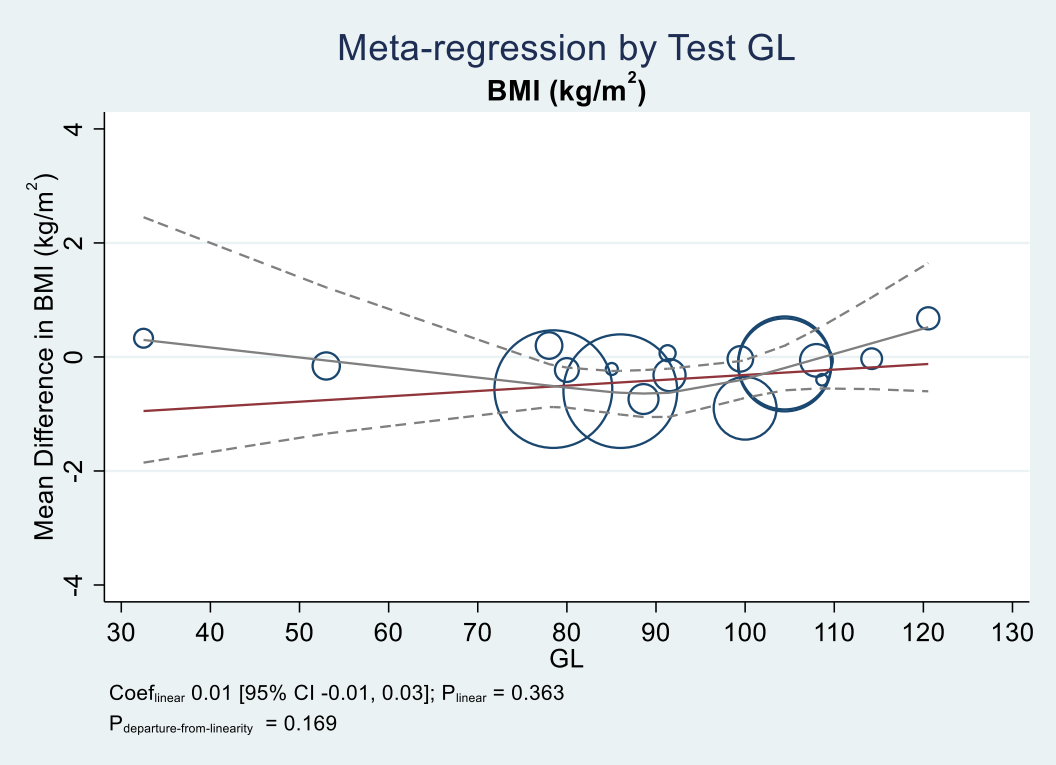
B



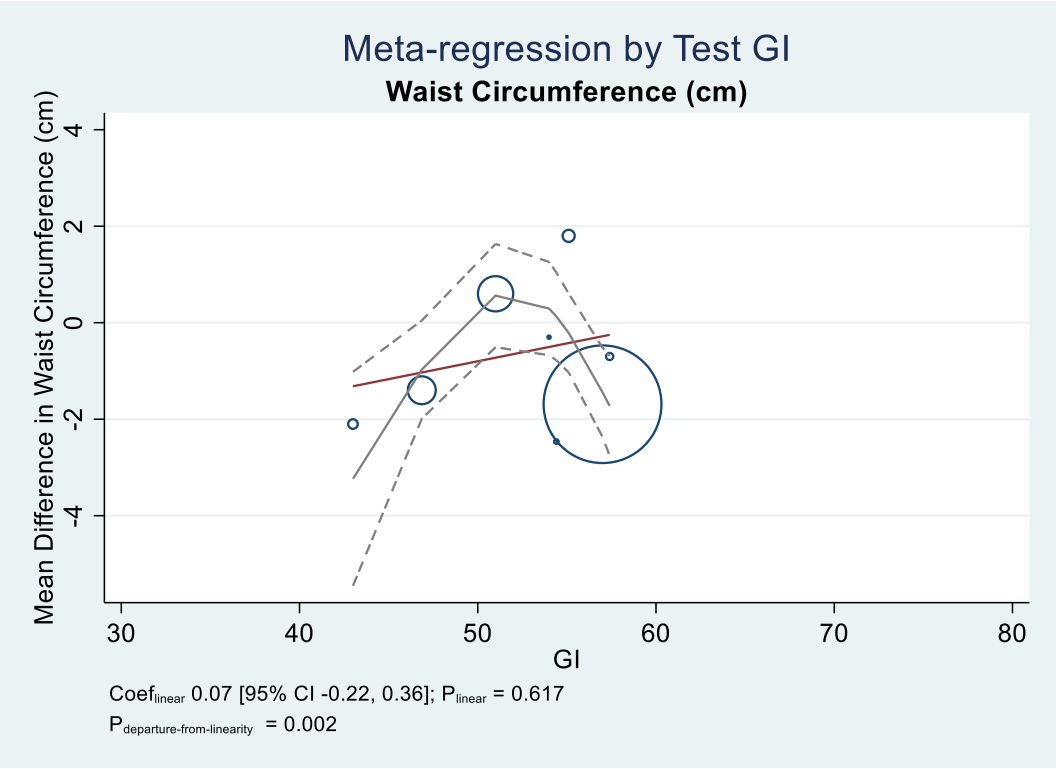
C


Meta-regression by Test GI
BMI (kg/m²)
Mean Difference in BMI (kg/m²)
4
2
0
-2
-4
30
40
50
60
70
80
GI
Coef_linear -0.00 [95% CI -0.07, 0.06]; P_linear = 0.943
P_departure-from-linearity = 0.375

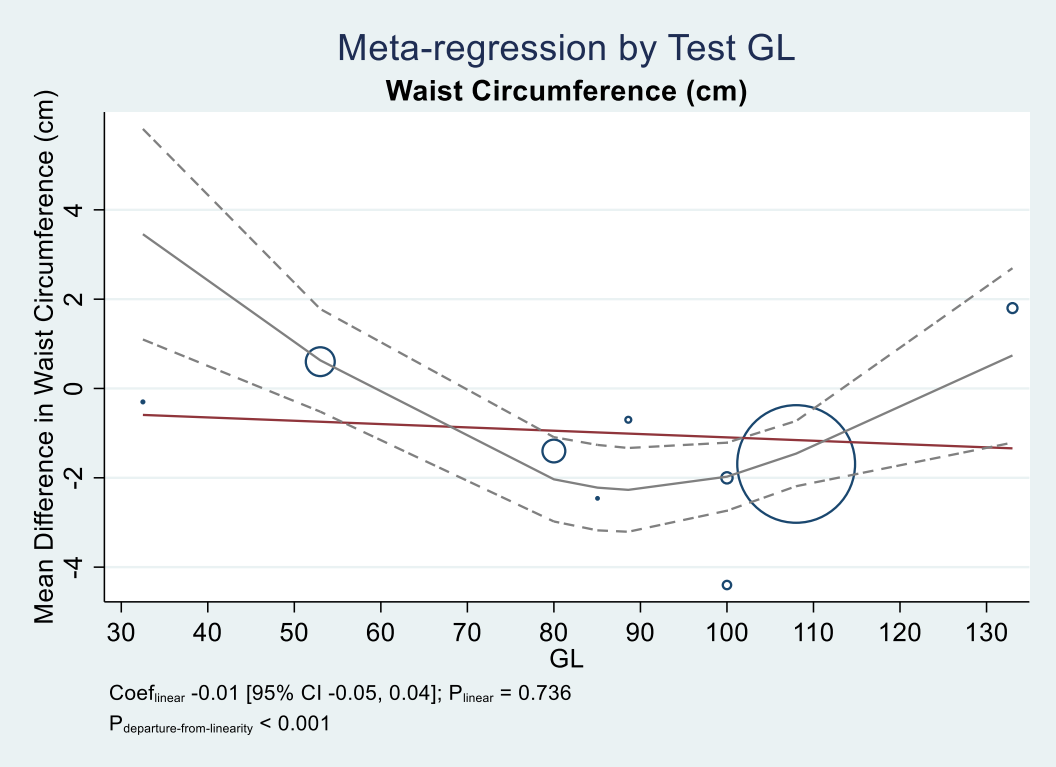
D


Meta-regression by Test GL
BMI (kg/m²)
Mean Difference in BMI (kg/m²)
4
2
0
-2
-4
30
40
50
60
70
80
90
100
110
120
130
GL
Coef_linear 0.01 [95% CI -0.01, 0.03]; P_linear = 0.363
P_departure-from-linearity = 0.169

E


Meta-regression by Test GI
Waist Circumference (cm)
Mean Difference in Waist Circumference (cm)
-4
-2
0
2
4
30
40
50
60
70
80
GI
Coef$_{linear}$ 0.07 [95% CI -0.22, 0.36]; P$_{linear}$ = 0.617
P$_{departure\text{-}from\text{-}linearity}$ = 0.002

F

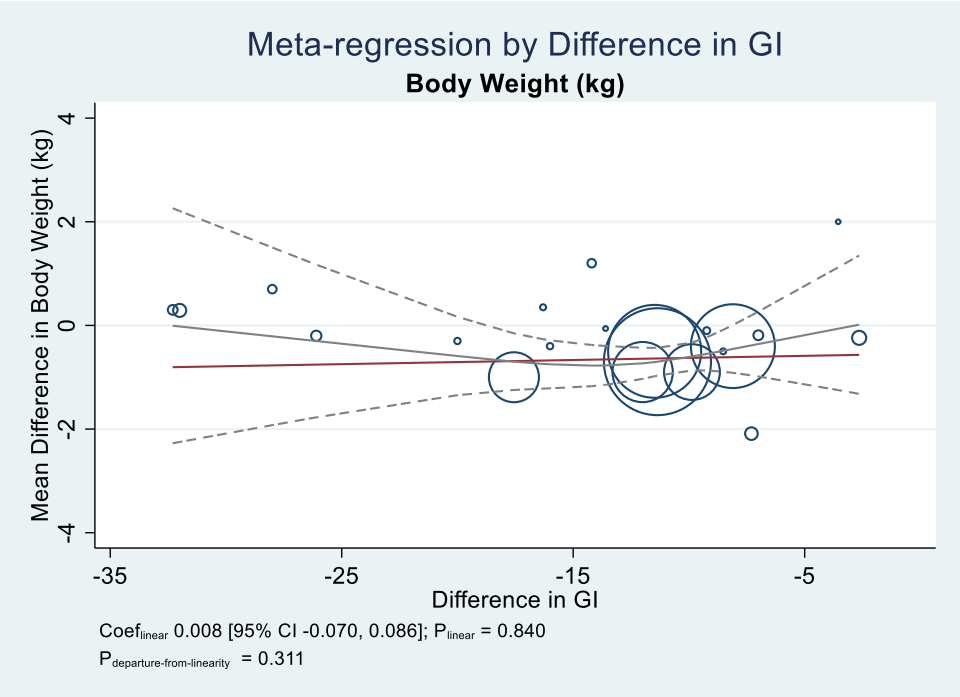

Meta-regression by Test GL
Waist Circumference (cm)
Mean Difference in Waist Circumference (cm)
-4
-2
0
2
4
30
40
50
60
70
80
90
100
110
120
130
GL
Coef$_{linear}$ -0.01 [95% CI -0.05, 0.04]; P$_{linear}$ = 0.736
P$_{departure\text{-}from\text{-}linearity}$ < 0.001

Individual studies are represented by the circles, with their weight in the overall analysis represented by the size of the circles. The straight red line represents the estimate linear dose response and the grey line the non-linear dose response for the GI of the diet and the dashed lines represent the upper and lower 95% confidence intervals. Linear and non-linear dose response analyses are presented for: **A**, Test GI and body weight; **B**, Test GL and body weight; **C**, Test GI and BMI; **D**, Test GL and BMI; **E**, Test GI and waist circumference; **F**, Test GL and waist circumference.

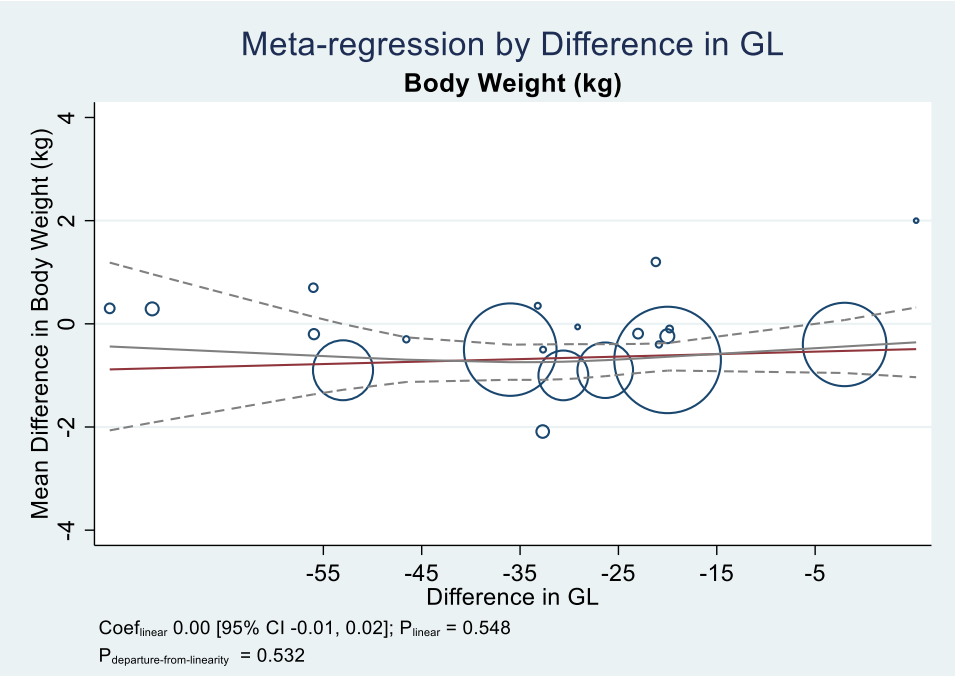
BMI, body mass index; CI, confidence interval; Coef, coefficient; GI, glycemic index; GL, glycemic load; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S68: Linear and non-linear meta-regression analyses for the effect of low-GI/GL dietary patterns by difference in GI or GL between the intervention and control groups on adiposity in diabetes

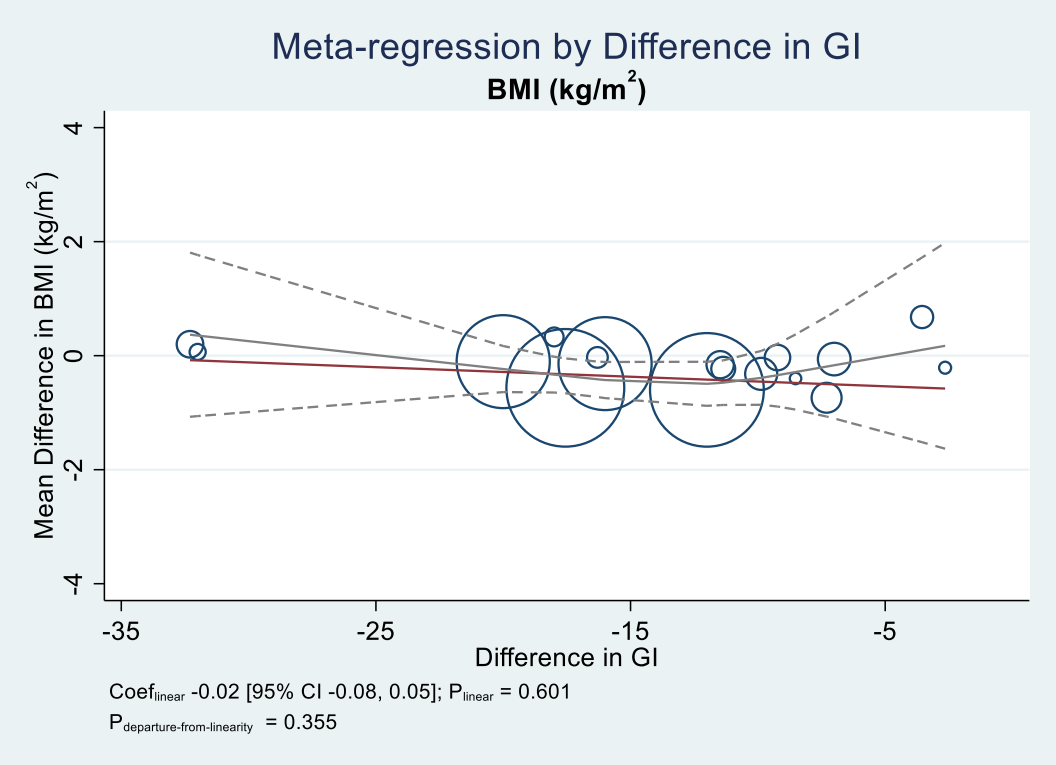
A



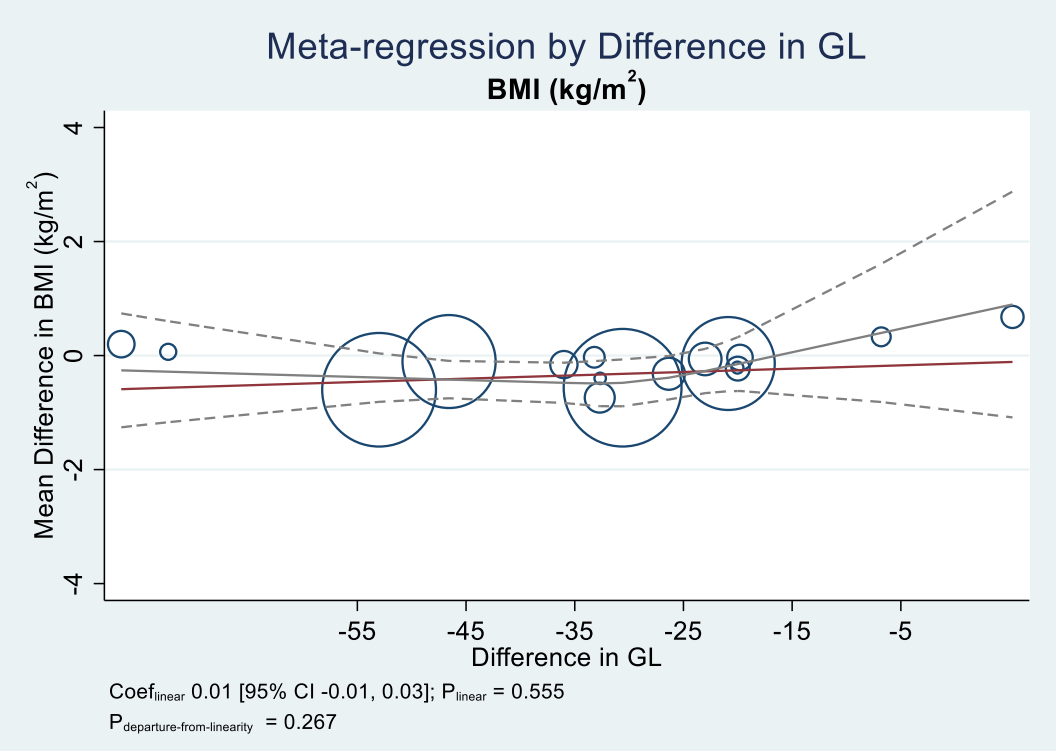
B



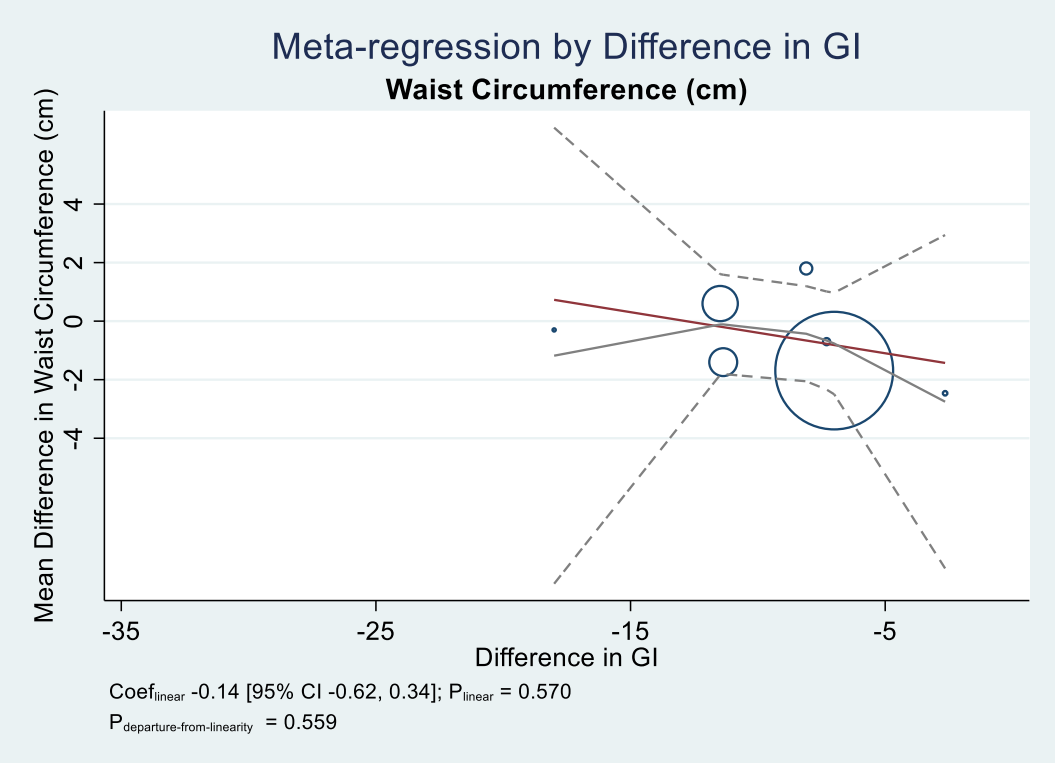
C


Meta-regression by Difference in GI
BMI (kg/m²)
Mean Difference in BMI (kg/m²)
4
2
0
-2
-4
-35
-25
-15
-5
Difference in GI
Coef_linear -0.02 [95% CI -0.08, 0.05]; P_linear = 0.601
P_departure-from-linearity = 0.355

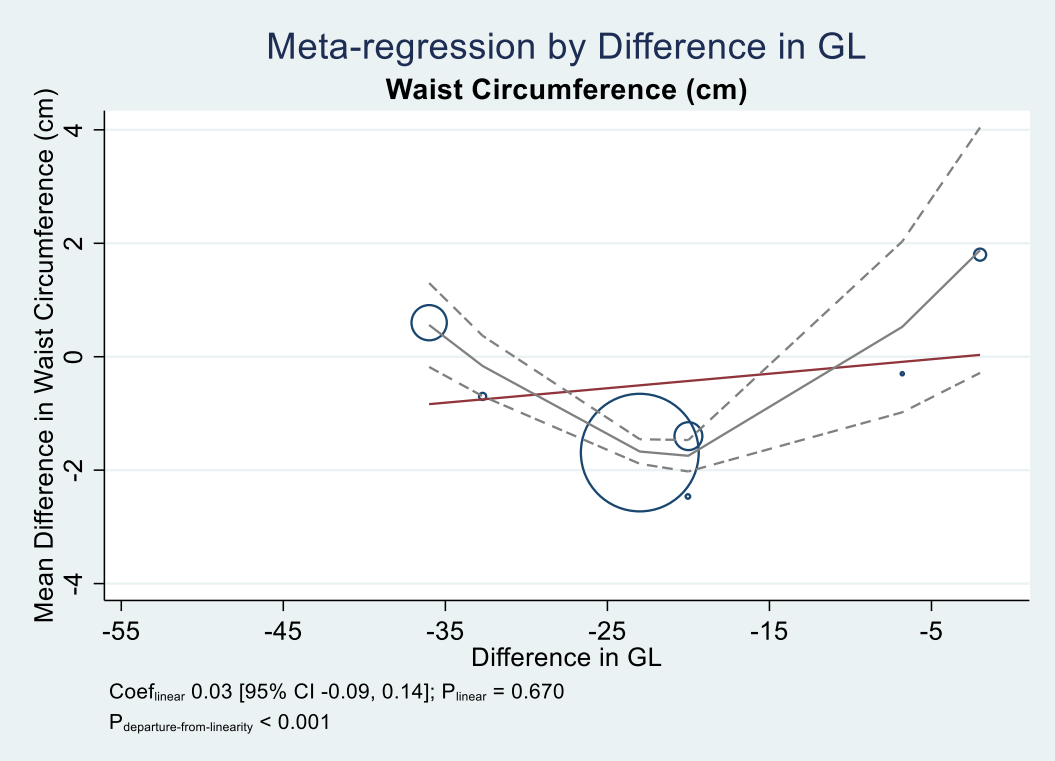
D


Meta-regression by Difference in GL
BMI (kg/m²)
Mean Difference in BMI (kg/m²)
4
2
0
-2
-4
-55
-45
-35
-25
-15
-5
Difference in GL
Coef_linear 0.01 [95% CI -0.01, 0.03]; P_linear = 0.555
P_departure-from-linearity = 0.267

E


Meta-regression by Difference in GI
Waist Circumference (cm)
Mean Difference in Waist Circumference (cm)
-4
-2
0
2
4
-35
-25
-15
-5
Difference in GI
Coef$_{linear}$ -0.14 [95% CI -0.62, 0.34]; P$_{linear}$ = 0.570
P$_{departure\text{-}from\text{-}linearity}$ = 0.559

F

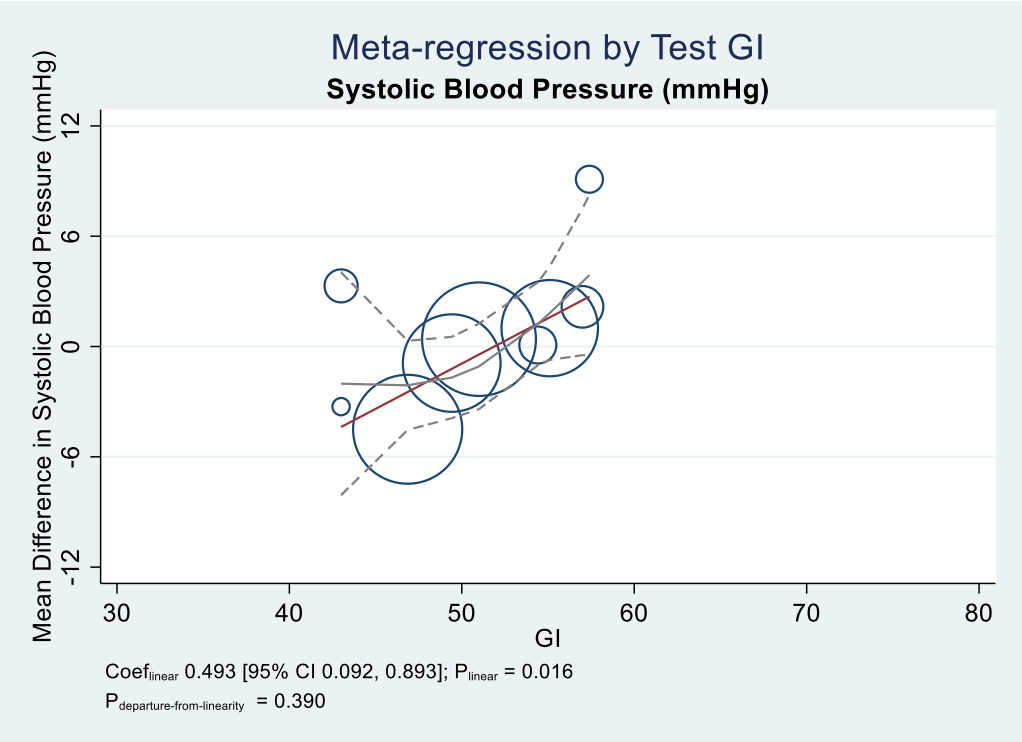

Meta-regression by Difference in GL
Waist Circumference (cm)
Mean Difference in Waist Circumference (cm)
-4
-2
0
2
4
-55
-45
-35
-25
-15
-5
Difference in GL
Coef$_{linear}$ 0.03 [95% CI -0.09, 0.14]; P$_{linear}$ = 0.670
P$_{departure\text{-}from\text{-}linearity}$ < 0.001

Individual studies are represented by the circles, with their weight in the overall analysis represented by the size of the circles. The straight red line represents the estimate linear dose response and the grey line the non-linear dose response for the GI of the diet and the dashed lines represent the upper and lower 95% confidence intervals. Linear and non-linear dose response analyses are presented for: **A**, Difference in GI and body weight; **B**, Difference in GL and body weight; **C**, Difference in GI and BMI; **D**, Difference in GL and BMI; **E**, Difference in GI and waist circumference; **F**, Difference in GL and waist circumference. Note "Difference in" denotes difference in either GI or GL between the low-GI/GL and control diets (test – control) during the interventions, so that negative numbers denote the magnitude of reductions in GI/GL.

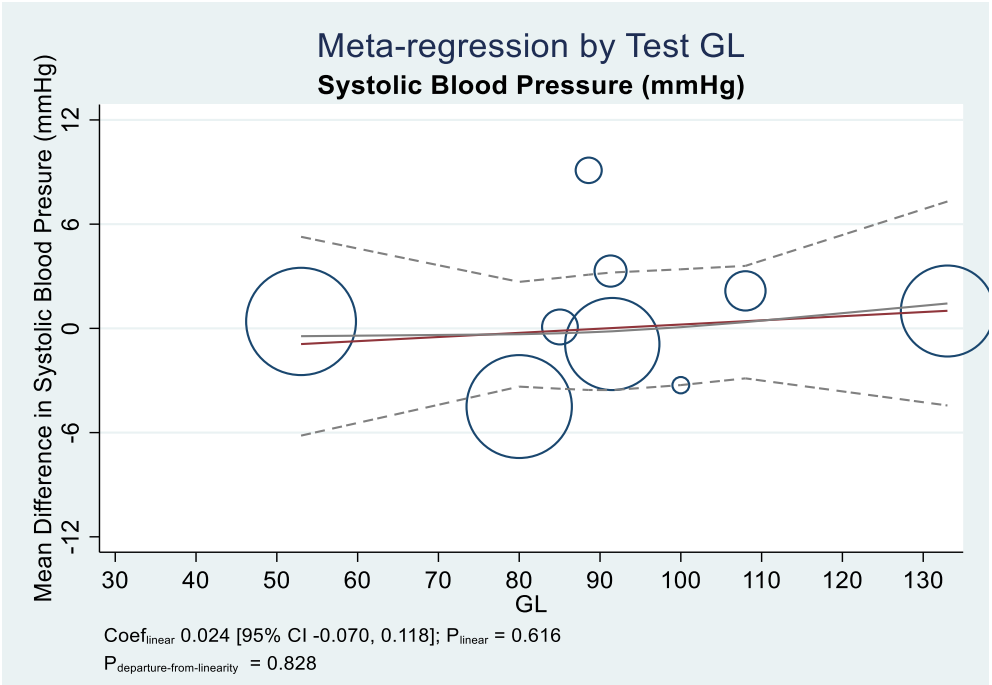
BMI, body mass index; CI, confidence interval; Coef, coefficient; GI, glycemic index; GL, glycemic load

Supplemental Figure S69: Linear and non-linear meta-regression analyses for the effect of low-GI and GL intervention dose on blood pressure in diabetes

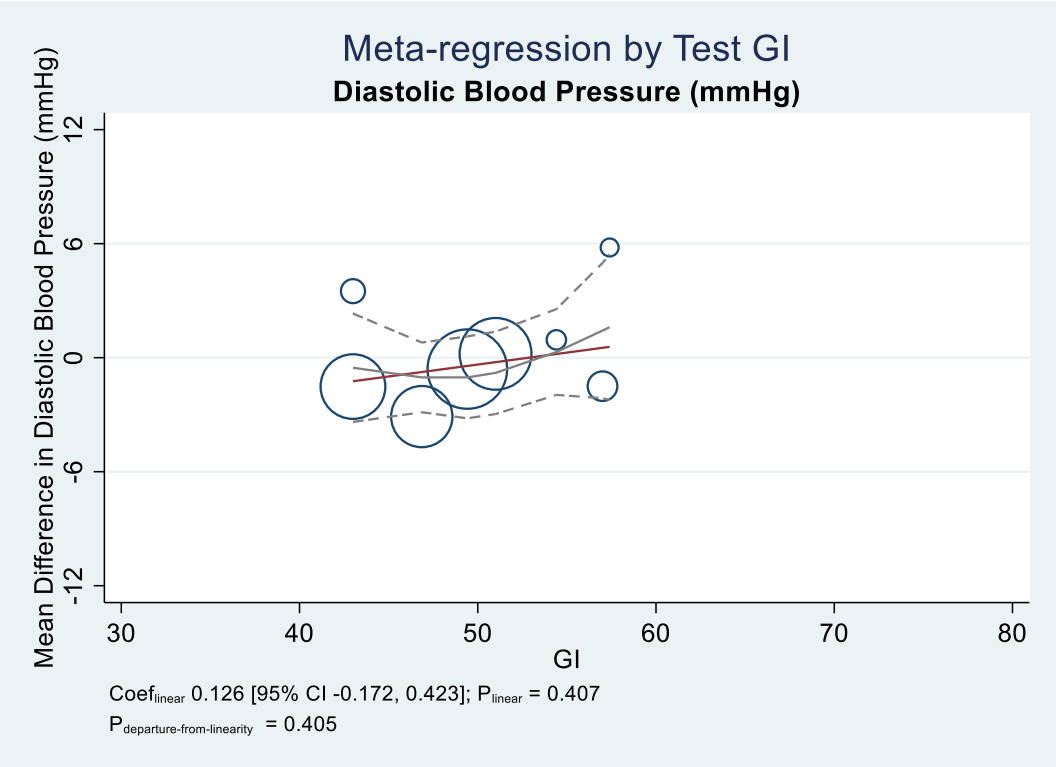
A



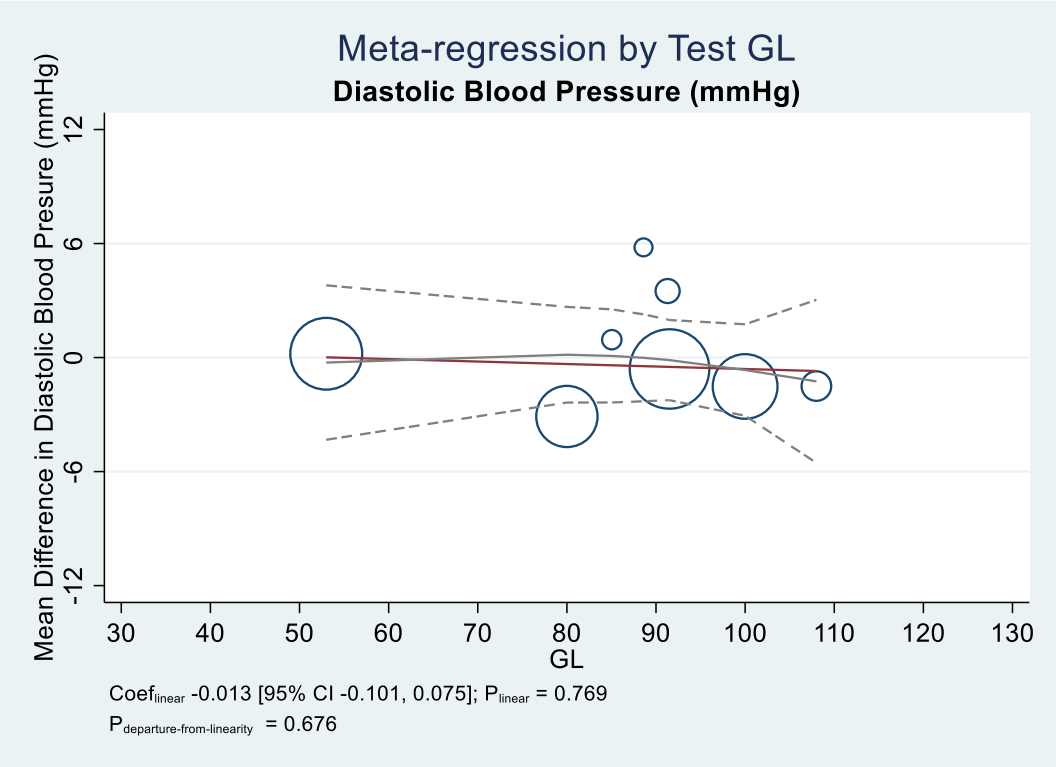
B



C


Meta-regression by Test GI
Diastolic Blood Pressure (mmHg)
Mean Difference in Diastolic Blood Pressure (mmHg)
-12
-6
0
6
12
30
40
50
60
70
80
GI
Coef$_{linear}$ 0.126 [95% CI -0.172, 0.423]; P$_{linear}$ = 0.407
P$_{departure\text{-}from\text{-}linearity}$ = 0.405

D

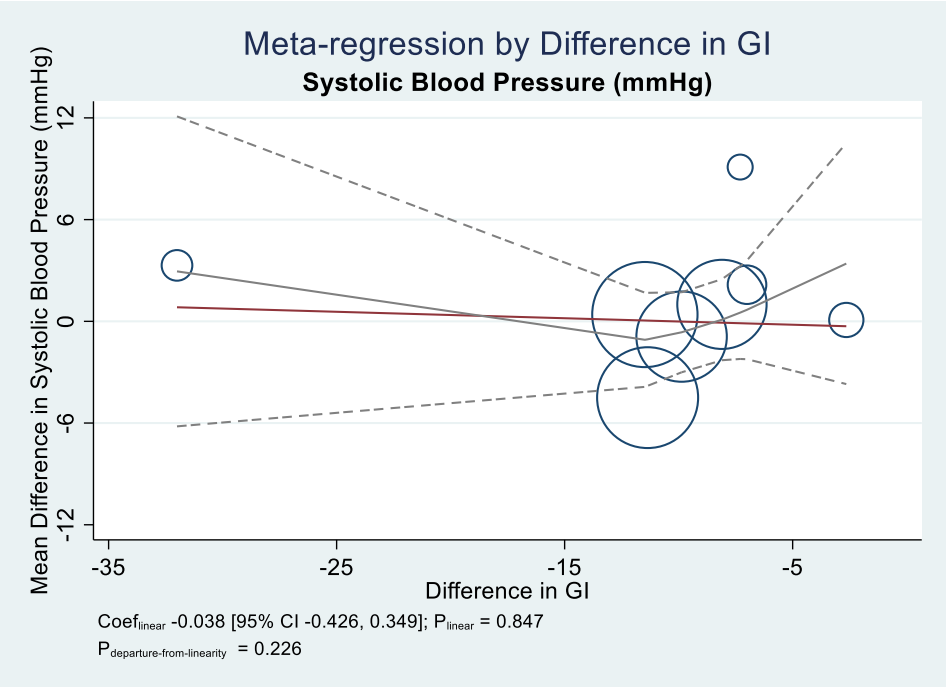

Meta-regression by Test GL
Diastolic Blood Pressure (mmHg)
Mean Difference in Diastolic Blood Presure (mmHg)
-12
-6
0
6
12
30
40
50
60
70
80
90
100
110
120
130
GL
Coef$_{linear}$ -0.013 [95% CI -0.101, 0.075]; P$_{linear}$ = 0.769
P$_{departure\text{-}from\text{-}linearity}$ = 0.676

Individual studies are represented by the circles, with their weight in the overall analysis represented by the size of the circles. The straight red line represents the estimate linear dose response and the grey line the non-linear dose response for the GI of the diet and the dashed lines represent the upper and lower 95% confidence intervals. Linear and non-linear dose response analyses are presented for: **A**, Test GI and systolic blood pressure; **B**, Test GL and systolic blood pressure; **C**, Test GI and diastolic blood pressure; **D**, Test GL and diastolic blood pressure.

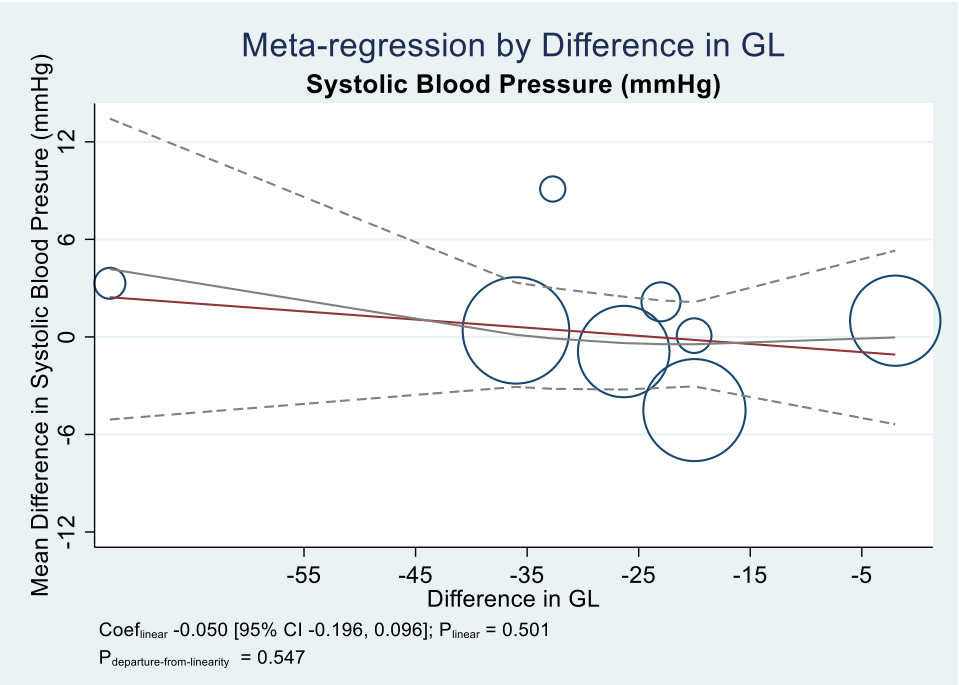
CI, confidence interval; Coef, coefficient; GI, glycemic index; GL, glycemic load; Test GI, the prescribed or in-trial achieved dietary GI on the low-GI/GL diet; Test GL, the prescribed or in-trial achieved dietary GL on the low-GI/GL diet

Supplemental Figure S70: Linear and non-linear meta-regression analyses for the effect of low-GI/GL dietary patterns by difference in GI or GL between the intervention and control groups on blood pressure in diabetes

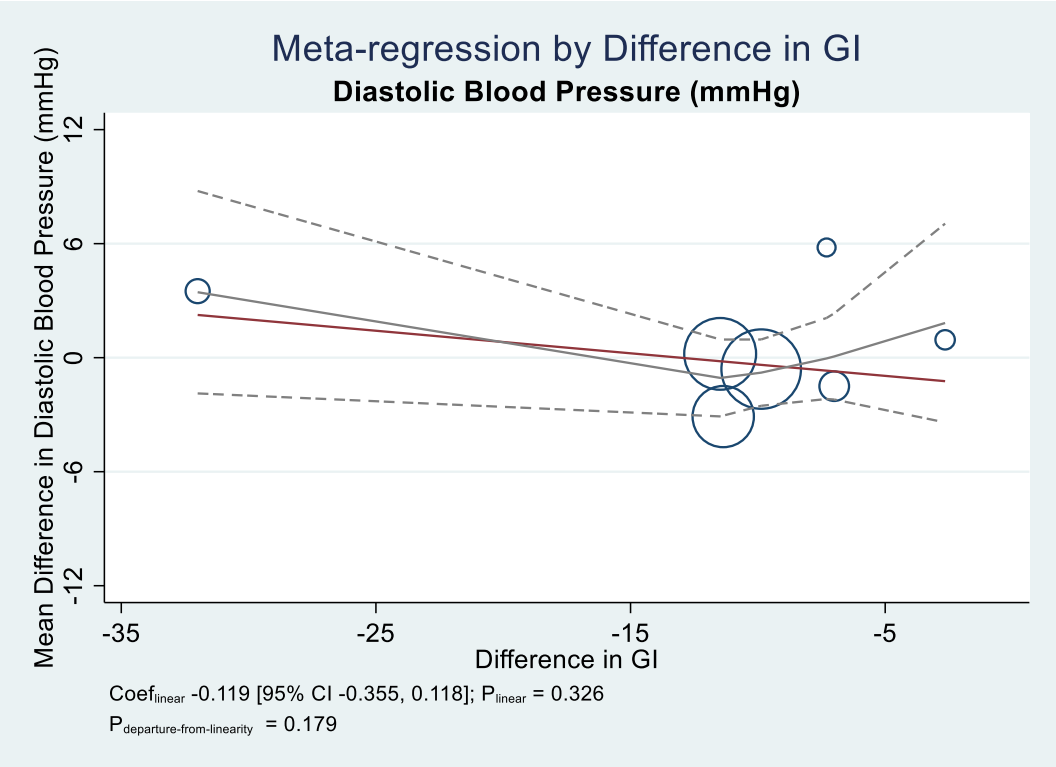
A



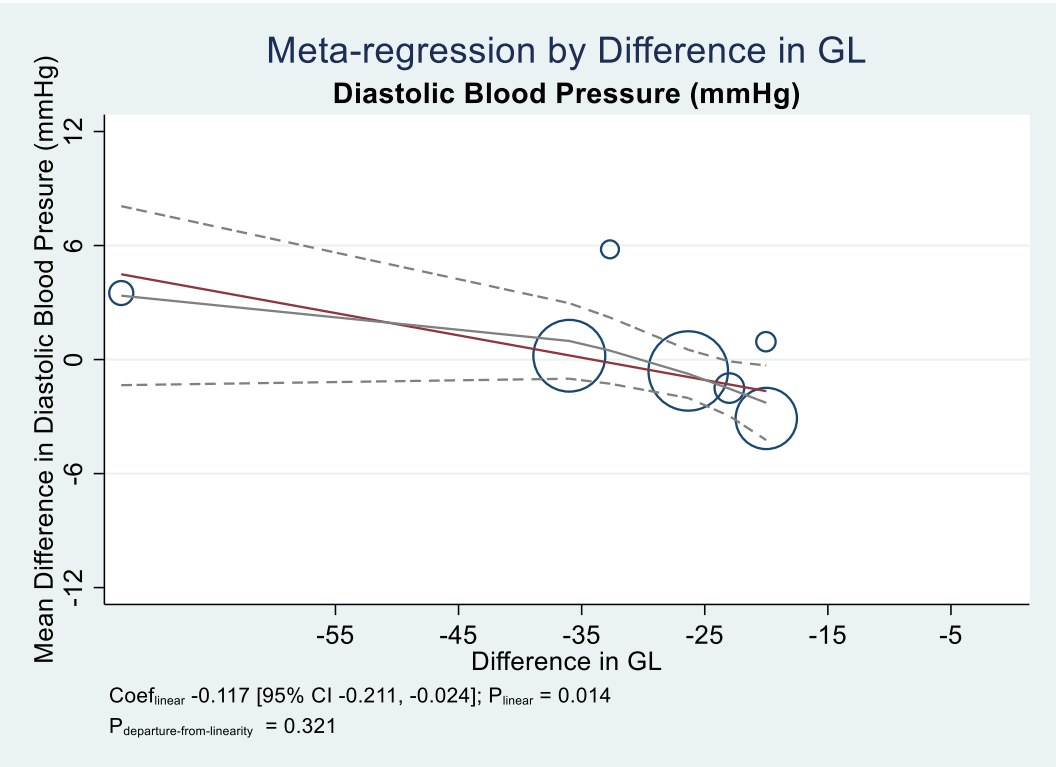
B



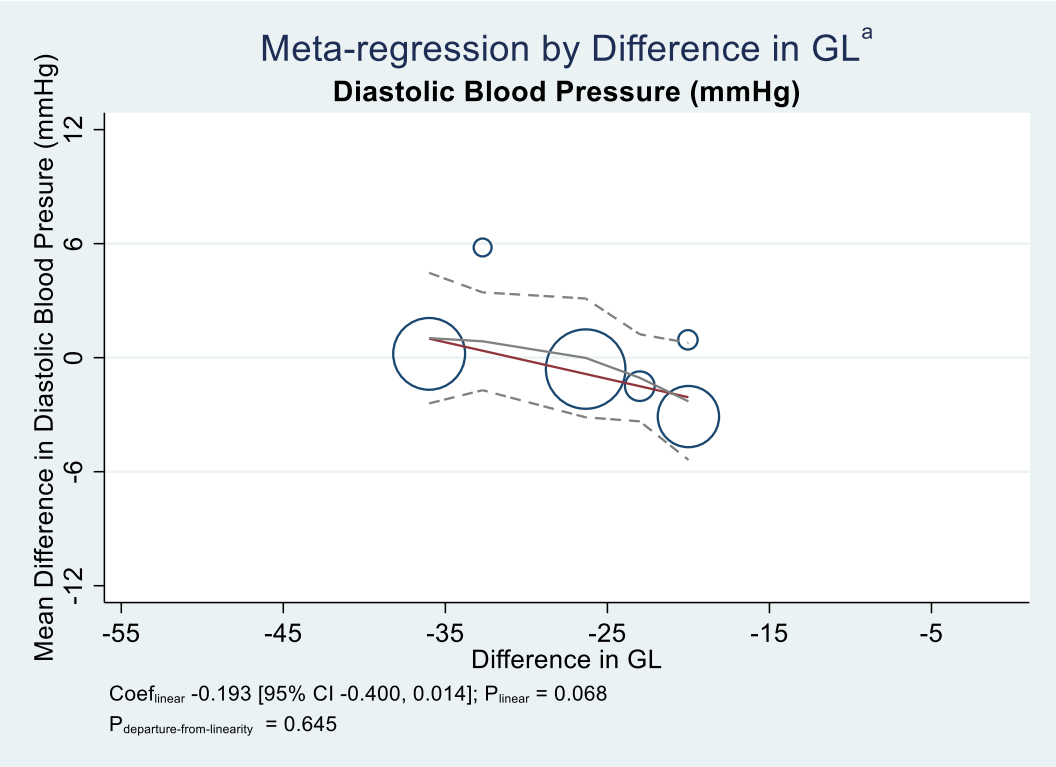
C


Meta-regression by Difference in GI
Diastolic Blood Pressure (mmHg)
Mean Difference in Diastolic Blood Pressure (mmHg)
12
6
0
-6
-12
-35
-25
-15
-5
Difference in GI
Coef$_{linear}$ -0.119 [95% CI -0.355, 0.118]; P$_{linear}$ = 0.326
P$_{departure\text{-}from\text{-}linearity}$ = 0.179

D


Meta-regression by Difference in GL
Diastolic Blood Pressure (mmHg)
Mean Difference in Diastolic Blood Presure (mmHg)
12
6
0
-6
-12
-55
-45
-35
-25
-15
-5
Difference in GL
Coef$_{linear}$ -0.117 [95% CI -0.211, -0.024]; P$_{linear}$ = 0.014
P$_{departure\text{-}from\text{-}linearity}$ = 0.321

E



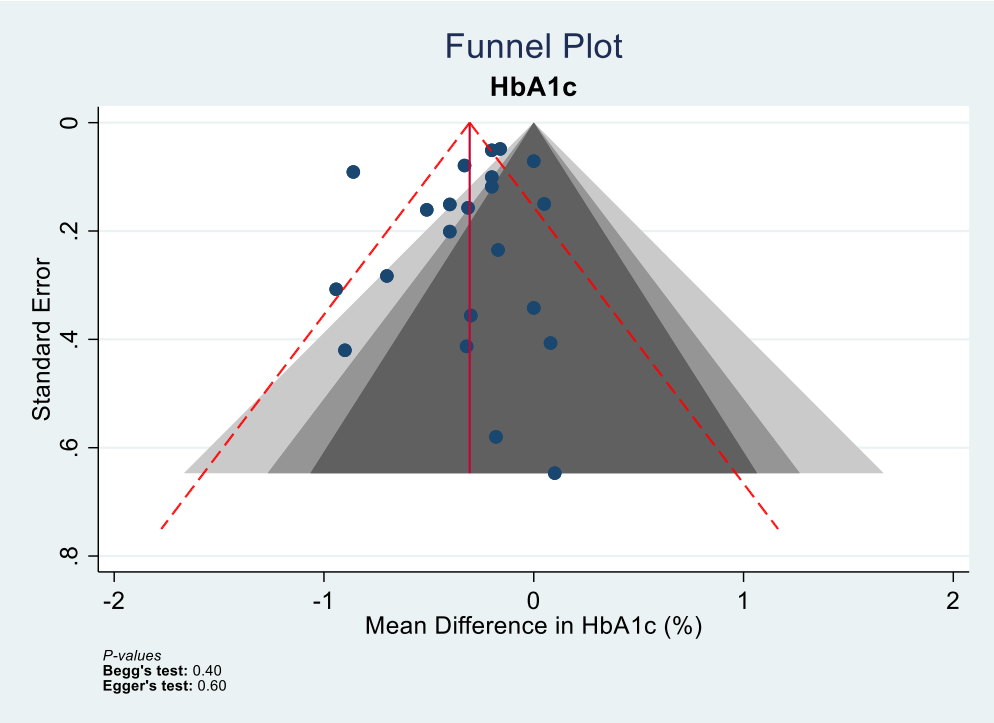
[a] meta-regression by difference in GL with removal of a single extreme outlier of exposure (Heilbronn et al. 2002)

Individual studies are represented by the circles, with their weight in the overall analysis represented by the size of the circles. The straight red line represents the estimate linear dose response and the grey line the non-linear dose response for the GI of the diet and the dashed lines represent the upper and lower 95% confidence intervals. Linear and non-linear dose response analyses are presented for: **A**, Difference in GI and systolic blood pressure; **B**, Difference in GL and systolic blood pressure; **C**, Difference in GI and diastolic blood pressure; **D**, Difference in GL and diastolic blood pressure; **E**, sensitivity analysis of Difference in GL and diastolic blood pressure after removal of an extreme exposure outlier. Note "Difference in" denotes difference in either GI or GL between the low-GI/GL and control diets (test – control) during the interventions, so that negative numbers denote the magnitude of reductions in GI/GL.

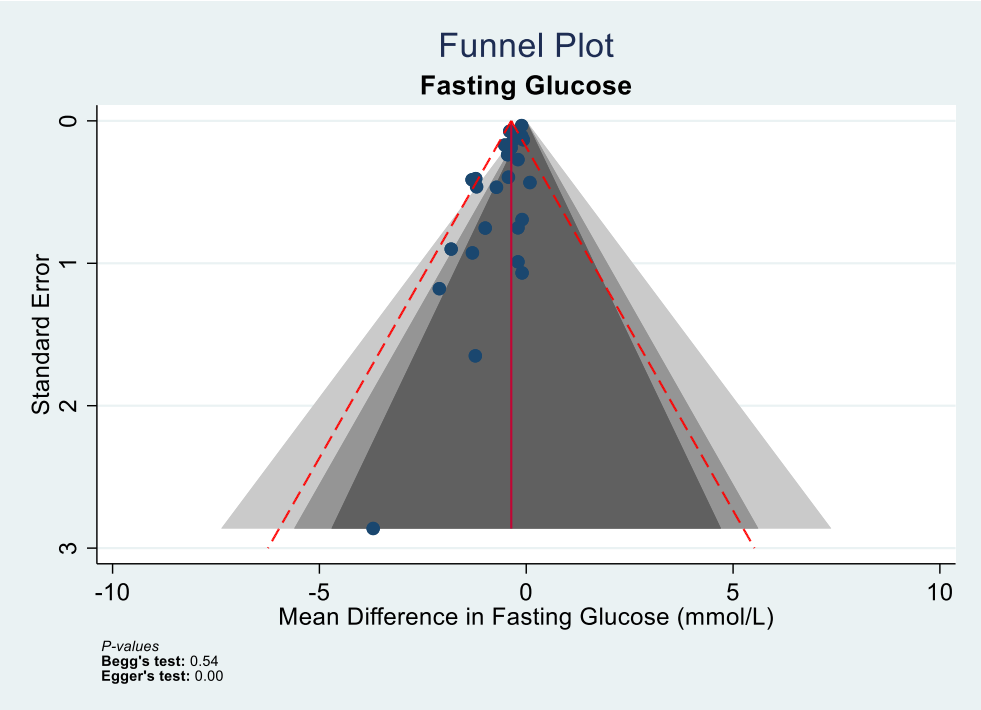
CI, confidence interval; Coef, coefficient; GI, glycemic index; GL, glycemic load

Supplemental Figure S71: Publication bias funnel plots for the effect of low-GI/GL dietary patterns on glycemic control in diabetes

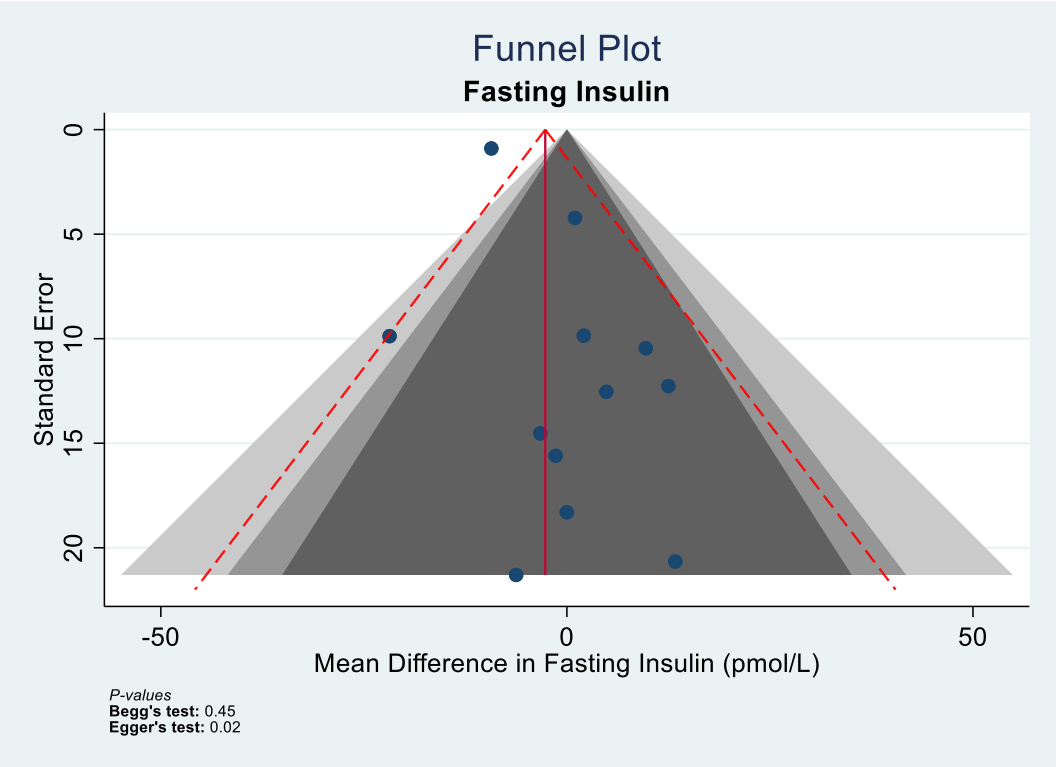
A



B



C

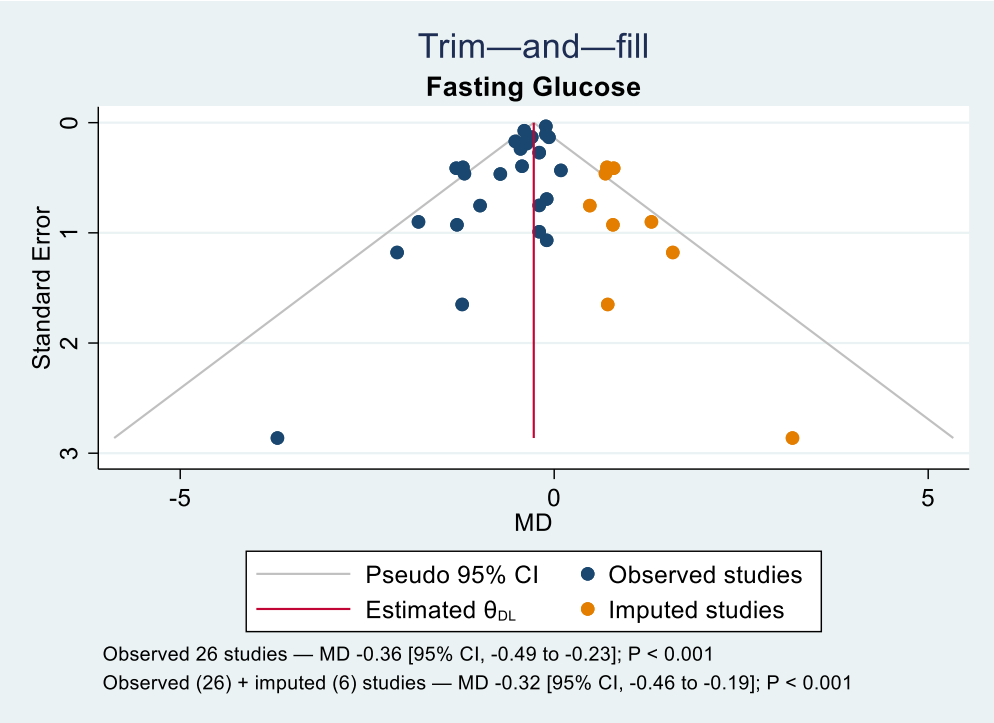


Contour-enhanced funnel plot is a scatter-plot of each trial comparison weighted mean difference (MD) on the x-axis with the standard error (SE) representing precision on the y-axis. The vertical solid red line represents the pooled effect estimate and the dashed red lines represent the pseudo-95% confidence limits. The blue dots represent individual trial comparisons. The contour regions define the regions for the test of significance of individual study effect size for a given p-value range >0.1 (dark grey), 0.5 to <0.1 (medium grey), 0.01 to <0.5 (light grey), <0.01 (white)]. The contour-enhanced funnel plots may suggest funnel-plot asymmetry is due to publication bias when less precise (smaller) studies are missing in the non-significant regions. Quantitative assessment of publication bias was also performed using Egger's and Begg's tests set at a significance level of p<0.05. Funnel plots are presented for: **A**, HbA1c; **B**, fasting glucose; **C**, fasting insulin.

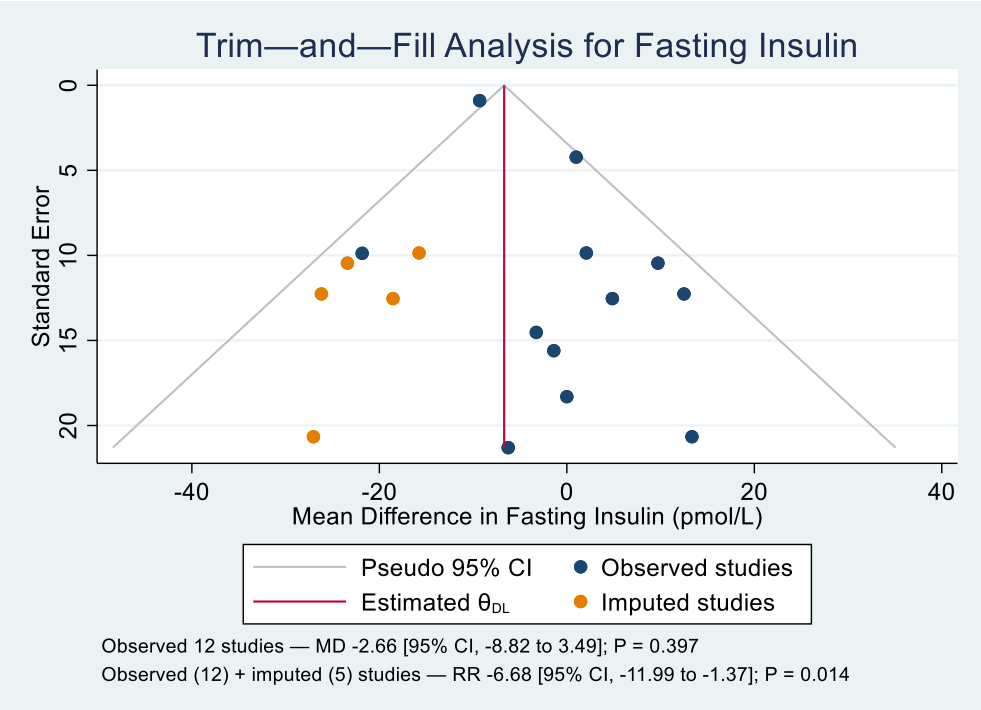
CI, confidence interval

Supplemental Figure S72: Trim and Fill analysis for the effect of low-GI/GL dietary patterns on fasting glucose (mmol/L) and insulin (pmol/L) in diabetes

A



B

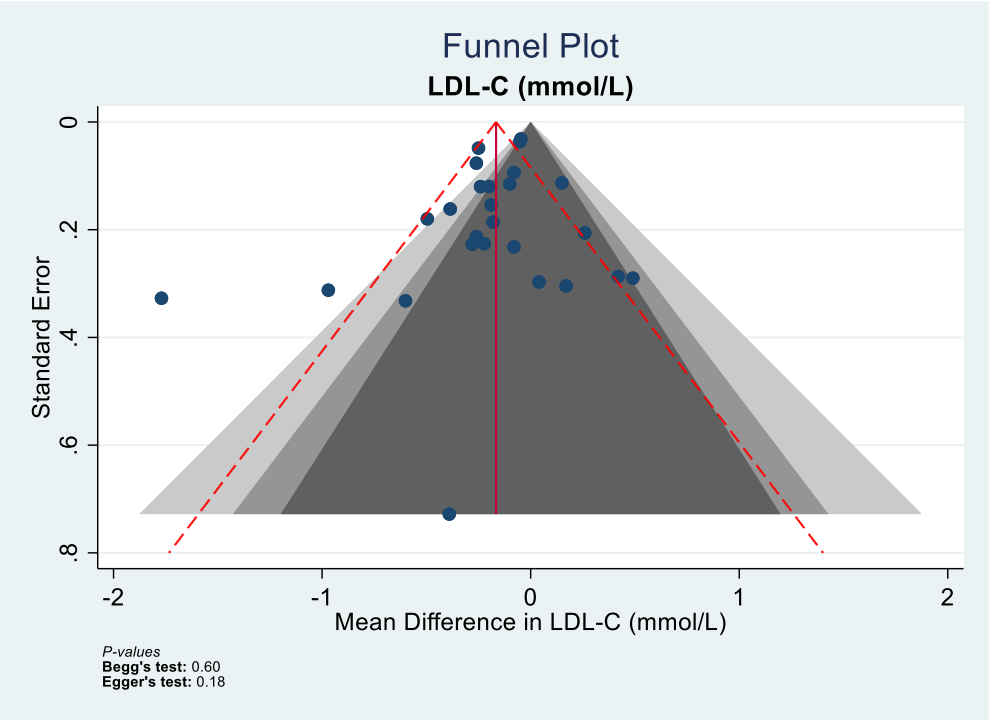


The vertical line represents the pooled effect estimate expressed as mean difference. The diagonal lines represent the pseudo-95% confidence limits, the blue circles represent the effect estimate for each included study, and orange circles represent the effect estimate for each imputed “missed” study. Imputed random mean difference is provided; when the imputed result differs from the primary result in either significance or magnitude (>1 MID =5pmol/L for fasting insulin), this is considered evidence of small-study effects. Trim-and-fill analyses are presented for: **A**, fasting glucose; **B**, fasting insulin.

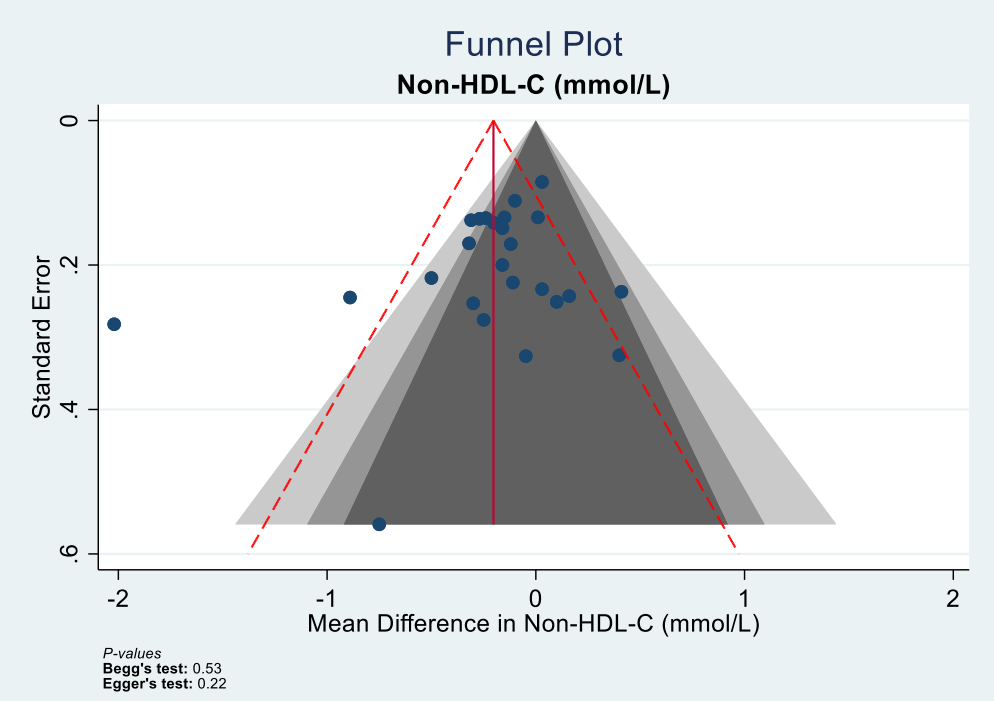
CI, confidence interval

Supplemental Figure S73: Publication bias funnel plots for the effect of low-GI/GL dietary patterns on blood lipids in diabetes

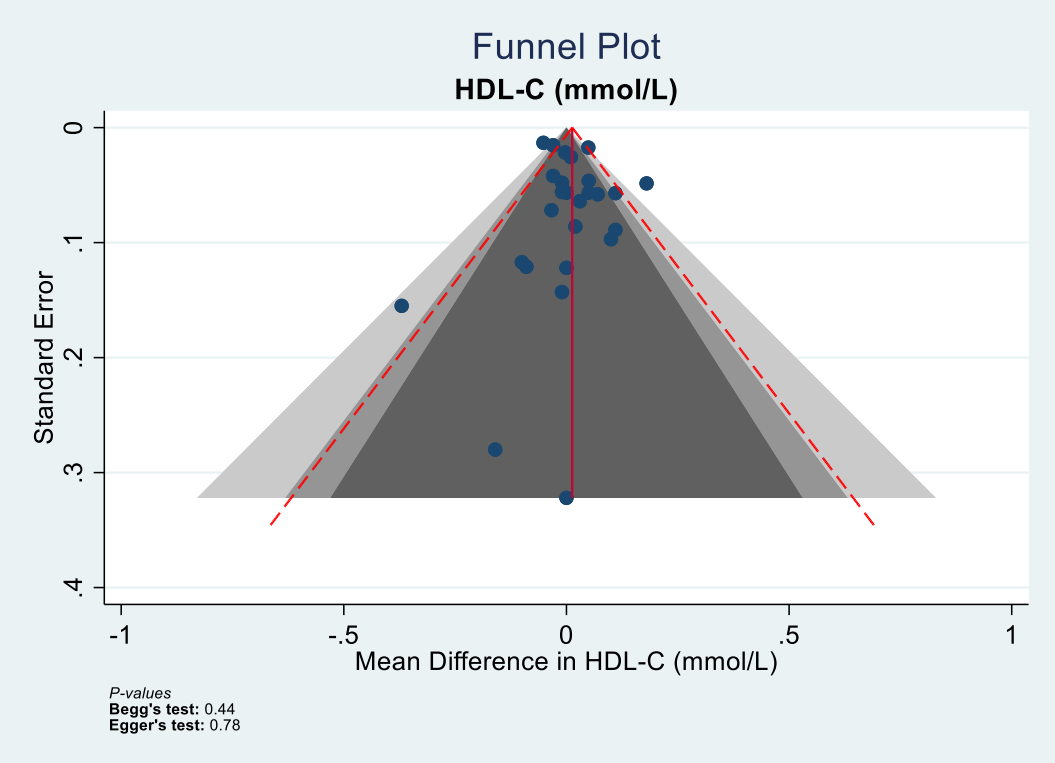
A



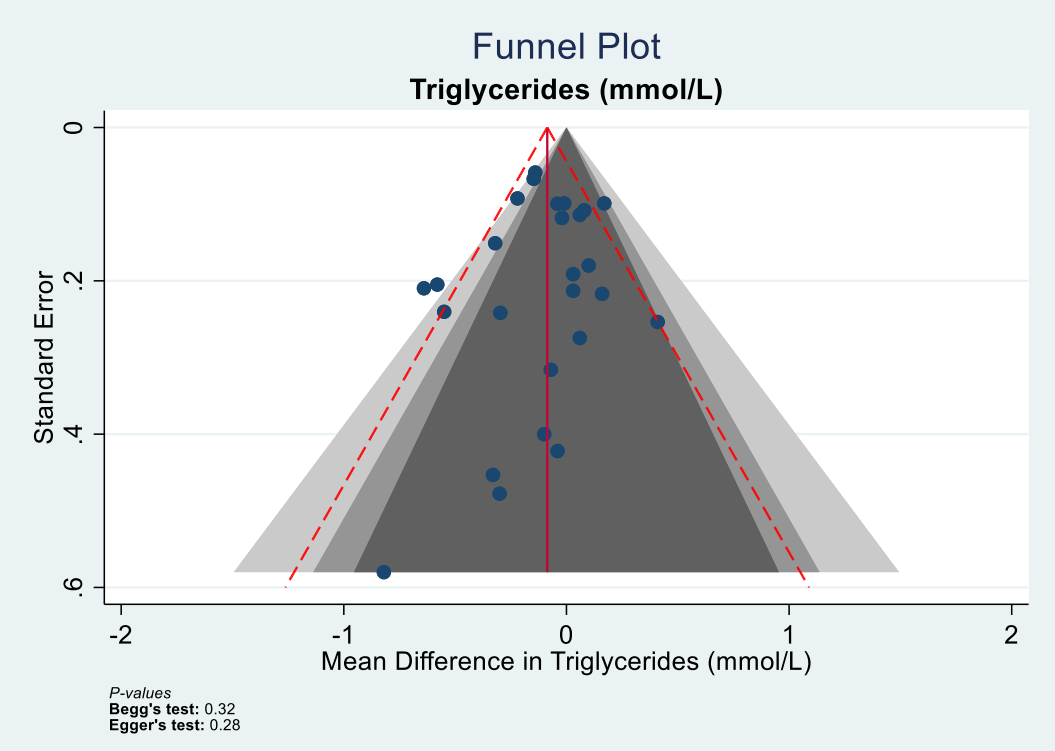
B



C


Funnel Plot
HDL-C (mmol/L)
Standard Error
0
.1
.2
.3
.4
-1
-.5
0
.5
1
Mean Difference in HDL-C (mmol/L)
P-values
Begg's test: 0.44
Egger's test: 0.78

D

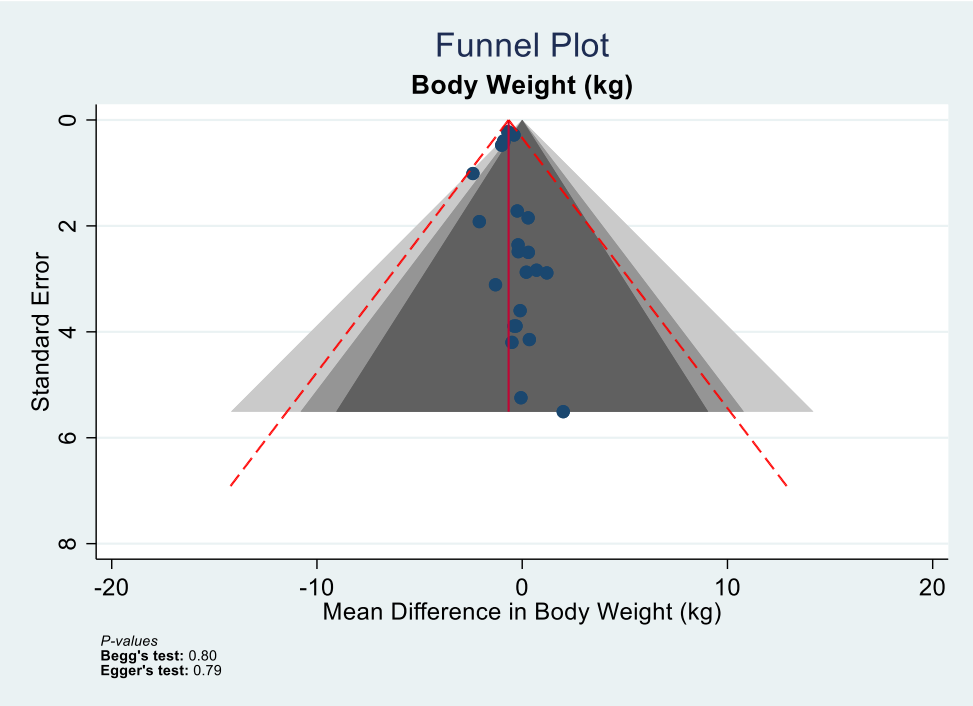

Funnel Plot
Triglycerides (mmol/L)
Standard Error
0
.2
.4
.6
-2
-1
0
1
2
Mean Difference in Triglycerides (mmol/L)
P-values
Begg's test: 0.32
Egger's test: 0.28

Contour-enhanced funnel plot is a scatter-plot of each trial comparison weighted mean difference (MD) on the x-axis with the standard error (SE) representing precision on the y-axis. The vertical solid red line represents the pooled effect estimate and the dashed red lines represent the pseudo-95% confidence limits. The blue dots represent individual trial comparisons. The contour regions define the regions for the test of significance of individual study effect size for a given p-value range >0.1 (dark grey), 0.5 to <0.1 (medium grey), 0.01 to <0.5 (light grey), <0.01 (white)]. The contour-enhanced funnel plots may suggest funnel-plot asymmetry is due to publication bias when less precise (smaller) studies are missing in the non-significant regions. Quantitative assessment of publication bias was also performed using Egger's and Begg's tests set at a significance level of $p<0.05$. Funnel plots are presented for: **A**, LDL-C; **B**, non-HDL-C; **C**, HDL-C; **D**, triglycerides. Note that publication bias was not assessed apoB as <10 trial comparisons were available (n=5).

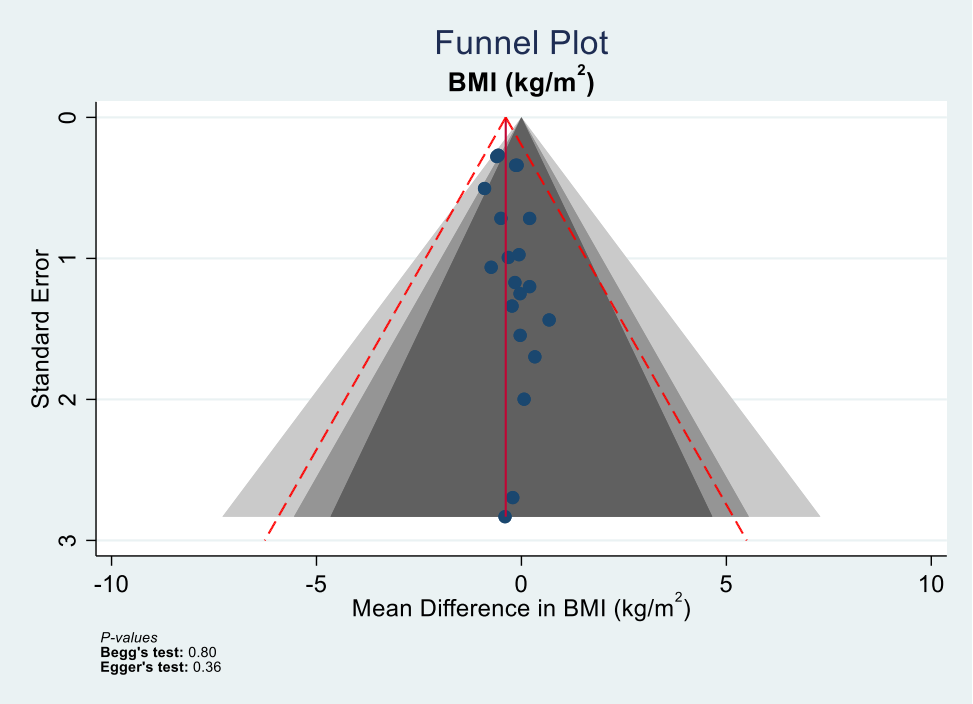
CI, confidence interval; HDL-C, high density lipoprotein-cholesterol; LDL-C, low density lipoprotein-cholesterol; Non-HDL-C, non-high-density lipoprotein-cholesterol

Supplemental Figure S74: Publication bias funnel plots for the effect of low-GI/GL dietary patterns on adiposity in diabetes

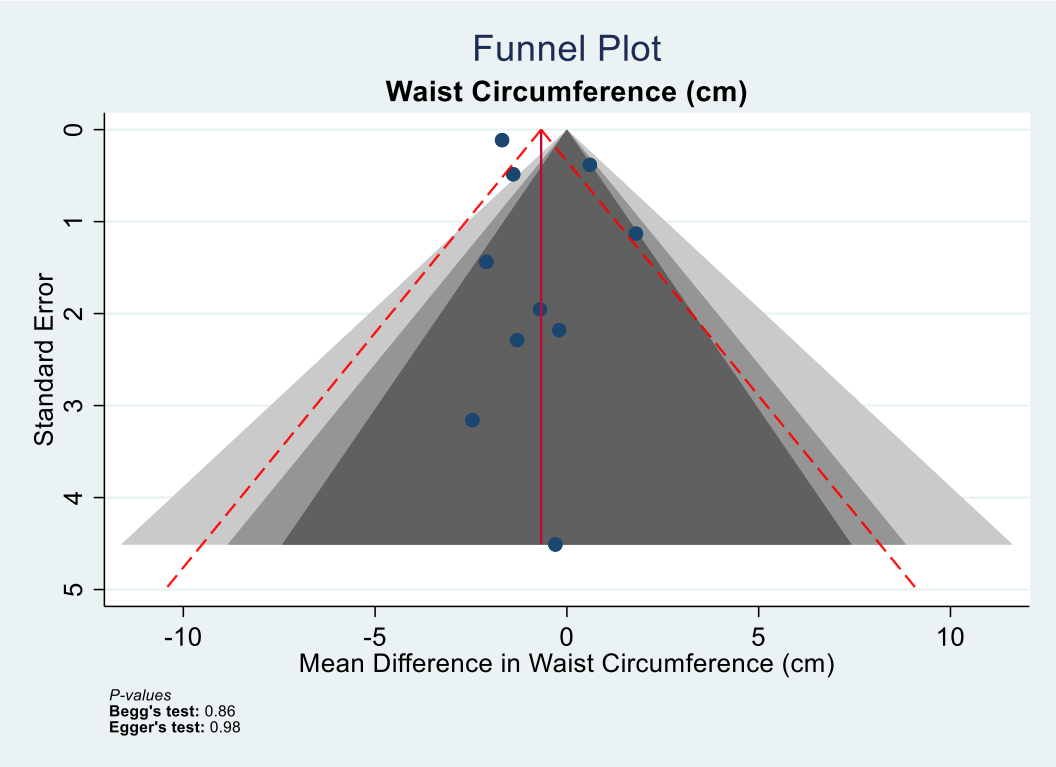
A



B



C



Contour-enhanced funnel plot is a scatter-plot of each trial comparison weighted mean difference (MD) on the x-axis with the standard error (SE) representing precision on the y-axis. The vertical solid red line represents the pooled effect estimate and the dashed red lines represent the pseudo-95% confidence limits. The blue dots represent individual trial comparisons. The contour regions define the regions for the test of significance of individual study effect size for a given p-value range >0.1 (dark grey), 0.5 to <0.1 (medium grey), 0.01 to <0.5 (light grey), <0.01 (white)]. The contour-enhanced funnel plots may suggest funnel-plot asymmetry is due to publication bias when less precise (smaller) studies are missing in the non-significant regions. Quantitative assessment of publication bias was also performed using Egger's and Begg's tests set at a significance level of p<0.05. Funnel plots are presented for: **A**, body weight; **B**, BMI; **C**, waist circumference.

BMI, body mass index; CI, confidence interval