BMJ Open

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Journal:	*BMJ Open*
Manuscript ID	bmjopen-2017-019438
Article Type:	Research
Date Submitted by the Author:	31-Aug-2017
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Primary Subject Heading:	Nutrition and metabolism
Secondary Subject Heading:	Cardiovascular medicine
Keywords:	body weight, pasta, glycemic index, glycaemic index, systematic review and meta-analysis, weight loss

Effect of Pasta in the Context of Low Glycemic Index Dietary Patterns on Body Weight and Markers of Global Adiposity: A Systematic Review and Meta-analysis of Randomized Controlled Trials in Adults

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Word Count: 4,816

Tables: 1

Figures: 3

Online only supplementary material included

Keywords: body weight, pasta, glycemic index, glycaemic index, systematic review and meta-analysis, weight loss

ABSTRACT

Objective: Carbohydrates have been implicated in the epidemic of obesity. To assess the effect of pasta alone or pasta in the context of low glycemic index (GI) dietary patterns on adiposity, we conducted a systematic review and meta-analysis.

Design: Systematic review and meta-analysis of randomized controlled trials (RCTs) with GRADE assessment.

Eligibility criteria for selecting studies: MEDLINE, Embase, CINAHL, and the Cochrane Library were searched through 07 February 2017. We included RCTs of ≥3-weeks assessing the effect of pasta alone or pasta in the context of low-GI dietary patterns on measures of global (body weight, BMI, body fat) and regional (waist circumference [WC], waist-to-hip ratio [WHR], sagittal abdominal diameter [SAD]) adiposity in adults. Two independent reviewers extracted data and assessed risk of bias. Data were pooled using the generic inverse-variance method and expressed as mean differences (MDs) with 95% confidence intervals (95% CIs). Heterogeneity was assessed (Cochran Q statistic) and quantified (I2-statistic). GRADE assessed the overall quality of the evidence.

Results: We identified no trials of the effect of pasta alone and 32 trial comparisons (n=2,448 participants) of the effect of pasta in the context of low-GI dietary patterns. Pasta in the context of low-GI dietary patterns significantly reduced body weight (MD=-0.63kg [95% CIs, -0.84, -0.42kg]) and BMI (MD=-0.26kg/m2 [95% CIs, -0.36, -0.16kg/m2]) compared with higher-GI dietary patterns. There was no effect on other measures of adiposity. The evidence was graded as

moderate for body weight, BMI, WHR, and SAD owing to a downgrade for indirectness and low for WC and body fat owing to downgrades for indirectness and inconsistency.

Conclusions: The available RCTs demonstrate that pasta in the context of low-GI dietary patterns does not adversely affect adiposity. Future trials should assess the effect of pasta in the context of other healthy dietary patterns**.**

Protocol registration: ClinicalTrials.gov Identifier, NCT02961088

Strengths and limitations of this study

- The present systematic review and meta-analysis was based on a comprehensive search and include a large number of randomized controlled trials which provide the best protection against bias
- We used the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) system to evaluate the strength and quality of the evidence.
- There was evidence of unexplained inconsistency in the treatment estimates across trials for waist circumference and body fat.
- The generalizability of our results is questionable with evidence of indirectness in the pooled estimates for all body and adiposity outcomes, as all of the trials assessed pasta in the context of low-GI dietary patterns (no trials assessed the effect of pasta alone or in the context of other dietary patterns) and most of the available trials did not quantify the amount of pasta consumed.

INTRODUCTION

As the role of saturated fat in chronic disease has been called into question, carbohydrates have come under attack in the media[1,2], popular books[3-9], statements of health advocacy groups[10] and commentaries in leading medical journals[11,12]. Much of the attention has focused on sugars but traditional carbohydrate staples like pasta, rice, and breads are increasingly being implicated in the epidemics of overweight and obesity[1,7]. Although advantages for weight related outcomes have been shown for dietary patterns that are high in these foods but low in glycemic index (GI)[13,14], high in whole grains[15,16], and/or high in dietary fibre[17,18], there has been a general lack of recognition of the importance of carbohydrate quality.

Pasta is an important example of a food which is considered a refined carbohydrate but has a low-GI, a property that has been exploited extensively in studies of low-GI dietary patterns. It remains unclear whether pasta alone or in the context of a low-GI dietary pattern contributes to weight gain. We undertook a systematic review and meta-analysis of randomized controlled trials (RCTs) with a full GRADE assessment to quantify the effect of pasta alone or in the context of low-GI dietary patterns on body weight and measures of adiposity relevant to the prevention and management of overweight and obesity.

METHODS

Design

Our protocol followed the guideline of the Cochrane Handbook for Systematic Reviews of Interventions[19], and findings are reported according to the guidelines of the Preferred Reporting

Items for Systematic Reviews and Meta-Analyses[20]. The protocol is registered at clinicaltrials.gov (identifier, NCT02961088).

Data sources and searches

We searched MEDLINE (http://www.nlm.nih.gov/bsd/pmresources.html), Embase (https://www.embase.com), CINAHL (https://health.ebsco.com/products/the-cinahl-database), and the Cochrane Library (http://www.cochranelibrary.com/) through 07 February 2017. The full search terms used in this study are presented in **Supplemental Tables S1-S2**. Briefly, we searched using variations of the terms pasta and glycemic index and glycemic load and body weight and BMI. The search was limited to human studies and had no language restrictions. Reference lists of selected studies and reviews were also searched to identify additional articles.

Study selection

We include RCTs that investigated the effect of pasta consumed alone or in the context of low-GI dietary patterns that emphasized pasta in comparison with higher GI diets that did not include pasta on body weight or other measures of global (BMI, body fat) or abdominal (waist circumference, waist-to-hip ratio, sagittal abdominal diameter, or visceral adipose tissue assessed by imaging modalities) adiposity in participants of all health backgrounds. Trials were excluded if they had diet duration of <3 weeks, did not intend to use a calorie- and macronutrient-matched comparator arm that was higher in GI; included pregnant or breast-feeding women or children; or did not provide suitable end-point data. When multiple publications existed for the same study, the article with the most information was included (n=6). Published abstracts were not included.

Data extraction

Two reviewers (LC, CB) assessed the titles and abstracts of all identified studies and independently reviewed and extracted relevant data from each report, including study design, blinding, sample size, participant characteristics, follow-up duration, identification of pasta in the low-GI diet only, comparator diet, macronutrient profile, funding source, and outcome data. The primary outcome was body weight, and secondary outcomes included markers of global (BMI, body fat) and abdominal (waist circumference, waist-to-hip ratio, sagittal abdominal diameter, or visceral adipose tissue assessed by imaging modalities) adiposity. Mean±SD differences between test and control arms were extracted for each outcome.

In those trials where the data were included in figures and not provided numerically, we used the software program Plot Digitizer (http://plotdigitizer.sourceforge.net/) to extract the data. Additional information was requested from the authors of all included trials. Disagreement were resolved by consensus or where necessary by a third author (SBM).

Risk of bias assessment

Risk of bias for each individual study was assessed using the Cochrane Risk of Bias tool[19]. The level of bias was evaluated for sequence generation, allocation concealment, blinding, incomplete outcome data, and selective reporting and determined overall as either low (proper methods taken to reduce bias), high (improper methods creating bias), or unclear (insufficient information provided to determine the bias level).

Statistical analysis

Baseline, end-of-study, and between-treatment differences in body weight, BMI, waist circumference, body fat, waist-to-hip ratio, sagittal abdominal diameter or visceral adipose tissue assessed by imaging modalities were recorded as means±SDs. If not provided, between-treatment differences in change-from-baseline or end differences were calculated by subtracting means and variance measures such as SEs were imputed with the use of published formulas[19]. Missing SDs were imputed with the use of the pooled SD from other studies included in the analysis[19].

Data analyses were conducted using Review Manager version 5.3 (RevMan) (Copenhagen, Denmark: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014) for primary analyses and Stata version 13 (College Station, TX: StataCorp LP) for subgroup analyses. A generic inverse-variance method with random-effects models was used to calculate pooled mean differences and 95% confidence intervals (CIs). Random-effects models were used even in the absence of statistically significant inter-study heterogeneity, as they yield more conservative summary effect estimates in the presence of residual heterogeneity. Change-from-baseline differences were preferred over end differences and paired analyses were applied to all crossover trials with the use of a within-individual correlation coefficient between treatments of 0.5 as described by Elbourne et al.[21]

Inter-study heterogeneity was assessed by the Cochran Q statistic, where $P<0.10$ was considered statistically significant, and quantified by the I^2 statistic, where $I^2 \geq 50\%$ indicates substantial heterogeneity[19]. Sensitivity analysis were performed which included the removal of each single study from the meta-analyses one at a time and recalculation of the summary effect. An influential outlier was considered a study whose removal changed the magnitude of the pooled effect by >10%. Sensitivity analysis were also conducted using different correlation coefficient

values for crossover trials (0.25 and 0.75) to test for the robustness of the effect size, conducting analyses using fixed effects models and restricting analyses to those trials for which pasta intake could be quantified.

If ≥10 trial comparisons were available, then sources of heterogeneity were explored by subgroup analyses. A priori categorical subgroups analyses were assessed by meta-regression analyses. These included patient type (normal body weight, overweight or obese [average baseline BMI >27kg/m^2]), diabetes, coronary heart disease), follow-up (<24-weeks, ≥24 weeks), baseline BMI (BMI ≤30, >30kg/m^2), design (parallel, crossover), energy balance (negative on both arms [weight loss diets], neutral on both arms [weight maintaining diets]) and dose of pasta (based on the median). A priori categorical subgroup analyses also included the following dietary factors: GI (absolute level [≤55, >55; glucose scale], within-treatment change, between-treatment change), fat intake (absolute level [<30%, ≥30% energy], within-treatment change, between-treatment change), carbohydrate intake (absolute level [<50%, ≥50% energy], within-treatment change, between-treatment change), protein intake (absolute level [<20%, ≥20% energy], within-treatment change, between-treatment change), dietary fibre intake (absolute level [<28g/day, ≥28g/day], within-treatment change, between-treatment change), and risk of bias. A priori continuous meta-regression analyses were conducted on the absolute levels and within- and between-treatment changes of these same dietary factors in the intervention arms of pasta in the context of low-GI dietary patterns. Linear and non-linear pasta intake dose-response analyses were assessed by using continuous meta-regression analyses and spline curve modeling (MKSPLINE procedure), respectively. Publication bias was assessed by visual inspection of funnel plots and the Egger[22] and Begg[23] tests, when ≥10 trial comparisons were available. If

publication bias was suspected, then adjustment for funnel plot asymmetry was done by imputing missing study data using the Duval and Tweedie trim and fill method [24].

Grading the evidence

The grading of recommendations assessment, development, and evaluation (GRADE) approach was used to assess the confidence in the pooled effect estimates by assessing the overall quality and strength of the evidence [25]. Evidence was graded as high, moderate, low or very low quality. The included RCTs were graded as high quality evidence by default and downgraded based on pre-specified criteria. Criteria to downgrade evidence included risk of bias (weight of studies show risk of bias assessed by the Cochrane Risk of Bias tool), inconsistency (substantial unexplained heterogeneity, I^2>50%, P<0.01), indirectness (presence of factors that limited the generalizability of the results), imprecision (the 95% CI for effect estimates were wide or crossed pre-specified minimally important differences [MIDs] for harm), and publication bias (significant evidence of small-study effects).

Patient involvement

No patients were directly involved in the development of the research question, selection of the outcome measures, design and implementation of the study, or interpretation of the results.

RESULTS

Search results

Figure 1 shows the flow of the literature. We identified 4876 reports of which 29 met eligibility criteria. No reports were identified that assessed the effect of pasta alone, while 29 reports (including 32 trial comparisons involving 2448 participants) were identified that assessed the effect of pasta in the context of low-GI dietary patterns on any adiposity outcome in adults[26-54]. Of the 32 trial comparisons that assessed the effect of pasta in the context of a low-GI dietary pattern, there were 32 trial comparisons for body weight, 18 trial comparisons for BMI[27,28,31-33,35,36,39-41,43-46,48,49,52,53], 18 trial comparisons for waist circumference[27,28,31,33,34,36,38-40,42,44-47,52,53], 10 trial comparisons for body fat[27,28,31-33,36,38,41,43,53], 6 trial comparisons for waist-to-hip ratio[31,39,40,44,45,52], and 3 trial comparisons for sagittal abdominal diameter[34,39]. There was only one trial comparison identified[35] for visceral adipose tissue assessed by imaging modalities, thus a meta-analysis could not be undertaken for this outcome.

Trial characteristics

Table 1 and **Supplemental Table S3** show the characteristics of all included trials of the effect of pasta in the context of low-GI dietary patterns. The majority of trials had a parallel design (26/32) with a median follow-up of 12 weeks (IQR: 9–21 weeks) and a median number of participants per trial of 43 (IQR: 21–112). Most participants were middle aged (median age: 50 y; IQR: 40-58 y) men and women (median ratio of men to women: 0.4:1 in available trials). The median baseline BMI across studies was 30.4kg/m^2 (IQR: 28.2–32.0). Regarding metabolic phenotype, 21 (66%) trials included participants who were overweight or obese (had a baseline BMI≥27kg/m^2), 10 (31%) had diabetes and one trial (3%) with coronary heart disease (CHD). We did not retrieve any trials where participants had a normal BMI at baseline (≤25kg/m^2),

although 6 trials did not include BMI >25 kg/m^2 as part of criteria, the average baseline BMI was ≥27 kg/m^2, therefore categorized as overweight.

Risk of bias

Supplemental Figures S1 and S2 show the individual Cochrane Risk of Bias tool assessments for each of the included trials of the effect of pasta in the context of low-GI dietary patterns. No serious risk of bias was detected.

Effect of Pasta in the Context of Low-GI dietary patterns and Body Weight

Figure 2 shows the effect of pasta in the context of low-GI dietary patterns on the primary outcome body weight. Pooled analyses showed pasta in the context of low-GI dietary patterns had the effect of reducing body weight by -0.63 kg (95% CI:-0.84, -0.42 kg; P<0.001) compared with higher GI control diets with no evidence of heterogeneity (I^2=0%, P-heterogeneity=0.51).

Effect of Pasta in the Context of Low-GI dietary patterns on Markers of Global Adiposity

Figure 3 and **Supplemental Figure S3-S4** show the effect of pasta in the context of low-GI dietary patterns on markers of global adiposity. Pooled analyses showed that pasta in the context of low-GI dietary patterns had the effect of reducing BMI by -0.26kg/m^2 (n=18 trials; MD=-0.26kg/m^2; 95% CI:-0.36, -0.16 kg/m^2; P<0.001) compared with higher GI control diets with no evidence of heterogeneity (I^2=0%, P-heterogeneity=0.90). There was no effect on body fat (n=10 trials; MD=-0.01%, 95% CI:-0.58, 0.56%; P=0.98) with evidence of substantial heterogeneity (I^2=65%, P<0.01).

Effect of Pasta in the Context of Low-GI dietary patterns on Markers of Abdominal Adiposity

Figure 3 and **Supplemental Figures S5-S7** show the pooled estimates for the markers of abdominal adiposity. Pooled analyses did not show a significant effect of pasta in the context of low-GI dietary patterns compared with higher-GI control diets on waist circumference (n=18 trials; MD=-0.46cm, 95% CI:-1.05, 0.14cm; P=0.13), waist-to-hip ratio (n=6 trials; MD=-0.00, 95% CI:-0.01, 0.00; P=0.27), or sagittal abdominal diameter (n=3 trials, MD=-0.09cm, 95% CI:-0.34, 0.16cm; P=0.48) There was only evidence of substantial heterogeneity in the analyses for waist circumference (I^2=62%, P-heterogeneity<0.01).

Sensitivity analyses

We conducted four sets of sensitivity analyses (**Supplemental Tables S4-5, Supplemental Figures S8-9**). The systematic removal of each trial did not modify the direction or significance of the effect estimates or the evidence of heterogeneity for any of the outcomes with the exception of waist circumference (**Supplemental Table S4**). In the sensitivity analysis for waist circumference, two studies were influential outliers in that their removal altered the magnitude of the pooled effect in the remaining studies by >10%, where the removal of the studies of McMillan-Price et al. (high protein comparison)[42] and Jenkins et al.[44] rendered the results for waist circumference statistically significant (MD= -0.62cm; 95% CI: -1.19, -0.05; P=0.03) and (MD= -0.61cm; 95% CI: -1.18, -0.04; P=0.04), respectively; forest plots not shown). Heterogeneity remained significant in both cases (I^2= 55%, P-heterogeneity <0.01 and I^2=50%, P-heterogeneity= 0.01, respectively). Sensitivity analyses using correlation coefficients of 0.25 and 0.75 for paired analyses of crossover trials also did not modify the results (**Supplemental**

Table S5). In the sensitivity analyses where fixed effects models were applied (**Supplemental Figure S8**), the direction, magnitude and significance of the pooled estimates were very similar to those produced by the random effects models with the exception of the sensitivity analysis for waist circumference which was significant (MD= -0.62; 95% CI: -0.93, -0.32; P<0.001). Finally, restricting analyses to the 11 trials for which pasta intake could be quantified (median pasta intake, 3.33 servings/week [range, 1.75 – 7 servings/week]) showed a similar reduction in body weight (MD= -0.70 kg; 95% CI:-1.10, -0.29 kg; P<0.001) when pasta was consumed in the context of low-GI dietary patterns compared with the higher GI control arms without evidence of heterogeneity (I^2=0%, P-heterogeneity=0.68) (**Supplemental Figure S9)**.

Subgroup analyses

We were only able to conduct a priori categorical and continuous subgroup analyses for body weight, BMI, body fat and waist circumference. Subgroup analyses for waist-to-hip ratio and sagittal abdominal diameter could not be assessed, owing to <10 trial comparisons in each case. **Supplemental Figures S10-S12** show the categorical a priori subgroup analyses for body weight. There was no evidence of significant effect modification in any of the subgroup analyses for body weight, including no effect modification of follow-up when comparing studies less than 24 weeks duration to those greater than or equal to 24 weeks (-0.63kg vs -0.57kg, respectively) (**Supplemental Figure S10**). Neither was there evidence of significant effect modification in any of the subgroup analyses for BMI, body fat or waist circumference (**Supplemental Figures S13-20**).

Supplemental Table S6 and **Supplemental Figures S21-22** show the continuous subgroup analyses for body weight. There was evidence of significant effect modification by carbohydrate and protein intake, where an increase in carbohydrate intake in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with weight loss (β= -0.07, 95% CI: -0.12, -0.01, I^2=0.00%, P=0.02), and an increase in protein intake in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with weight gain (β=0.15, 95% CI: 0.03, 0.27, I^2=0.00%, P=0.02). None of the other continuous subgroup analyses were significant. There was no evidence of significant effect modification in any of the continuous subgroup analyses for BMI (**Supplemental Table S7**). For body fat, there was evidence of significant effect modification in the continuous meta-regression subgroup analysis of difference in GI between intervention and control groups, where greater difference in GI between the groups was associated with greater reduction in body fat in the intervention group (β=-0.09, 95% CI: -0.15, -0.03, I^2=19.39%, P=0.01) (**Supplemental Table S8**). None of the other continuous subgroup analyses were significant. For waist circumference, there was evidence of significant effect modification in the continuous meta-regression subgroup analysis of absolute carbohydrate level and absolute protein level, where greater carbohydrate level in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with greater loss in waist circumference (β=-0.11, 95% CI: -0.19, -0.04, I^2=27.06%, P<0.01) and a lower protein level in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with an increase in waist circumference (β= 0.20, 95% CI: 0.01, 0.38, I^2=43.92%, P=0.04) (**Supplemental Table S9**). None of the other continuous subgroup analyses were significant.

Dose-response analyses

Supplemental Tables S6 and S10 and **Supplemental Figure S23** show the dose-response analysis for the 11 trials for which pasta intake could be quantified. No evidence of a linear dose response was seen for pasta intake by meta-regression analyses (**Supplemental Table S6**). There was also no evidence of a non-linear dose response by MKSPLINE (p=0.85) (**Supplemental Figure S23**) or piecewise linear meta-regression analyses (**Supplemental Table S10**).

Publication Bias

Supplemental Figures S24-S27 shows the funnel plots for body weight, BMI, body fat and waist circumference. There was no evidence of funnel-plot asymmetry. Formal testing with the Egger and Begg tests did not show evidence of small-study effects (P>0.05 for both). Publication bias was not assessed for waist-to-hip ratio and sagittal abdominal diameter, owing to <10 trial comparisons.

GRADE Assessment

Supplemental Table S11 shows a summary of the GRADE assessments for the effect of pasta in the context of low-GI dietary patterns on body weight and measures of adiposity**.** The evidence was graded as moderate for body weight, BMI, waist-to-hip ratio and sagittal abdominal diameter owing to downgrades for indirectness and low for waist circumference and body fat, owing to downgrades for indirectness and inconsistency (I^2=59%, P-heterogeneity<0.001; I^2=66%, P-heterogeneity<0.01, respectively).

DISCUSSION

The present systematic review and meta-analysis quantified the effect of pasta alone and pasta in the context of low-GI dietary patterns on body weight and other markers of adiposity. We identified no trial comparisons for the effect of pasta alone and 32 trial comparisons involving 2448 participants who were predominantly middle-aged and overweight or obese for the effect of pasta in the context of low-GI dietary patterns. The primary pooled analysis demonstrated that pasta in the context of low-GI dietary patterns did not contribute to weight gain, resulting in a significant weight loss of -0.63 kg when compared to diets higher in GI over a median follow-up of 12 weeks. The lack of harm was reflected in the established clinical secondary outcome measures of global (BMI and body fat) and abdominal adiposity (waist circumference, waist-to-hip ratio and sagittal abdominal diameter). These findings were robust across subgroups. The findings did not differ by metabolic phenotype in those who were overweight or obese or had diabetes, which is noteworthy since these are populations who would benefit from weight management strategies. There was also no effect modification by the energy balance of the design such that the weight loss was seen even under conditions of neutral energy balance (in which participants were instructed to consume dietary advice ad libitum), suggesting that encouragement of the consumption of pasta in the context of a low-GI dietary patterns does not cause harm and may even lead to spontaneous weight loss. There was also no effect modification by follow-up either in continuous meta-regression or categorical, where the 24 trials with <24weeks follow-up had a weight reduction similar to those 8 trials with ≥24weeks follow-up (-0.63kg vs -0.57kg, respectively). This is of relevance since many dietary studies are successful in demonstrating weight loss in the short term but not over the long term.

Findings in the context of existing studies

We are not aware of any RCTs directly assessing the effect of pasta intake on health parameters including body weight. Our findings, however, agree with earlier systematic reviews and meta-analyses of RCTs of the effect of low-GI dietary patterns irrespective of pasta intake on body weight and adiposity. The systematic review and meta-analysis by Thomas et al. in 2007 found a significant -1.1kg weight loss and -1.3kg/m^2 reduction in BMI favouring low-GI or glycemic load (GL) diets compared to control diets in 6 RCTs of 5 weeks to 6 months in duration in overweight or obese individuals[13]. Another systematic review and meta-analysis by Schwingshackl et al. in 2013 found a -0.62kg weight loss favouring low-GI/GL diets compared to higher GI/GL diets in 14 RCTs with greater than 6 months duration in overweight individuals (BMI>25kg/m^2)[14].

Our findings also agree with trials in which pasta was emphasized in the context of other healthy dietary patterns. One trial done in children in Spain given Mediterranean dietary advice which included increasing the intake of pasta found that approximately 11.3% of the participants in the Mediterranean diet group who were classified as overweight and obese changed their weight status to normal weight compared to only approximately 2.6% of the participants in the control group [55].

Other lines of evidence from observational studies have demonstrated benefits of pasta consumption on body weight and adiposity. Pasta intake was recently assessed in the Moli-sani study and the Italian Nutrition & HEalth Survey (INHES), a cross-sectional study of over 20,000 Italians from all over Italy[56]. The study demonstrated that higher pasta consumption was

associated with lower BMI, waist circumference and waist-to-hip ratio and with a lower prevalence of overweight and obesity[56]. Furthermore, greater pasta intake was associated with better adherence to the Mediterranean diet, a dietary pattern which has a demonstrated cardiovascular benefit[57]. Similar associations between greater pasta intake and lower body weight have also been observed in prospective U.S. cohorts[58]. A pooled analysis of the 3 Harvard cohorts also showed that higher GI diets (which would preclude pasta) were associated with weight gain[59].

Although the product form of pasta can vary widely, including in shape (e.g. macaroni, spaghetti, linguine), ingredients (e.g. type of wheat, egg content), and processing technique (e.g. drying temperature), studies have demonstrated that when comparing pastas varying in these parameters, despite slight variations in glycemic response among pastas, glycemic responses are still lower compared to a control, e.g. white bread[60,61]. One concern of the choice of pasta as a carbohydrate food is that is it a refined food low in fibre. Although there are whole grain pasta options available, studies have demonstrated that fiber added to pasta, does not significantly affect the glucose or insulin response, the secretion of gut hormones or satiety[62,63]. Furthermore, pasta has a similar glycemic response compared to many fiber-rich carbohydrates, including barley, legumes and steel cut oats, and still a lower glycemic response compared to other fiber-rich foods including whole wheat bread, breakfast cereals like bran flakes and potatoes with skin[64].

The mechanism by which pasta in the context of low-GI dietary patterns lead to weight loss even under conditions of ad libitum dietary advice is unclear. Lower GI diets may result in greater

body weight reduction compared to higher GI diets because lower GI foods may be more satiating [65] and have been shown to delay hunger and decrease subsequent energy intake[13]. Low-GI dietary patterns are also characterized by high fiber content[64,66] which may also contribute to improvements in satiety and hunger[17]. Furthermore, studies which have compared ad libitum low-GI dietary patterns to standard energy-restricted low-fat diets, demonstrated equivalency or better weight loss when following the low-GI diet, despite the fact that they could eat as much as they desired[13,67]. Thus, voluntary energy intake may be lower after low-GI meals, as has been previously demonstrated[68].

Strengths and limitations

The strengths of the present systematic review and meta-analysis include that it is comprehensive, includes RCTs which protects against bias and uses GRADE to evaluate the quality of evidence. Additionally, a large number of trials were identified (32 trials) for the primary outcome of body weight, the median follow-up period was 12 weeks which allows for the assessment of a moderate duration of intervention, none of the trials were rated as having a serious risk of bias, and there was no evidence of publication bias.

There are several limitations. First, the present systematic review and meta-analysis showed evidence of inconsistency in the treatment estimates across trials for some of the outcomes assessed. There was evidence of unexplained heterogeneity in waist circumference (I^2=62%) and in body fat (I^2=65%). Although the inconsistency in these outcomes may have related to measurement error[69] in the different techniques for measuring waist circumference and body fat, we were unable to conduct sensitivity or subgroup analyses to explore this source of

heterogeneity. Second, there was evidence of indirectness. Most of the available trials did not quantify the amount of pasta consumed in the context of the low-GI dietary patterns. Although sensitivity analyses in which analyses were restricted to the 11 trials that did quantify (providing a median 3.33 servings/week) pasta intake did not meaningfully alter our estimates (-0.70kg versus -0.63kg), it is difficult to quantify the effect of pasta in these diets. There is also the question of translation to other background diets. Whether the observed effect of pasta in the context of low-GI dietary patterns will hold in the context of other healthy dietary patterns, such as Mediterranean and Vegetarian dietary patterns, is unclear. Although there is no biological reason to doubt that the findings would hold, there is a lack of direct evidence to support this conclusion. If the question had been asked from the perspective of benefit as opposed to that of harm, then the relatively short duration of the included trials may be another reason to downgrade for indirectness. In the absence of long-term trials (>1 year diet duration), it is difficult to conclude with certainty that the observed lack of harm implies an actual sustainable benefit. Finally, there was some evidence of imprecision for benefit but not harm. Whereas the 95% CI of the pooled estimates did not overlap with our pre-specified MID for harm (that is, they did not contain evidence for harm) and so were not downgraded for imprecision, the upper bound of the 95% CI would overlap with the lower bound of the same MID to assess the precision of the evidence for benefit for some outcomes.

Balancing these strengths and limitations, GRADE assessed the overall quality and strength of the available evidence of the effect of pasta in the context of low-GI dietary patterns as moderate for the primary outcome of body weight and the secondary outcomes of BMI, waist-to-hip ratio and sagittal abdominal diameter owing to downgrades for indirectness. The evidence was

assessed as low for the other secondary outcomes of body fat and waist circumference owing to downgrades for indirectness and inconsistency.

Implications

These results are important considering the negative messages directed at the public regarding carbohydrates, which is influencing their food choices, as is evident in recent reductions in carbohydrate intake[70-72], and in particular reductions in pasta consumption[70,73-76]. Contrary to these concerns, the available evidence shows that when pasta is consumed in the context of low-GI dietary patterns that there is not weight gain but rather marginally clinically significant weight loss (>0.5kg)[77].

Although we were able to approximate the amount of pasta consumed in one third of included trials, it is unclear what the effect of pasta would be in the context of other dietary patterns. Low-GI dietary patterns, however, shares many similarities with a Mediterranean diet, which emphasizes many low-GI foods and has demonstrated a CVD benefit[57].

Current clinical practice guidelines already suggest the replacement of high GI foods with low-GI foods for improvement of glycemic control and cardiovascular risk factors[78,79]. The present evidence means that pasta may be highlighted as an important example of a low-GI food which can contribute to a low-GI dietary pattern, a pattern which in turn may potentially improve cardio-metabolic risk without an adverse effect on weight control.

CONCLUSIONS

In conclusion, pasta consumed in the context of low-GI dietary patterns does not have an adverse effect on body weight and adiposity outcomes of importance in the prevention and management of overweight and obesity. The results are generalizable in the context of a high carbohydrate diet composed of low-GI foods with or without the intention of weight loss in middle-aged individuals who are overweight or obese or have diabetes. Although the clinical significance of the observed weight loss is debatable, this finding increases our confidence that pasta in the context of low-GI dietary patterns does not result in weight gain. Further research may change our confidence in the estimates for our primary outcome body weight and several key secondary outcomes including BMI and two measures of abdominal adiposity, waist-to-hip ratio and sagittal abdominal diameter. More research, however, is needed, to improve our estimates for the secondary outcomes, body fat and waist circumference and assess whether our findings extend to related cardio-metabolic outcomes. There is also a need for more randomized trials of >1 year diet duration to clarify whether the lack of harm for pasta in the context of low-GI dietary patterns will translate into meaningful long-term benefits. Other randomized trials should focus on whether pasta will have similar effects in the context of other healthy dietary patterns such as a Mediterranean diet.

Figure Legend

Figure 1: Literature Search

Figure 2: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg). n = 2448. Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.
CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids; Pro, protein.

Figure 3: Plot of pooled effect estimates from randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on global and abdominal markers of adiposity. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.
BMI; body mass index; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet.

Acknowledgements

We wish to thank Teruko Kishibe, an information specialist in the Scotiabank Health Sciences Library, St Michael's Hospital, for her help in the development of search terms used. Aspects of this work were presented at the Società Italiana di Nutrizione Umana (SINU) Congress, in Parma, Italy, on April 12, 2017 and at the 35th International Symposium on Diabetes and Nutrition, of the Diabetes and Nutrition Study Group (DNSG) of the European Association for the Study of Diabetes (EASD) in Skagen, Denmark, on June 20 and 21, 2017.

Data Sharing

No additional data available.

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Ethics Approval

Not required.

Authorship:

All authors had full access to all of the data (including statistical reports and tables) in this study and take full responsibility for the integrity of the data and the accuracy of the data analysis.

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Transparency declaration: As guarantor, J.L. Sievenpiper affirms that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

Funding:

This work was funded by the Canadian Institutes of Health Research (funding reference number, 129920) through the Canada-wide Human Nutrition Trialists' Network (NTN). The Diet, Digestive tract, and Disease (3-D) Centre, funded through the Canada Foundation for Innovation (CFI) and the Ministry of Research and Innovation's Ontario Research Fund (ORF),

provided the infrastructure for the conduct of this project. **L Chiavaroli** was funded by a Toronto 3D Postdoctoral Fellowship Award. **DJA Jenkins** was funded by the Government of Canada through the Canada Research Chair Endowment. **JL Sievenpiper** was funded by a PSI Graham Farquharson Knowledge Translation Fellowship, Canadian Diabetes Association (CDA) Clinician Scientist award, CIHR INMD/CNS New Investigator Partnership Prize, and Banting & Best Diabetes Centre Sun Life Financial New Investigator Award. None of the sponsors had a role in any aspect of the present study, including design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, approval of the manuscript or decision to publish.

Conflicts of Interest:

All authors have completed the Unified Competing Interest form (available on request from the corresponding author) and declare: **CWC Kendall** has received research support from the Advanced Foods and Material Network, Agrifoods and Agriculture Canada, the Almond Board of California, the American Pistachio Growers, Barilla, the California Strawberry Commission, the Calorie Control Council, CIHR, the Canola Council of Canada, the Coca-Cola Company (investigator initiated, unrestricted grant), Hain Celestial, the International Tree Nut Council Nutrition Research and Education Foundation, Kellogg, Kraft, Loblaw Companies Ltd., Orafti, Pulse Canada, Saskatchewan Pulse Growers, Solae and Unilever. He has received travel funding, consultant fees and/or honoraria from Abbott Laboratories, the Almond Board of California, the American Peanut Council, the American Pistachio Growers, Barilla, Bayer, the Canola Council of Canada, the Coca-Cola Company, Danone, General Mills, the International Tree Nut Council Nutrition Research and Education Foundation, Kellogg, Loblaw Companies Ltd., the Nutrition Foundation of Italy, Oldways Preservation Trust, Orafti, Paramount Farms, the Peanut Institute,

PepsiCo, Pulse Canada, Sabra Dipping Co., Saskatchewan Pulse Growers, Solae, Sun-Maid, Tate and Lyle, and Unilever. He is on the Clinical Practice Guidelines Expert Committee for Nutrition Therapy of the European Association for the Study of Diabetes (EASD) and has served on the scientific advisory board for the Almond Board of California, the International Tree Nut Council, Oldways Preservation Trust, Paramount Farms and Pulse Canada. **DJAJ** has received research grants from Saskatchewan Pulse Growers, the Agricultural Bioproducts Innovation Program through the Pulse Research Network, the Advanced Foods and Material Network, Loblaw Companies Ltd., Unilever, Barilla, the Almond Board of California, Agriculture and Agri-food Canada, Pulse Canada, Kellogg's Company, Canada, Quaker Oats, Canada, Procter & Gamble Technical Centre Ltd., Bayer Consumer Care, Springfield, NJ, Pepsi/Quaker, International Nut & Dried Fruit (INC), Soy Foods Association of North America, the Coca-Cola Company (investigator initiated, unrestricted grant), Solae, Haine Celestial, the Sanitarium Company, Orafti, the International Tree Nut Council Nutrition Research and Education Foundation, the Peanut Institute, the Canola and Flax Councils of Canada, the Calorie Control Council (CCC), the CIHR, the Canada Foundation for Innovation and the Ontario Research Fund. He has received in-kind supplies for trial as a research support from the Almond board of California, Walnut Council of California, American Peanut Council, Barilla, Unilever, Unico, Primo, Loblaw Companies, Quaker (Pepsico), Pristine Gourmet, Bunge Limited, Kellogg Canada, WhiteWave Foods. He has been on the speaker's panel, served on the scientific advisory board and/or received travel support and/or honoraria from the Almond Board of California, Canadian Agriculture Policy Institute, Loblaw Companies Ltd, the Griffin Hospital (for the development of the NuVal scoring system, the Coca-Cola Company, EPICURE, Danone, Diet Quality Photo Navigation (DQPN), FareWell, Verywell, True Health Initiative, Saskatchewan Pulse Growers,

Sanitarium Company, Orafti, the Almond Board of California, the American Peanut Council, the International Tree Nut Council Nutrition Research and Education Foundation, the Peanut Institute, Herbalife International, Pacific Health Laboratories, Nutritional Fundamental for Health, Barilla, Metagenics, Bayer Consumer Care, Unilever Canada and Netherlands, Solae, Kellogg, Quaker Oats, Procter & Gamble, the Coca-Cola Company, the Griffin Hospital, Abbott Laboratories, the Canola Council of Canada, Dean Foods, the California Strawberry Commission, Haine Celestial, PepsiCo, the Alpro Foundation, Pioneer Hi-Bred International, DuPont Nutrition and Health, Spherix Consulting and WhiteWave Foods, the Advanced Foods and Material Network, the Canola and Flax Councils of Canada, the Nutritional Fundamentals for Health, Agri-Culture and Agri-Food Canada, the Canadian Agri-Food Policy Institute, Pulse Canada, the Saskatchewan Pulse Growers, the Soy Foods Association of North America, the Nutrition Foundation of Italy (NFI), Nutra-Source Diagnostics, the McDougall Program, the Toronto Knowledge Translation Group (St. Michael's Hospital), the Canadian College of Naturopathic Medicine, The Hospital for Sick Children, the Canadian Nutrition Society (CNS), the American Society of Nutrition (ASN), Arizona State University, Paolo Sorbini Foundation and the Institute of Nutrition, Metabolism and Diabetes. He received an honorarium from the United States Department of Agriculture to present the 2013 W.O. Atwater Memorial Lecture. He received the 2013 Award for Excellence in Research from the International Nut and Dried Fruit Council. He received funding and travel support from the Canadian Society of Endocrinology and Metabolism to produce mini cases for the Canadian Diabetes Association (CDA). He is a member of the International Carbohydrate Quality Consortium (ICQC). His wife, ALJ, is a director and partner of Glycemic Index Laboratories, Inc., and his sister received funding through a grant from the St. Michael's Hospital Foundation to develop a cookbook for

one of his studies. **JL Sievenpiper** has received research support from the Canadian Institutes of health Research (CIHR), Canadian Diabetes Association (CDA), PSI Foundation, Banting and Best Diabetes Centre (BBDC), Canadian Nutrition Society (CNS), American Society for Nutrition (ASN), Calorie Control Council, INC International Nut and Dried Fruit Council Foundation, National Dried Fruit Trade Association, The Coca-Cola Company (investigator initiated, unrestricted), Dr. Pepper Snapple Group (investigator initiated, unrestricted), The Tate and Lyle Nutritional Research Fund at the University of Toronto, and The Alberta Pulse Growers Glycemic Control and Cardiovascular Disease in Type 2 Diabetes Fund at the University of Toronto. He has received in-kind research support from the Almond board of California, Walnut Council of California, American Peanut Council, Barilla, Unilever, Unico, Primo, Loblaw Companies, Quaker (Pepsico), Pristine Gourmet, Bunge Limited, Kellogg Canada, WhiteWave Foods. He has received reimbursement of travel expenses, speaker fees, and/or honoraria from the Canadian Diabetes Association (CDA), Canadian Nutrition Society (CNS), University of Alabama at Birmingham, Oldways Preservation Trust, Nutrition Foundation of Italy (NFI), Diabetes and Nutrition Study Group (DNSG) of the European Association for the Study of Diabetes (EASD), International Life Sciences Institute (ILSI) North America, Dr. Pepper Snapple Group, Corn Refiners Association, World Sugar Research Organization, Dairy Farmers of Canada, Società Italiana di Nutrizione Umana (SINU), III World Congress of Public Health Nutrition, C3 Collaborating for Health, Sprim Brasil, WhiteWave Foods, Rippe Lifestyle, mdBriefcase, Federation of European Nutrition Societies (FENS), New York Academy of Sciences, International Diabetes Federation, American Heart Association (AHA), American Society for Nutrition (ASN), FoodMinds LLC, Memac Ogilvy & Mather LLC, Pulse Canada, Pepsico, BCFN Foundation, The Ginger Network LLC, and Dietitians of Canada. He has ad hoc

consulting arrangements with Winston & Strawn LLP, Perkins Coie LLP, and Tate & Lyle. He is a member of the European Fruit Juice Association Scientific Expert Panel. He is on Clinical Practice Guidelines Expert Committees of the Canadian Diabetes Association (CDA) European Association for the study of Diabetes (EASD), and Canadian Cardiovascular Society (CCS), as well as an expert writing panel of the American Society for Nutrition (ASN). He serves as an unpaid scientific advisor for the Food, Nutrition, and Safety Program (FNSP) and the Technical Committee on Carbohydrates of the International Life Science Institute (ILSI) North America. He is a member of the International Carbohydrate Quality Consortium (ICQC), Executive Board Member of the Diabetes and Nutrition Study Group (DNSG) of the EASD, and Director of the Toronto 3D Knowledge Synthesis and Clinical Trials foundation. His wife is an employee of Unilever Canada. No competing interests were declared by **CR Braunstein**, **S** Blanco Mejia**, and LA Leiter.**

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Table 1: Summary of trial characteristics

Trial Characteristics*	ALL	Neutral Energy Balance	Negative Energy Balance
Trial Number (n)	32	23	9
Trial Size (total, range)	2448 (8 - 250)	1989 (8 - 250)	459 (13 - 123)
Male: Female[a] (%)	40:60	47:53	27:73
Age (years)	50 (40 - 58)	52.0 (42.1 – 59.5)	49.5 (34.2 – 53.0)
Metabolic Phenotype (OW/OB:DM:CHD) (%)	66:31:3	57:39:4	89:11:0
Setting (IP:OP) (%)	3:97	4:96	0:100
Baseline Body Weight (kg)[b]	85.5 (80.0 - 91.9)	84.1 (79.5 – 87.5)	92.5 (86.1 – 93.9)
Baseline BMI (kg/m^2)[c]	30.4 (28.2 - 32.0)	29.5 (27.4 – 31.4)	31.7 (30.1 – 32.9)
Study Design (C:P) (%)	19:81	26:74	0:100
Dose Pasta (servings/week)[d]	3.3 (2.3 - 3.5)	3.4 (2.9 – 4.1)	2.3 (2.3-3.5)
GI in Pasta/LGI group	49.0 (44.0 - 55.1)	46.5 (49.9 – 55.5)	44.0 (42.3 - 49.4)
GI in Higher GI group	62.5 (61.6 - 63.2)	63.3 (60.1 – 64.4)	61.0 (59.2 – 66.6)
Calorie reduction in Pasta/LGI group (kcal)[e]	-179 (-90 - -448)	-165 (-74 - -313)	-447 (-134 - -594)
Calorie reduction in Higher GI group (kcal)[e]	-181 (-93 - -401)	-160 (-40 - -248)	-470 (-172 - -561)
Feeding Control (Met:Supp:DA) (%)	6:44:50	4:48:48	11:33:56
Follow-Up Duration (Weeks)	12 (9-21)	12 (6-24)	12 (10 - 21)
Funding Sources (A:I:AI:NR) (%)	47:9:25:19	44:13:26:17	56:0:22:22

* median (inter quartile range), unless otherwise indicated
[a]24/32 trials provided data on sex
[b] 30/32 trials reported baseline body weight
[c] 28/32 trials reported baseline BMI
[d] 11/32 trials provided data from which dose could be approximated
[e] 20/32 trials provided data from which to approximate changes in caloric intake
A, agency; AI, agency and industry; BMI, body mass index; C, crossover design; CHD, coronary heart disease; DA, dietary advice; DM, diabetes; GI, glycemic index; I, industry; IP, inpatient; LGI, low glycemic index; Met, metabolic; NR, not reported; OB, obese; OP, outpatient; OW, overweight; P, parallel design; Suppl, supplemented/provision of certain food

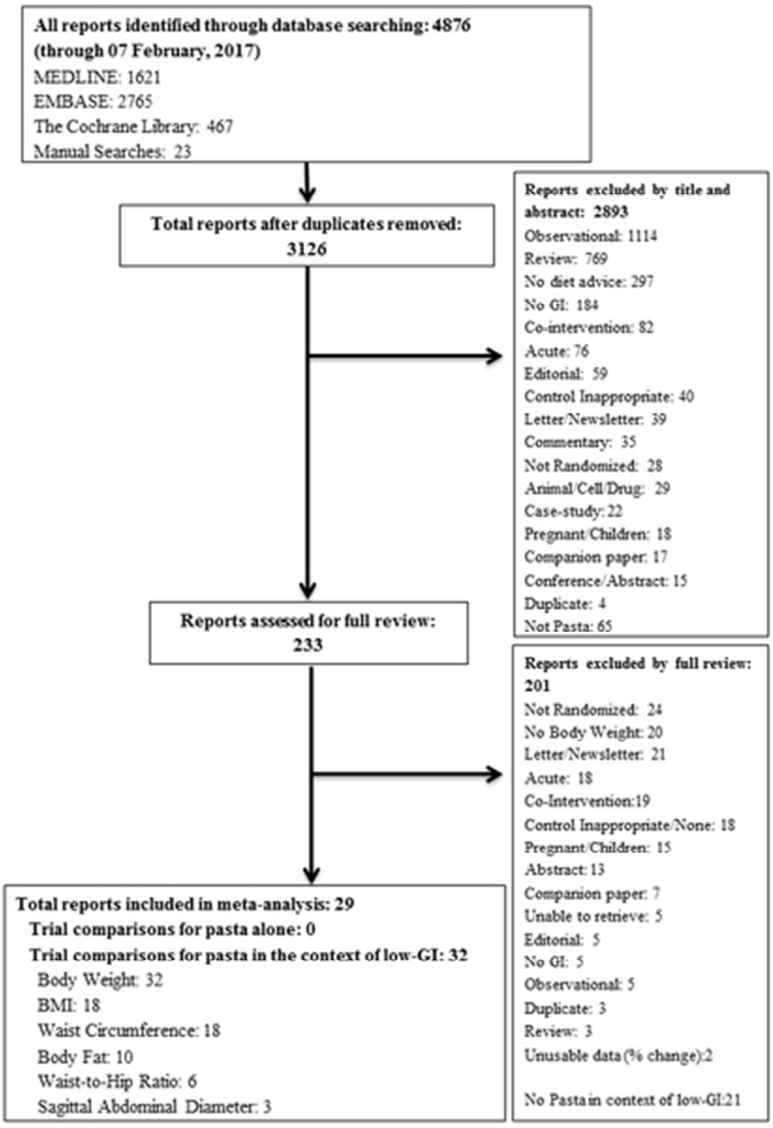

Figure 1: Literature Search

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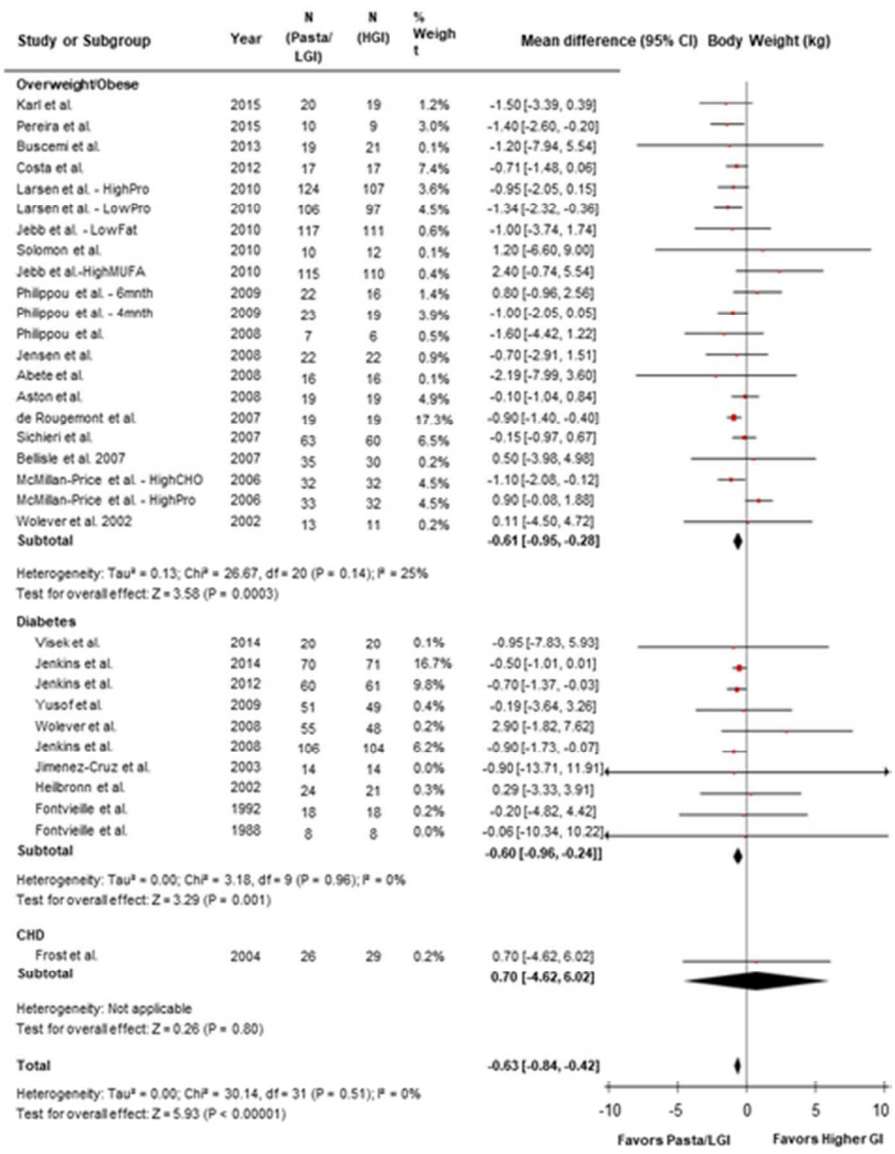

Figure 2: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg). n = 2448. Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I2 statistic where I2≥50% is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.

CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids; Pro, protein.

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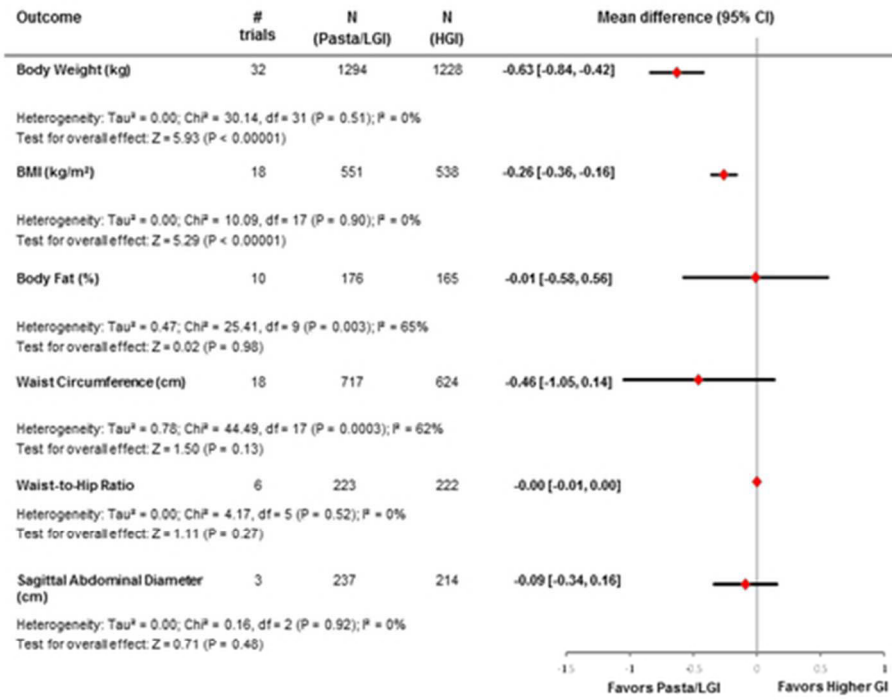


Figure 3: Plot of pooled effect estimates from randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on global and abdominal markers of adiposity. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I2 statistic where I2≥50% is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.
BMI; body mass index; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet.

40x54mm (300 x 300 DPI)

Online Supplemental Information

Table of Contents

Supplemental Tables

Supplemental Table S1: Search strategy for studies assessing the effect of pasta in the context of low-GI dietary patterns on body weight in randomized controlled trials

Database	Search Period	Search Terms
Medline	1946 to February 07, 2017	1. pasta/ 2. spaghetti/ 3. macaroni/ 4. lasagna/ 5. fusilli/ 6. noodle/ 7. glycaemic index.tw. 8. glycemic index.tw. 9. glycaemic ind*.tw. 10. glycemic ind*.tw. 11. glycemic load*.tw. 12. glycaemic load*.tw. 13. glycemic index/ 14. body mass index/ 15. body mass index.tw. 16. BMI.tw. 17. overweight.tw. 18. weight*.tw. 19. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 20. 14 or 15 or 16 or 17 or 18 21. 19 and 20 22. limit 21 to animals 23. 21 not 22
Embase	1946 to February 07, 2017	1. pasta/ 2. spaghetti/ 3. macaroni/ 4. lasagna/ 5. fusilli/ 6. noodle/ 7. glycaemic index.tw. 8. glycemic index.tw. 9. glycaemic ind*.tw. 10. glycemic ind*.tw. 11. glycemic load*.tw. 12. glycaemic load*.tw. 13. glycemic index/ 14. body mass index/ 15. body mass index.tw. 16. BMI.tw.

		17. overweight.tw. 18. weight*.tw. 19. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 20. 14 or 15 or 16 or 17 or 18 21. 19 and 20 22. limit 21 to animals 23. 21 not 22
The Cochrane Library	1946 to February 07, 2017	1. pasta/ 2. spaghetti/ 3. macaroni/ 4. lasagna/ 5. fusilli/ 6. noodle/ 7. glycemic index/ 8. glycaemic ind*.tw. 9. glycemic ind*.tw. 10. glycemic load*.tw. 11. glycaemic load*.tw. 12. exp body weight/ 13. body weight*.tw. 14. BMI.tw. 15. body mass index/ 16. body mass index.tw. 17. weight*.tw. 18. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 19. 12 or 13 or 14 or 15 or 16 or 17 20. 18 and 19 21. limit 20 to animals 22. 20 not 21

Supplemental Table S2: PICO framework of the search strategy

PICO framework[a] defined in the present systematic review and meta-analysis			
Participants	**Interventions**	**Comparators**	**Outcomes**
Adult men and women excluding pregnant or breastfeeding women	Low glycemic index interventions where pasta is included as part of the intervention	Higher glycemic index diets where pasta is not included as part of the intervention	Body weight Body mass index (BMI) Body Fat (%) Waist circumference Waist-to-hip ratio

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

Supplement Table S3a: Trial characteristics

Overweight/Obese Trials

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Karl et al. 2015 ‡‡‡	39 (19M:20F)**	OB, OP					2.33	USA	P	Negative+ Neutral	17		CR to 2/3kcal; Metabolic	Agency
Low GI	20		56 (5)*	92.9 (13.6)*	32.3 (3.4)*	42:133						68:15:16		
Higher GI	19		56 (5)*	94 (9.7)*	33.4 (2.6)*	61:201						70:16:14		
Pereira et al. 2015	19 (4M:15F)**	OW, IP/OP					NR	Brazil	P	Neutral	6.4		Ad libitum	Unknown
Low GI	10		28(5)	80.0(12.6)	29.9 (2.1)	41.2(2.2) ¥ **						48.3:16.1:32.8		
Higher GI	9		26(3)	79.1(12.2)	29.1 (2.0)	74.1(2.9) ¥ **						54.6:12.7:34.4		
Buscemi et al. 2013	40 (19M:21F)**	OW/OB, high CVD risk, OP					NR	Italy	P	Negative	12		CR to 20kcal/kg/d; Ad libitum	Unknown
Low GI	19		51 (8)	93.8 (17.3)	34.3 (6.6)	48.1: 138						56:18:26		
Higher GI	21		49 (8)	93.2 (14.4)	34.5 (5.1)	59.3: 174						57:16:27		
Costa et al. 2012	17 (7M:10F)	OW, IP/OP	25.4 (5.8)	84.1(16.3)	26.3(3.2)		NR	Brazil	C	Neutral	4		Ab libitum,2 meals+3 fruit/d provided	NR
Low GI						47.5(3.8)						58.6:13.9:25.5		
Higher GI						61.6(2.8)						55.4:14.2:30.3		
Jebb et al. 2010 - High MUFA	225**	OH, some CV risk factors, OP	~52		~28.5+		1.75	UK	P	Neutral	24		Ad Libitum, key foods provided	Agency, foods by industry
Low GI	115			83.7 (69.6-93.1)¶¶		~55.2						~44.6:16.4:35.7****		
Higher GI	110			80.5 (70.0-92.1)¶¶		~63.3						~44.9:15.3:35.6****		
Jebb et al. 2010 - Low Fat	250**	OH, some CV risk factors, OP	~52		~28.5+		3.5	UK	P	Neutral	24		Ad Libitum, key foods provided	Agency, foods by industry
Low GI	117			79.4 (70.1- 91.8)¶¶		~56.3						~51.5:14.2:26.1****		
Higher GI	111			80.7 (71.4- 91.4)¶¶		~64.4						~51.1:15.7:27.5****		
Larsen et al. 2010-High Pro	231**	OW/OB, OP			NR		NR	Europe	P	Neutral	26		Ad libitum	Agency
Low GI	124		42.1 (6.5)	88.5 (15.6)		~56.5: 108.9						~43:22:32		
Higher GI	107		42 (5.7)	89.5 (17.1)		~61.4: 113.1						~45:23:31		
Larsen et al. 2010 - Low Pro	203 **	OW/OB, OP			NR		NR	Europe	P	Neutral	26		Ad libitum	Agency
Low GI	106		42.2 (5.7)	88.4 (15.7)		~56.2: 121						~51:18:30		
Higher GI	97		42 (5.9)	86.6 (13.8)		~61.6: 137.9						~51:17:31		

Supplement Table S3b: Trial characteristics continued

Overweight/Obese Trials continued

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Solomon et al. 2010	22 (8M:14F)**	OB, Pre-T2DM, OP					7	USA	P	Neutral	12		Metabolic plus excerise program	Agency
Low GI	10 (3M:7F)		67 (6)	97.4 (12.0)	34.9 (1.1)	39.8 (0.9)						54.7(0.3):28.3(0.3):17.0(0.3)		
Higher GI	12 (5M:7F)		64 (3)	94.7 (15.2)	34.1 (1.1)	80.0 (2.1)						55.6(0.7):27.8(0.7):16.6(0.3)		
Philippou et al. 2009- 6 mo	38**	≥1 CHD risk factors, OP	(35-65) ¶				NR	UK	P	Negative (for all but n=2)	24		500kcal CR; Ad libitum	Agency
Low GI	22			91.3(14.8)***	29.1 (3.6)***	50.6 (4.6): 114.4(31.5)								
Higher GI	16			97.5(16.4)***	30.5 (3.5)***	63.2(5.6): 175.0(45.6)								
Philippou et al. 2009- 4 mo	42**	OW, OP	(18-65)¶				NR	UK	P	Neutral	16		Ad libitum	Unknown
Low GI	23			87.2 (15.3)	32.5 (4.8)	49.7(5.7):89.7(27.5)						47.6(6.7):19.5(4.2):31.8(5.8)		
Higher GI	19			83.6 (13.4)	31.3 (4.8)	63.7(9.4):136.8(56.3)						48.9(7):19.3(4.9):30.9(9)		
Abete et al. 2008	32 (18M:14F)	OB, OP	36(7)				2.33	Spain	P	Negative	8		30% CR; Ad libitum, 3-day menus	Agency
Low GI	16			94.3(16.1)	32.8 (4.3)	(40-45)¶						50.2 (1.8);18.3(1.6);31.5(1.6)		
Higher GI	16			94.4(13.1)	32.2 (4.4)	(60-65)¶						47.8(6.8);19.6(5.6);32.6(4.3)		
Aston et al. 2008	19 (0M:19F)**	OW/OB, OP	51.9(7.6)	87.5 (15)	33.1 (4.9)		3.33	UK	C	Neutral	12		Ad libitum, key CHO foods provided	Agency
Low GI						55.5(3.8): 133.8(22.9)****						51.4(6.0):17.0(2.4):32.2(5.1)***		
Higher GI						63.9(3): 138.8(30.5)****						47.6(6.1):17.6(3.3):34.1(5.7)***		
Jensen et al. 2008	44 (0M:44F)**	OW, OP	(20-40)¶				3	Denmark	P	Neutral	10		Ad libitum, partial provision, menu plans	Agency, Industry
Low GI	22 (0M:22F)			77.9(6.9)	27.4 (1.5)	72¥						~57(5):17(0):23(5) ‡		
Higher GI	22 (0M:22F)			80.2(1.4)	27.6 (0.3)	95¥						~57(5):17(0):22(5) ‡		
Philippou et al. 2008	13 (5M:8F)**	≥1 CHD risk factors, OP					NR	UK	P	Negative	12		500kcal CR; Ad libitum	Agency
Low GI	7 (3M:4F)		54 (49-58)¶¶	81.5 (4.7)***	28.7 (2.1)***	51.3(51.0-52.0): 105.6 (76.9-110.1)¶¶						46.0(37.8-51.0): 17.1(15.7-17.4): 32.8(31.3-37.1)¶¶		
Higher GI	6 (2M:4F)		45 (39-50)¶¶	89.7 (12.8)***	31.5 (4.4)***	59.3(59.2-64.0): 114.7(98.5-134.9)¶¶						49.4(47.8-51.7):19.6(14.0-23.1):29.2(25.2-34.5)¶¶		

Supplement Table S3c: Trial characteristics continued

Overweight/Obese Trials continued

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Bellisle et al. 2007	65 (0M:65F)**	OW/OB, OP					NR	France	P	Neutral	12		Ad libitum	Industry
Low GI	35		46.1 (13.6)	80 (13.2)	30.2 (4.1)	na								
Higher GI	30		45.3 (12.0)	79 (13.1)	30.4 (4.4)	na								
de Rougemont et al. 2007	38 (22M:16F)**	OW, OP					2.8	France	P	Neutral	5		Ad libitum, some foods provided	Agency, Industry
Low GI	19		36.3 (8.7)	77.2 (9.6)	27.5 (1.3)	46.5 (1.3)						42.6 (3.9):19.8 (1.7):37.7 (4.4)		
Higher GI	19		40.4 (9.6)	77.3 (9.2)	27.2 (1.3)	66.3 (2.6)						44.1 (3.5):17.6 (1.7):38.4 (3.1)		
Sichieri et al. 2007	123 (OM:123F)**	OW, OP					NR	Brazil	P	Negative	72		100-300kcal CR; 6-d menu and exchange lists provided	Agency
Low GI	63		37.2 (5.4)*	67.7 (6.6)*	na	21(38): 74(84)						59.5 (6.3): 13.3: 27.2(4.6)		
Higher GI	60		37.5 (5.6)*	68.5 (7.5)*	na	51(28): 199(43)						61.6 (6.2): 12.3: 26.1(4.7)		
McMillian-Price et al. 2006-High Carb	64(16M:48F)	OW/OB, OP					NR	Australia	P	Negative	12		Ad libitum, key foods and meals provided	Agency-Industry
Low GI	32		30.5 (7.9)	87.1 (15.3)	30.6 (4.5)	45 (6):89 (28)						56 (6):19 (0):22 (6)		
Higher GI	32		31.8 (9.6)	86 (10.7)	30.9 (3.4)	70 (6):129 (45)						60 (6):18 (6):19 (6)		
McMillian-Price et al. 2006-High Protein	65(15M:50F)	OW/OB, OP					NR	Australia	P	Negative	12		Ad libitum, key foods and meals provided	Agency-Industry
Low GI	33		34.6 (8.6)	88.4 (17.2)	32.1 (5.2)	44 (6):59 (23)						40 (11):26 (6):28 (6)		
Higher GI	32		30.2 (8.5)	87.7 (16.4)	31.3 (4.5)	59 (6):75 (17)						42 (6):28 (6):27 (6)		
Wolever et al. 2002	24 (5M:19F)**	IGT, OP					NR	Canada	P	Neutral	16		Ad libitum, partial provision	Agency
Low GI	13(3M:10F)		55.2 (10.8)	79.7 (13.1)***	29.7 (4.3)	54.4 (2.5):91.8 (9.4)						54.8 (6.1):19.4 (1.8):24.7 (5.8)		
Higher GI	11(2M:9F)		58.8 (13.3)	76.4 (20.4)***	29.3 (7.3)	59.3 (2.0):96.8 (11.6)						52.8 (6.6):17.4 (2.3):27.9 (6.3)		

Supplement Table S3d: Trial characteristics continued

Diabetes Trials

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Jenkins et al. 2014	141(77M:64F)	T2DM, OP					NR	Canada	P	Neutral	12		Ad libitum, bread supplement	Industry Association
Low GI	70 (38M:32F)		59 (10)	85 (20)	30 (5)	~51:53						~38.5:19.8:37.2		
Higher GI	71 (39M:32F)		59 (10)	84 (19)	31 (6)	~62:89						~49.2:19.8:27.4		
Visek et al. 2014	20 (12M:8F)	T2DM, OP	62.7 (5.8)	91.9 (14.1)	32 (4.2)		NR	Czech Republic	C	Neutral	12		Ad libitum	Agency
Low GI						49 (48-51)¶ ¶						~37.2:18.0:36.0		
Higher GI						68 (61-72)¶ ¶						~36.2:17.3:40.0		
Jenkins et al. 2012	121 (61M: 60F)	T2DM, OP					NR	Canada	P	Neutral	12		Ad libitum	Agency
Low GI	60		58 (10.1)	85.6 (20.1)	31.4 (7.0)	47: 80						45.4:22.8:30.5		
Higher GI	61		61 (7.8)	82.5 (17.2)	29.9 (5.5)	58: 100						48.3:21.4:28.5		
Yusof et al. 2009	100**	T2DM, OP	NR				NR	Malaysia	P	Neutral	12		Ad libitum, key foods provided to lowGI group	Agency
Low GI	51			69.12 (13.33)	27.05 (4.91)	57(6): 108(32)						52(4):18(3):30(4)		
Higher GI	49			66.83 (11.50)	26.79 (4.65)	64(5): 131(30)						54(4):17(3):28(5)		
Jenkins et al 2008	210 (125M:82F)	T2DM, OP					NR	Canada	P	Neutral	24		Ad libitum	Agency
Low GI	106 (65M:41F)		60 (10)	87.0 (20.0)	30.6 (6.0)	49.4: 91.5						44.0:21.2:33.3		
Higher GI	104 (63M:41F)		61 (9)	87.8 (19.4)	31.2 (5.8)	59.3: 117.9						47.5:20.7:30.5		
Wolever et al. 2008	103	T2DM, OW/OB, OP					NR	Canada	P	Neutral	52		Ad libitum, key foods provided	Agency
Low GI	55		60.6 (7.5)*	81.1 (18.7)*	31.6 (4.5)*	55.1 (3.0): 133 (14.8)						51.9 (6.7):20.6(3.0):26.5 (5.9)		
Higher GI	48		60.4 (7.9)*	84.4(18.0)*	30.1 (4.3)*	63.2 (2.8): 135 (20.8)						46.5 (6.2):20.4 (2.8):30.8 (4.8)		

Supplement Table S3e: Trial characteristics continued

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Diabetes Trials continued														
Jimenez-Cruz et al. 2003	14 (6M:8F)**	T2DM, OP	59 (34)				NR	Mexico	C	Neutral	6		Ad libitum	Industry
Low GI				91.6 (24.3)	32.4 (6.0)	44(3.4): 86(19.8)						60:21:23		
Higher GI				92.6 (25.4)	32.3 (6.0)	56(4.9): 139(27.3)						64:18:20		
Heilbronn et al. 2002	45 (23M:22F)**	T2DM, OW, OP			NR		3.5	Australia	P	Negative	8		CR to1500kcal/d; Ad libitum, partial provision	Unknown
Low GI	24 (11M:13F)		56.0(9.4)	91.7(16.2)		43						58.9 (2.9):22.2 (1.5):17.9 (3.9)		
Higher GI	21 (12M:9F)		57.5(9.6)	93.2 (13.3)		75						60.8 (2.3):21.7 (0.9):17.1 (2.3)		
Fontvieille et al. 1992	18 (12M:6F)	T1DM/T2DM, OP	47.2(11.6)	NR	24.8(2.6)		4.7	France	C	Neutral	5		Ad libitum	Agency, Industry
Low GI						38.1(5.3)						45.8(7.2):18.0(2.5):36.2(6.8)		
Higher GI						64.2(3.1)						44.9(7.3):18.8(1.6):36.3(6.0)		
Fontvielle et al. 1988	8 (4M:4F)	T1DM, OP	43.5 (9.9)	NR	24.1 (6.8)		3.5	France	C	Neutral	3		Ad libitum, partial provision	Agency, Industry
Low GI						46.5(2.5)						46.1 (4.5):17.4 (1.4):35.0 (2.8)		
Higher GI						60.1 (5.1)						45.4 (4.5):16.9 (1.7):36.0 (2.8)		
CHD Trial														
Frost et al. 2004	55 (48M:7F)**	CHD, OP					NR	UK	P	Neutral §§§	12		Ad Libitum	Unknown
Low GI	26 (23M:3F)		63.6 (9.4)	81.2 (12.2)	26.9 (3.3)	50(4):115(39)						49 (5):18 (5):31 (5)		
Higher GI	29 (25M: 4F)		61.8 (9)	81.7 (16.7)	28.7 (4.6)	57(4):106(34)						47 (10):18 (5):32 (10)		

Abbreviations: BMI=Body Mass Index; C=Crossover design; Carb=Carbohydrate; CED= carbohydrate exchange diet; CHD= Coronary Heart Disease; CR= calorie restriction; CV=cardiovascular; GI= glycemic index; GL=glycemic load; IGT= impaired glucose tolerance; IP=inpatient; IR= insulin resistant; High Carb= high carbohydrate; HI= hyperinsulinemic; M = male; mo=months; MUFA=monounsaturated fatty acids; na=not available; T1DM=type 1 diabetes mellitus; T2DM = type 2 diabetes mellitus; OB= obese; OH=otherwise healthy; OP = outpatient; OW= overweight; P=Parallel design; SD= standard deviation; F = female; UK= United Kingdom; USA= United States of America; wks=weeks; y= years

§Negative energy balance diets were designed for weight loss, Neutral energy balance diets for weight maintenance, where all diets are isocaloric between test and control groups; §§Energy from carbohydrate: protein: fat for the planned diet or if not planned; §§§ Participants were advised to lose weight if they had a BMI>28kg/m2.

¶ Range of values; ¶¶ median and interquartile range (IQR); ¶ ¶ ¶ Actual GI/GL or if not available, planned;

* Calculated before dropout; **Completer Analysis, as used in data analysis; *** completer analysis, data obtained from authors; **** based on less participants than in analysis;

+ approximate based on all study arms; ¥ approximate based on test meals; ‡ approximate based on n=45 from Sloth et al. 2004, the original publication of this study;

‡‡‡ analysis includes weight loss and weight maintenance phases

Supplemental Table S4: Sensitivity analysis of the systematic removal of each trial*

	MD [95% CI], P-value I^2, P-value					
	Body Weight (kg) N=32	**BMI (kg/m^2) N=18**	**Body Fat (%) N=10**	**Waist Circumference (cm) N=18**	**Waist-to-hip Ratio N=6**	**Sagittal Abdominal Diameter (cm) N=3**
All studies	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.51	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.91	-0.01 [-0.58, 0.56], P=0.98 65%, P=0.003	-0.46 [-1.05, 0.14], P=0.13 62%, P<0.01	-0.00 [-0.01, 0.00], P=0.27 0.00%, P=0.52	-0.09 [-0.34, 0.16], P=0.48 0.00%, P=0.92
Removal of:						
OW/OB						
Pereira et al. 2015	-0.61 [-0.82, -0.39], P<0.01 0.00%, P=0.54	-0.24 [-0.34, -0.14], P<0.01 0.00%, P=0.93	0.19 [-0.25, 0.63], P=0.39 36%, P=0.13	-0.37 [-1.04, 0.31], P=0.28 62%, P<0.01	-0.00 [-0.01, 0.01], P=0.67 0.00%, P=0.58	n/a
Karl et al. 2015	-0.62 [-0.83, -0.41], P<0.01 0.00%, P=0.50	-0.25 [-0.35, -0.16], P<0.01 0.00%, P=0.91	0.10 [-0.51, 0.71], P=0.74 64%, P<0.01	n/a	n/a	n/a
Buscemi et al. 2013	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.86	-0.08 [-0.63, 0.48], P=0.79 64%, P<0.01	-0.46 [-1.06, 0.15], P=0.14 64%, P<0.01	n/a	n/a
Costa et al. 2012	-0.62 [-0.84, -0.41], P<0.01 0.00%, P=0.46	-0.26 [-0.37, -0.16], P<0.01 0.00%, P=0.86	-0.06 [-0.73, 0.61], P=0.86 66%, P<0.01	-0.33 [-0.94, 0.28], P=0.29 59%, P<0.01	n/a	n/a
Jebb et al. 2010 - LowFat	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
Larsen et al. 2010 - LowPro	-0.60 [-0.81, -0.38], P<0.01 0.00%, P=0.57	n/a	n/a	-0.45 [-1.09, 0.18], P=0.16 64%, P<0.01	n/a	-0.05 [-0.39, 0.29], P=0.79 0.00%, P=0.86
Jebb et al. 2010- HighMUFA	-0.64 [-0.85, -0.43], P<0.01 0.00%, P=0.65	n/a	n/a	n/a	n/a	n/a
Larsen et al. 2010 - HighPro	-0.62 [-0.83, -0.41], P<0.01 0.00%, P=0.48	n/a	n/a	-0.37 [-1.00, 0.26], P=0.26 62%, P<0.01	n/a	-0.13 [-0.45, 0.19], P=0.42 0.00%, P=0.92
Solomon et al. 2010	-0.63 [-0.84, -0.42], P<0.01	-0.26 [-0.36, -0.16], P<0.01	n/a	n/a	n/a	n/a

	0.00%, P=0.47	0.00%, P=0.88				
Philippou et al. 2009 - 6mnth	-0.65 [-0.86, -0.44], P<0.01 0.00%, P=0.59	-0.28 [-0.37, -0.18], P<0.01 0.00%, P=0.97	-0.12 [-0.71, 0.48], P=0.70 65%, P<0.01	-0.55 [-1.15, 0.04], P=0.07 61%, P<0.01	n/a	n/a
Philippou et al. 2009 - 4mnth	-0.61 [-0.83, -0.40], P<0.01 0.00%, P=0.48	-0.25 [-0.35, -0.15], P<0.01 0.00%, P=0.89	-0.09 [-0.72, 0.54], P=0.78 66%, P<0.01	-0.44 [-1.08, 0.19], P=0.17 64%, P<0.01	n/a	n/a
Abete et al. 2008	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	n/a	n/a	n/a	n/a	n/a
Philippou et al. 2008	-0.62 [-0.83, -0.42], P<0.01 0.00%, P=0.48	-0.26 [-0.35, -0.16], P<0.01 0.00%, P=0.89	-0.06 [-0.68, 0.55], P=0.84 68%, P<0.01	-0.41 [-1.02, 0.19], P=0.18 63%, P<0.01	n/a	n/a
Aston et al. 2008	-0.66 [-0.87, -0.44], P<0.01 0.00%, P=0.52	n/a	-0.00 [-0.70, 0.69], P=0.99 68%, P<0.01	-0.54 [-1.14, 0.07], P=0.08 62%, P<0.01	n/a	n/a
Jensen et al. 2008	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.27 [-0.36, -0.17], P<0.01 0.00%, P=0.88	n/a	-0.44 [-1.05, 0.18], P=0.16 64%, P<0.01	-0.00 [-0.01, 0.00], P=0.24 0.00%, P=0.41	-0.09 [-0.35, 0.18], P=0.51 0.00%, P=0.69
de Rougemont et al. 2007	-0.57 [-0.80, -0.34], P<0.01 0.00%, P=0.53	-0.25 [-0.36, -0.14], P<0.01 0.00%, P=0.87	0.06 [-0.57, 0.70], P=0.84 67%, P<0.01	n/a	n/a	n/a
Sichieri et al. 2007	-0.66 [-0.88, -0.45], P<0.01 0.00%, P=0.53	n/a	n/a	n/a	n/a	n/a
Bellisle et al. 2007	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.88	n/a	-0.47 [-1.08, 0.13], P=0.13 64%, P<0.01	-0.00 [-0.01, 0.00], P=0.14 0.00%, P=0.70	n/a
McMillan-Price et al. 2006 - HighCHO	-0.61 [-0.82, -0.39], P<0.01 0.00%, P=0.51	n/a	n/a	-0.38 [-1.01, 0.25], P=0.23 63%, P<0.01	n/a	n/a
McMillan-Price et al. 2006 - HighPro	-0.70 [-0.91, -0.49], P<0.01 0.00%, P=0.91	n/a	n/a	**-0.62 [-1.19, -0.05], P=0.03 55%, P<0.01**	n/a	n/a
Wolever et al. 2002	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
Diabetes:						
Visek et al. 2014	-0.63 [-0.84, -0.42], P<0.01	-0.26 [-0.36, -0.16], P<0.01	0.01 [-0.57, 0.60], P=0.96	n/a	n/a	n/a

	0.00%, P=0.46	0.00%, P=0.86	68%, P<0.01			
Jenkins et al. 2014	-0.66 [-0.88, -0.43], P<0.01 0.00%, P=0.47	-0.29 [-0.39, -0.18], P<0.01 0.00%, P=0.94	n/a	**-0.61 [-1.18, -0.04], P=0.04 50%, P=0.01**	-0.00 [-0.01, 0.00], P=0.21 0.00%, P=0.43	n/a
Jenkins et al. 2012	-0.62 [-0.84, -0.40], P<0.01 0.00%, P=0.46	-0.25 [-0.35, -0.15], P<0.01 0.00%, P=0.87	n/a	-0.44 [-1.05, 0.17], P=0.16 64%, P<0.01	-0.00 [-0.01, 0.01], P=0.64 0.00%, P=0.53	n/a
Yusof et al. 2009	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.87	n/a	-0.33 [-0.95, 0.28], P=0.29 58%, P<0.01	n/a	n/a
Jenkins et al. 2008	-0.61 [-0.83, -0.40], P<0.01 0.00%, P=0.48	-0.25 [-0.36, -0.15], P<0.01 0.00%, P=0.87	n/a	n/a	n/a	n/a
Wolever et al. 2008	-0.64 [-0.84, -0.43], P<0.01 0.00%, P=0.57	n/a	n/a	-0.50 [-1.10, 0.09], P=0.10 62%, P<0.01	n/a	n/a
Jimenez-Cruz et al. 2003	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.26 [-0.35, -0.16], P<0.01 0.00%, P=0.86	n/a	n/a	n/a	n/a
Heilbronn et al. 2002	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	n/a	n/a	n/a	n/a	n/a
Fontvieille et al. 1992	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
Fontvielle et al. 1988	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
CHD						
Frost et al. 2004	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	-0.26 [-0.36, -0.17], P<0.01 0.00%, P=0.89	n/a	-0.48 [-1.09, 0.12], P=0.12 63%, P<0.01	-0.00 [-0.01, 0.00], P=0.25 1%, P=0.40	n/a

*Sensitivity analysis included the removal of each single study from the meta-analyses one at a time and the summary effect was recalculated. An influential outlier was considered a study whose removal changed the magnitude of the pooled effect by >10%.

BMI, body mass index; CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; MD, mean difference; mnth, month; MUFA, monounsaturated fatty acids; n/a, not applicable; OB, obese; OW, overweight; Pro, protein

Supplemental Table S5: Sensitivity analyses of the use of correlation coefficients of 0.25 and 0.75 for crossover trials

	MD (95% CI), P-value I^2, P-value		
	Correlation Coefficient used in the Primary Analysis	Correlation Coefficient used in Sensitivity Analyses	
Outcome (no. crossover trials/total)	**0.5**	**0.25**	**0.75**
Body Weight, kg (6*/32)	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.51	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.51	-0.63 [-0.84, -0.43], P<0.01 0.00%, P=0.51
BMI, kg/m^2 (3/18)	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.91	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.90	-0.26 [-0.35, -0.17], P<0.01 0.00%, P=0.90
Body Fat, % (3*/10)	-0.01 [-0.58, 0.56], P=0.98 65%, P<0.01	-0.00 [-0.58, 0.58], P=0.99 64%, P<0.01	-0.02 [-0.57, 0.54], P=0.96 66%, P<0.01
Waist Circumference, cm (2*/18)	-0.46 [-1.05, 0.14], P=0.13 62%, P<0.01	-0.44 [-1.04, 0.15], P=0.14 60%, P<0.01	-0.46 [-1.07, 0.14], P=0.13 65%, P<0.01
Waist-to-hip Ratio (0/6)	-0.00 [-0.01, 0.00], P=0.27 0.00%, P=0.52	n/a	n/a
Sagittal Abdominal Diameter, cm (0/6)	-0.09 [-0.34, 0.16], P=0.48 0.00%, P=0.92	n/a	n/a

* one of these crossover trials, however, did not require the use of a correlation coefficient as complete data was available

BMI, body mass index; CI, confidence interval; MD, mean difference; no., number

Supplement Table S6. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on body weight (kg)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	24.1 – 37.1 kg/m^2	32	2448	-0.04 (-0.13, 0.06)	0.00%	0.454
Follow-up	3 – 72 wks	32	2448	0.01 (-0.01, 0.02)	0.00%	0.314
Dose Pasta	1.75 – 7.0 serv/wk	11	740	0.29 (-0.91, 1.50)	0.00%	0.595
GI[3]	21 - 72	31	2383	-0.01 (-0.04, 0.02)	0.45%	0.482
Difference in GI[2]	4.9 - 40.2	31	2383	-0.00 (-0.04, 0.03)	2.75%	0.811
Fiber[3]	8.0 - 39.4g/d	27	1851	0.02 (-0.01, 0.06)	0.00%	0.221
Change in Fiber[4]	-7.5 - +13.8g/d	17	1571	-0.02 (-0.07, 0.04)	23.13%	0.595
Difference in Fiber[2]	-8.5 - +15.3g/d	27	1851	-0.01 (-0.06, 0.04)	0.00%	0.701
Saturated Fat[3]	5.1 - 12.5%	14	1396	-0.05 (-0.30, 0.20)	34.32%	0.648
Change in Saturated Fat[4]	-8.2 - -1.2%	12	1309	-0.01 (-0.29, 0.28)	46.27%	0.965
Difference in Saturated Fat[2]	-2.0 - +2.3%	14	1396	-0.34 (-1.08, 0.39)	32.80%	0.329
CHO[3]	37.2 - 68.0%	30	2345	-0.01 (-0.05, 0.03)	0.00%	0.541
Change in CHO[4]	-7.2 - +10.1%	19	2046	-0.07 (-0.12, -0.01)	0.00%	**0.016**
Difference in CHO[2]	-11.1 - +5.4%	30	2345	0.02 (-0.05, 0.10)	0.00%	0.547
Protein[3]	13.3 - 26.1%	30	2345	0.04 (-0.04, 0.12)	0.00%	0.332

Change in Protein[4]	-0.5 - +8.6%	19	2046	0.15 (0.03, 0.27)	0.00%	**0.017**
Difference in Protein[2]	-2.5 - +3.4%	30	2345	-0.18 (-0.38, 0.02)	0.00%	0.069
Fat[3]	16.0 - 37.7%	30	2345	0.01 (-0.05, 0.06)	0.00%	0.852
Change in Fat[4]	-12.2 - +5.4%	19	2046	0.05 (-0.02, 0.12)	17.73%	0.122
Difference in Fat[2]	-4.4 - +10.6%	30	2345	0.02 (-0.05, 0.09)	0.00%	0.610

1 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 body weight on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in body weight. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.

2 Difference in diet variable between the intervention and control arms

3 Intake at the end of study in the intervention arm

4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index

Supplemental Table S7. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on BMI (kg/m^2)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	26.3 – 37.1 kg/m^2	18	1038	-0.01 (-0.06, 0.03)	0.00%	0.559
Follow-up	4 – 24 wks	18	1038	0.00 (-0.01, 0.02)	0.00%	0.559
Dose Pasta*	2.33 – 7.0 serv/wk	4	143			
GI[3]	39.8 - 72	17	973	0.01 (-0.01, 0.03)	0.00%	0.153
Difference in GI[2]	7.0 - 40.2	17	973	-0.01 (-0.03, 0.01)	0.00%	0.275
Fiber[3]	8.0 - 39.4g/d	16	935	0.01 (-0.01, 0.02)	0.00%	0.366
Change in Fiber[4]	-1.8 - +12.4g/d	10	758	0.00 (-0.03, 0.03)	0.00%	0.846
Difference in Fiber[2]	-4.9 - +13.0g/d	16	935	-0.00 (-0.02, 0.02)	0.00%	0.821
Saturated Fat*[3]	7.6 - 12.5%	7				
Change in Saturated Fat*[4]	-2.4 - -1.2%	6				
Difference in Saturated Fat*[2]	-1.0 - +2.3%	7				
CHO[3]	37.2 - 68.0%	16	935	-0.00 (-0.02, 0.01)	0.00%	0.594
Change in CHO[4]	-5.6 - +3.2%	10	758	-0.02 (-0.06, 0.02)	0.00%	0.325
Difference in CHO[2]	-11.1 - +2.0%	16	935	-0.01 (-0.03, 0.02)	0.00%	0.531
Protein[3]	13.9 – 22.8%	16	935	0.00 (-0.04, 0.04)	0.00%	0.905

Change in Protein[4]	-0.2 - +3.0%	10	758	0.01 (-0.11, 0.14)	0.00%	0.786
Difference in Protein[2]	-2.5 - +3.4%	16	935	-0.03 (-0.12, 0.06)	0.00%	0.441
Fat[3]	16.0 - 37.7%	16	935	0.00 (-0.02, 0.02)	0.00%	0.717
Change in Fat[4]	-4.8 - +5.4%	10	758	0.02 (-0.02, 0.06)	0.00%	0.240
Difference in Fat[2]	-4.4 - +10.6%	16	935	0.01 (-0.01, 0.04)	0.00%	0.299

*For Dose, there were <10 trials so subgroup analyses were not performed.

1 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 BMI on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in BMI. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.

2 Difference in diet variable between the intervention and control arms

3 Intake at the end of study in the intervention arm

4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index; wk, week

Supplemental Table S8. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on body fat (%)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	26.3 – 36.0 kg/m^2	10	285	-0.02 (-0.26, 0.23)	67.69%	0.890
Follow-up	4 – 24 wks	10	285	0.06 (-0.06, 0.17)	66.00%	0.303
Dose Pasta*	2.33 - 3.33 serv/wk	3	96			
GI[3]	39.8 - 72	10	285	0.12 (-0.00, 0.25)	52.21%	0.053
Difference in GI[2]	7.0 - 40.2	10	285	-0.09 (-0.15, -0.03)	19.39%	**0.008**
Fiber*[3]	8.0 - 39.4g/d	8	228			
Change in Fiber*[4]	-1.8 - +12.4g/d	4	87			
Difference in Fiber*[2]	-4.9 - +13.0g/d	8	228			
Saturated Fat*[3]	7.6 - 12.5%	3	93			
Change in Saturated Fat*[4]	-2.4 - -1.2%	2	51			
Difference in Saturated Fat*[2]	-1.0 - +2.3%	3	93			
CHO*[3]	37.2 - 68.0%	9	247			
Change in CHO*[4]	-5.6 - +3.2%	4	87			
Difference in CHO*[2]	-11.1 - +2.0%	9	247			
Protein*[3]	13.9 – 22.8%	9	247			

Change in Protein*[4]	-0.2 - +3.0%	4	87			
Difference in Protein*[2]	-2.5 - +3.4%	9	247			
Fat*[3]	16.0 - 37.7%	9	247			
Change in Fat*[4]	-4.8 - +5.4%	4	87			
Difference in Fat*[2]	-4.4 - +10.6%	9	247			

*There were <10 trials so subgroup analyses were not performed.

1 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 body fat on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in body fat. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.
2 Difference in diet variable between the intervention and control arms
3 Intake at the end of study in the intervention arm
4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index; wk, week

Supplemental Table S9. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on waist circumference (cm)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	26.3 – 36.0 kg/m^2	18	1380	0.10 (-0.13, 0.32)	59.02%	0.372
Follow-up	4 – 52 wks	18	1380	0.05 (-0.03, 0.13)	60.50%	0.225
Dose Pasta*	3.0 – 3.33 serv/wk	2	63			
GI[3]	41.2 – 72.0	17	1315	-0.01 (-0.12, 0.10)	66.00%	0.841
Difference in GI[2]	4.9 – 32.9	17	1315	-0.02 (-0.10, 0.06)	64.51%	0.596
Fiber[3]	8.0 - 39.4g/d	15	1258	0.05 (-0.05, 0.14)	59.60%	0.342
Change in Fiber[4]	-1.8 - +13.8g/d	13	1176	0.01 (-0.17, 0.20)	70.59%	0.861
Difference in Fiber[2]	-4.9 - +15.3g/d	15	1258	-0.00 (-0.14, 0.13)	64.86%	0.939
Saturated Fat*[3]	6.1 – 10.1%	8	604			
Change in Saturated Fat*[4]	-7.6 - -1.2%	7	562			
Difference in Saturated Fat*[2]	-2.0 - +2.3%	8	604			
CHO[3]	38.5 - 58.6%	16	1277	-0.11 (-0.19, -0.04)	27.06%	**0.007**
Change in CHO[4]	-7.2 - +10.1%	13	1176	-0.09 (-0.23, 0.04)	57.19%	0.148
Difference in CHO[2]	-11.1 - +5.4%	16	1277	-0.01 (-0.18, 0.17)	60.81%	0.947
Protein[3]	13.9 – 26.1%	16	1277	0.20 (0.01, 0.38)	43.92%	**0.038**

Change in Protein[4]	-0.3 - +8.6%	13	1176	0.19 (-0.09, 0.46)	63.38%	0.165
Difference in Protein[2]	-2.5 - +3.4%	16	1277	-0.14 (-0.66, 0.38)	62.79%	0.570
Fat[3]	22.0 - 37.2%	16	1277	0.10 (-0.05, 0.26)	53.48%	0.185
Change in Fat[4]	-12.2 - +5.4%	13	1176	0.08 (-0.07, 0.24)	61.15%	0.274
Difference in Fat[2]	-4.4 - +10.6%	16	1277	0.09 (-0.06, 0.25)	47.17%	0.220

*There were <10 trials so subgroup analyses were not performed.

1 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 waist circumference on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in waist circumference. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.

2 Difference in diet variable between the intervention and control arms

3 Intake at the end of study in the intervention arm

4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index; wk, week

Supplementary Table S10. Post-hoc piecewise linear continuous dose-response meta-regression analyses for the effect of pasta intake in the context of low-GI dietary patterns on body weight (kg)

Dose threshold of servings of pasta consumed per week	Dose ranges of servings of pasta consumed per week	β (95% CIs) *	Residual I^2 †	*p*-value
3.0	≤3.0	-0.70 (–3.27, 1.86)	0.00%	0.890
	>3.0	0.91 (–0.89, 2.70)		
3.33	≤3.33	0.05 (–1.80, 1.89)	0.00%	0.518
	>3.33	0.44 (–1.75, 2.63)		
3.5	≤3.5	0.09 (–1.65, 1.82)	0.00%	0.888
	>3.5	0.46 (–1.89, 2.81)		

* β is the slope derived from the piecewise linear meta-regression analyses and represents the treatment effect on body weight for doses above and below each dose-threshold representing servings of pasta consumption per week; † The residual I^2 value indicates heterogeneity unexplained by each dose-threshold.

Supplementary Table S11: GRADE assessment of study quality

Quality assessment*							№ of patients		Effect	Quality Importance
№ of studies	**Design**	**Risk of bias**	**Inconsistency**	**Indirectness**	**Imprecision**	**Publication Bias**	**Pasta/LGI diet**	**Higher GI diet**	**Absolute (95% CI)**	
Body Weight (follow up: median 12 weeks)										
32	randomised trials	not serious	not serious	serious [a]	not serious	none	1294	1228	MD **-0.63 kg** (-0.84 to -0.42)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness
BMI (follow up: median 12 weeks)										
18	randomised trials	not serious	not serious	serious [a]	not serious	none	551	538	MD **-0.26 kg/m^2** (-0.36 to -0.16)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness
Waist Circumference (follow up: median 12 weeks)										
18	randomised trials	not serious	serious [b]	serious [a]	not serious	none	717	624	MD **-0.46 cm** (-1.05 to 0.14)	⊕⊕◯◯ LOW [a,b] Due to downgrade for inconsistency and indirectness

Quality assessment*							№ of patients		Effect	Quality Importance
№ of studies	Design	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Pasta/LGI diet	Higher GI diet	Absolute (95% CI)	
Body Fat (follow up: median 12 weeks)										
10	randomised trials	not serious	serious [c]	serious [a]	not serious	none	176	165	MD **-0.01 %** (-0.58 to 0.56)	⊕⊕◯◯ LOW [a,c] Due to downgrade for inconsistency and indirectness
Waist-to-hip Ratio (follow up: median 12 weeks)										
6	randomised trials	not serious	not serious	serious [a]	not serious	none [d]	223	222	MD **-0.00** (-0.01 to 0.00)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness
Sagittal Abdominal Diameter (follow up: median 26 weeks)										
3	randomised trials	not serious	not serious	serious [a]	not serious	none [d]	237	214	MD **-0.09 cm** (-0.34 to 0.16)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness

CI, Confidence interval; GI, glycemic index; LGI, low glycemic index; MD, Mean difference

*All outcomes started with high quality evidence since all studies were randomized controlled trials. Risk of Bias –We rated down for risk of bias if the majority of studies were considered to be at high risk of bias. Inconsistency – We assessed inconsistency using I^2 estimates where an I^2 of 50% or higher indicates substantial heterogeneity. I^2 is the percentage of variability in the treatment estimates that is attributable to heterogeneity between studies. We rated down for inconsistency if there was substantial heterogeneity that was unexplained by any a priori sensitivity or subgroup analyses. Indirectness – We rated down for indirectness if there were factors present relating to the population, interventions, and outcomes that limited the generalizability of the results. Imprecision – We rated down for imprecision if the 95% confidence interval (95% CI) crossed the minimally important difference (MID) for harm. MIDs used for each outcome are: 0.5kg for body weight (based on Johnston et al. JAMA 2014;312(9):923-33); 0.2kg/m^2 for BMI; 2.0cm for waist circumference; 2.0% for body fat; 0.02 for waist-to-hip ratio; 2.0cm for sagittal abdominal diameter.

a. Downgrade for serious indirectness, since most of the available trials did not quantify the amount of pasta consumed in the context of the low-GI dietary patterns

b. Downgrade for serious inconsistency, as there was evidence of substantial inter-study (I^2=62%, P-heterogeneity<0.001), which could not be explained

c. Downgrade for serious inconsistency, as there was evidence of substantial inter-study (I^2= 65%, P-heterogeneity=0.003), which could not be explained

d. No downgrade for publication bias, as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (<10 trials included in the meta-analysis)

Supplemental Table S12: PRISMA Checklist

PRISMA 2009 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.	2-3
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	4
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	4
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 Table S2
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, Supplemental Table S1
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	Supplemental Table S1

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, Figure 1
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
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, Supplemental Table S2
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
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	7
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.	7-9

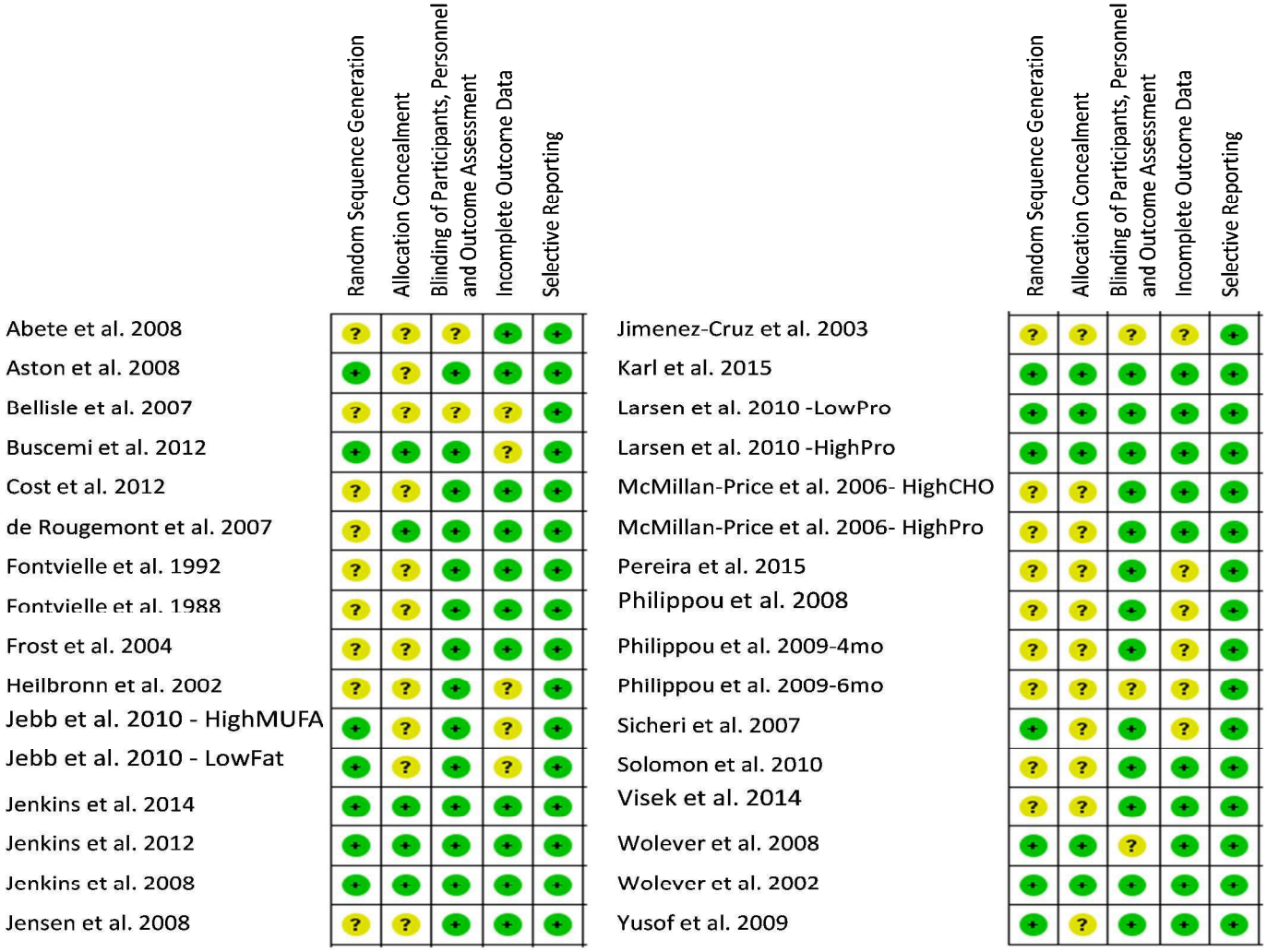
Section/topic	#	Checklist item	Reported on page #
Risk of bias across studies	15	Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies).	9
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	7-8
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.	9-11, 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.	10-11, Table 1, Supplemental Tables S2-S3
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).	11, 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.	11-12, Figures 2-3, Supplemental Figures S3-S7
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	11-12, Figures 2-3, Supplemental Figures S3-S7
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	11, Supplemental Table S1, S2, S12, S15, S17, S20
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	12-15, Supplemental Tables S4-S10, Supplemental Figures S8-S26
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).	16-19
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).	19-21
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	21-22
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.	25-26

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

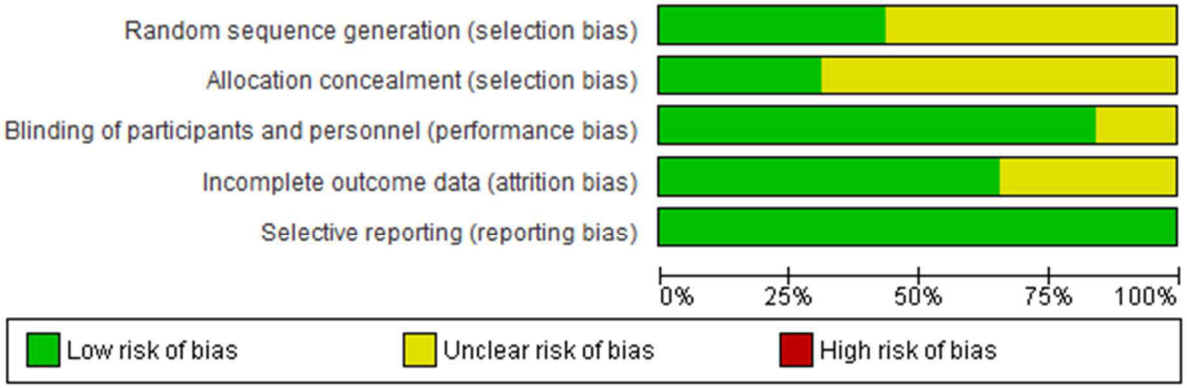
For more information, visit: **www.prisma-statement.org**.

Supplemental Figures

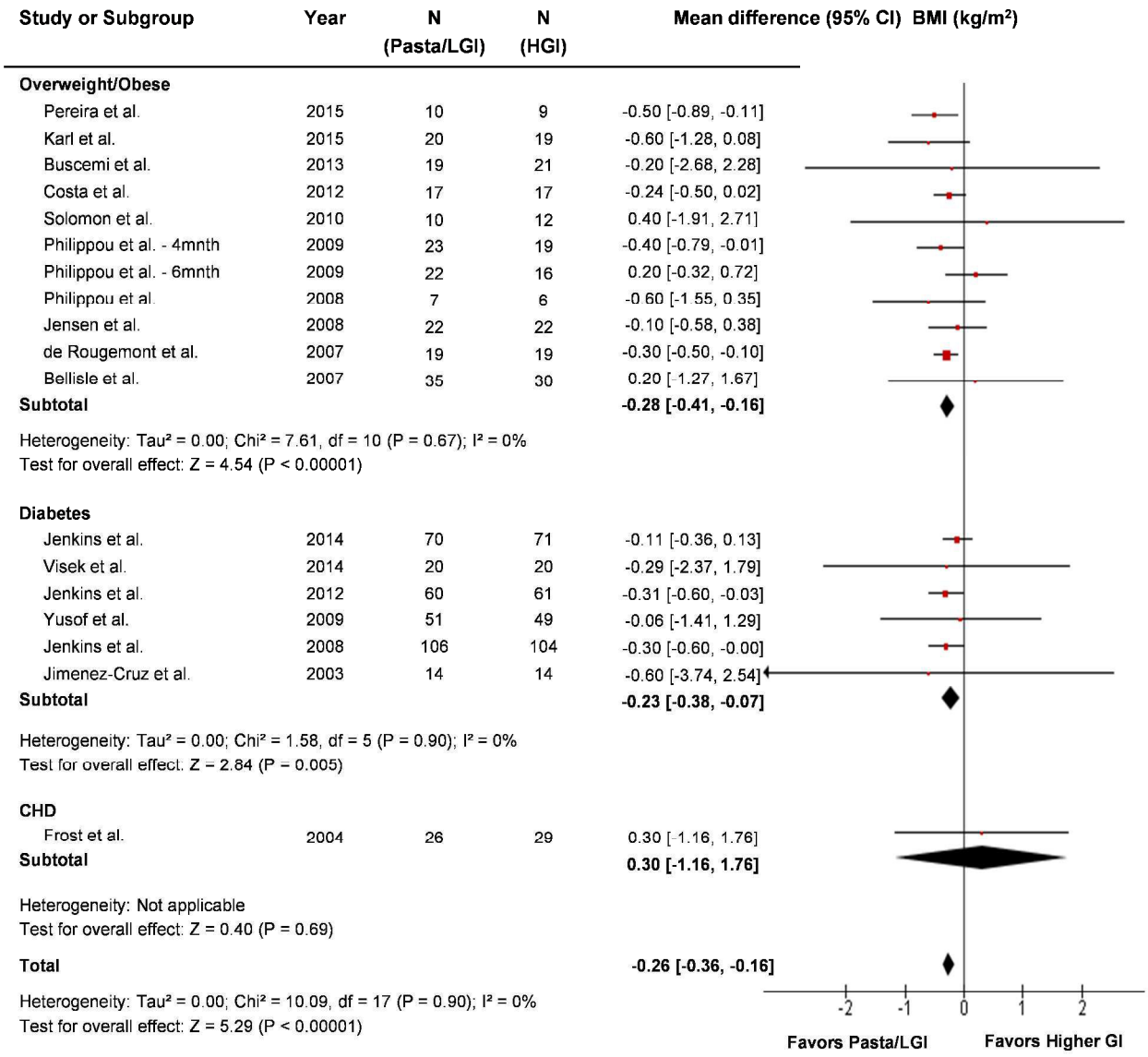


Supplemental Figure S1: Cochrane risk of bias summary for all included trials

Summary of risk of bias ratings for each individual study included in the meta-analysis.



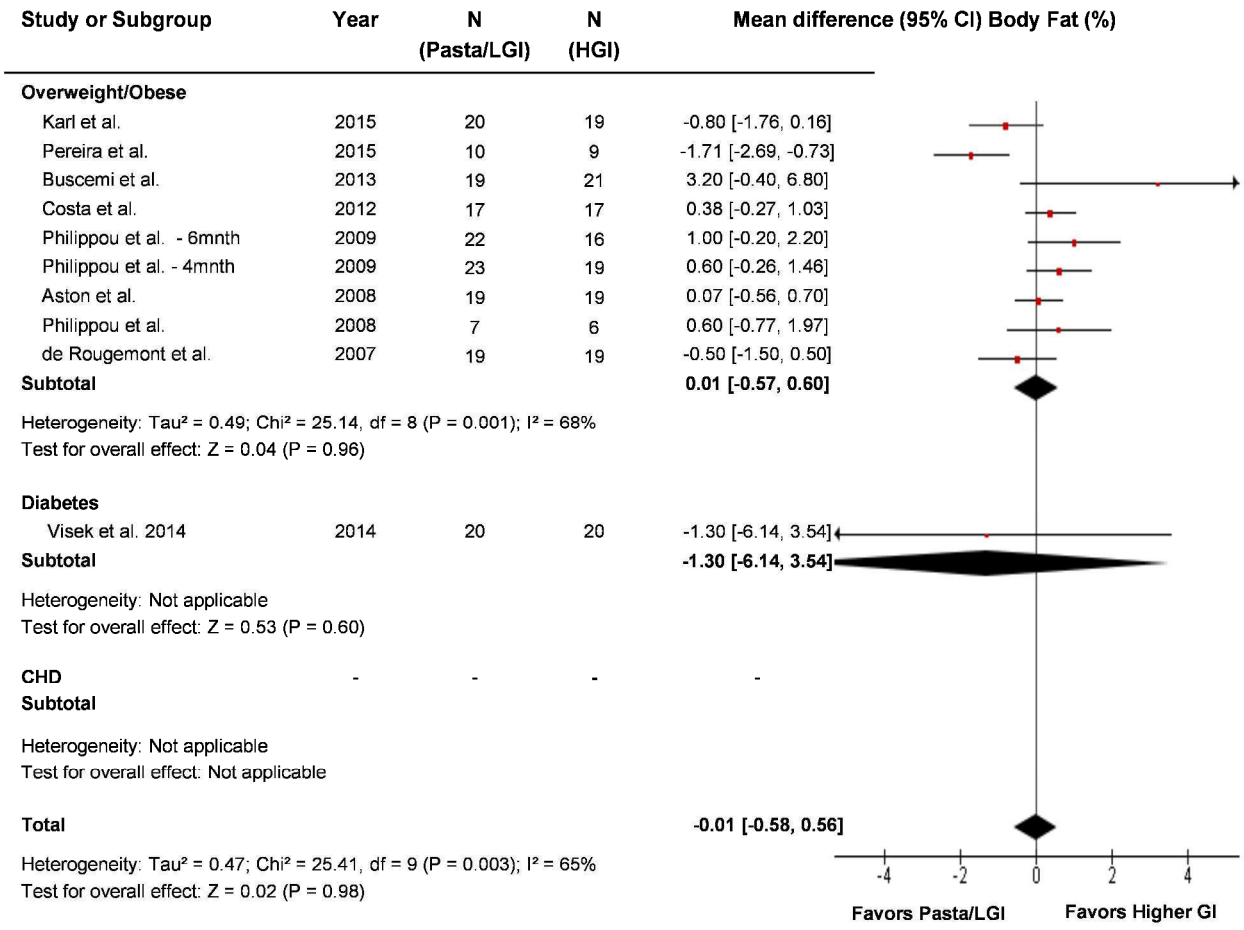
Supplemental Figure S2: Risk of bias proportion graph for all included trials



Supplemental Figure S3: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on BMI (kg/m^2) (n= 1038)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.

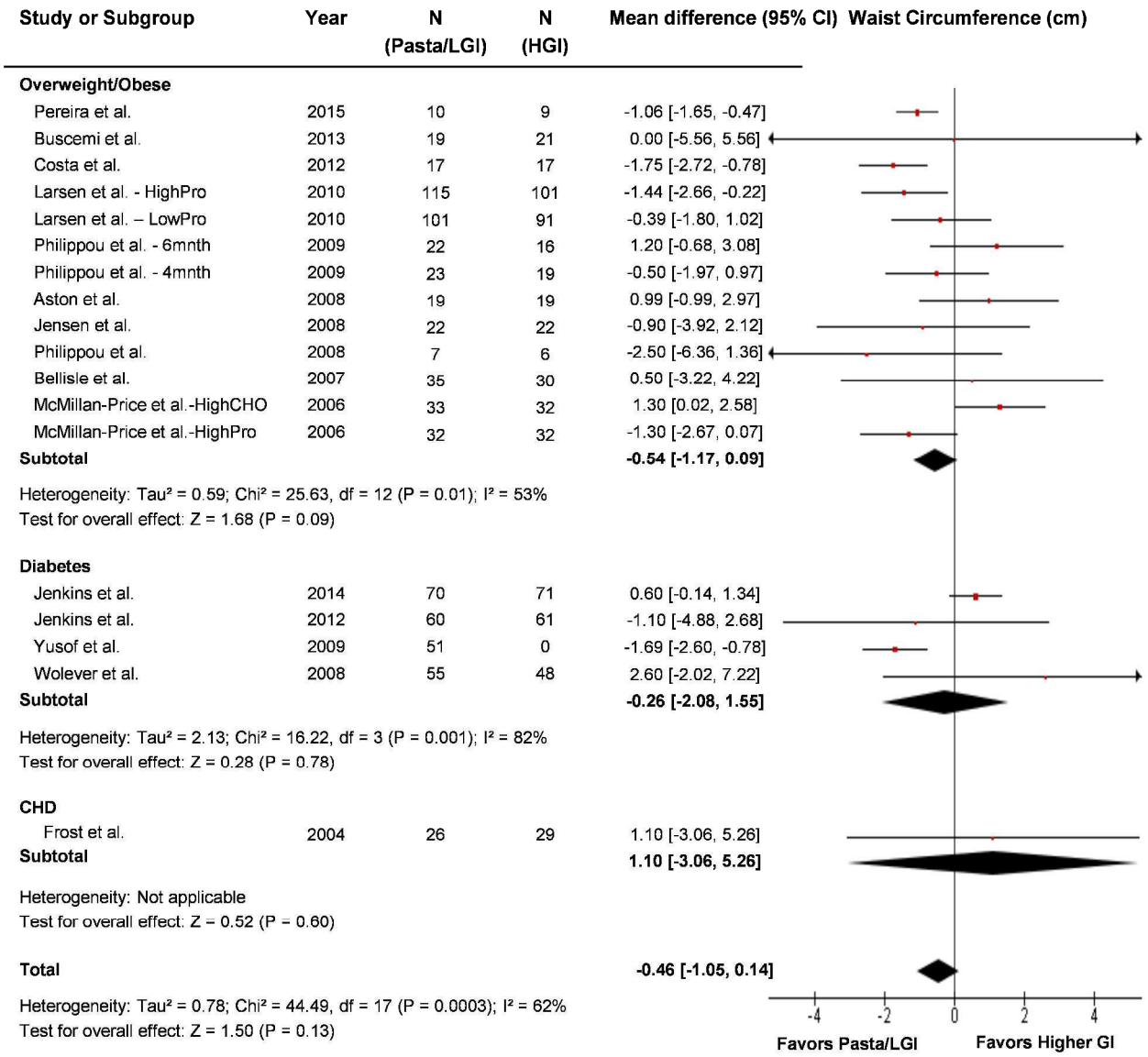
BMI, body mass index; CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet



Supplemental Figure S4: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body fat (%) (n= 285)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

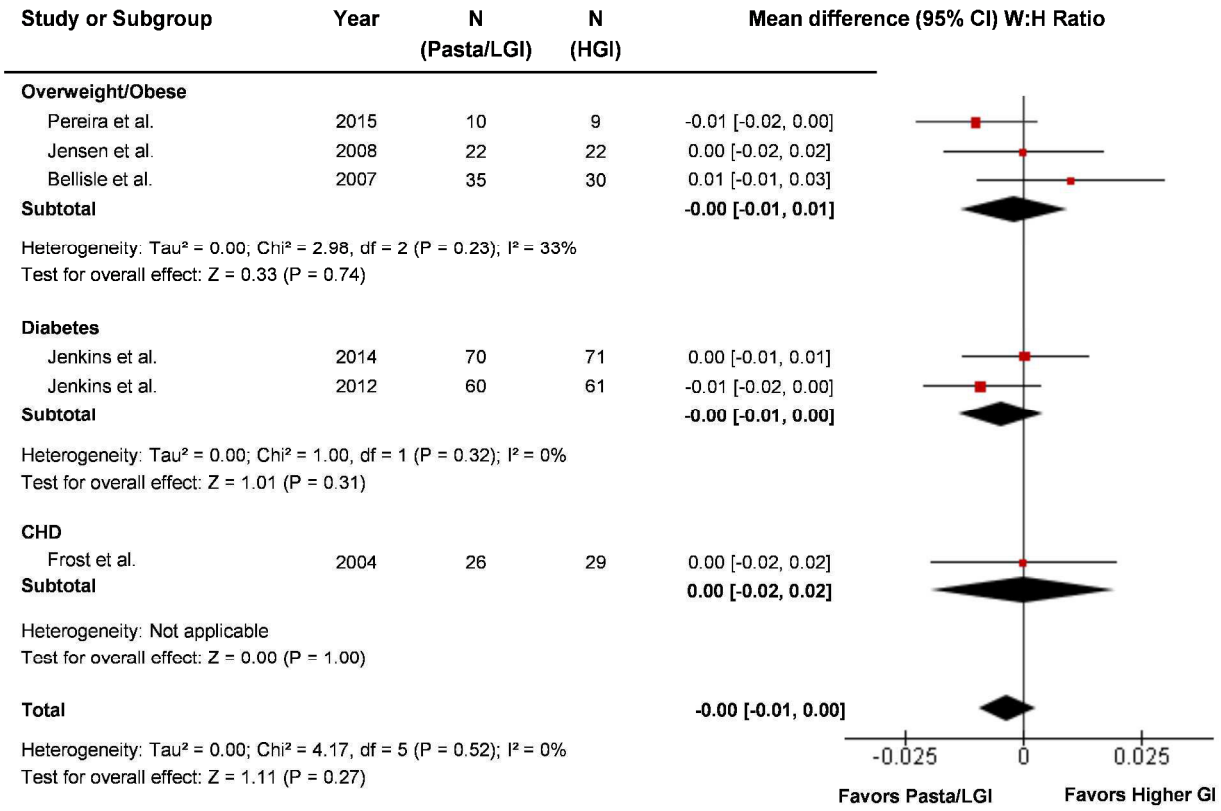
CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet



Supplemental Figure S5: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on waist circumference (cm) (n = 1380)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

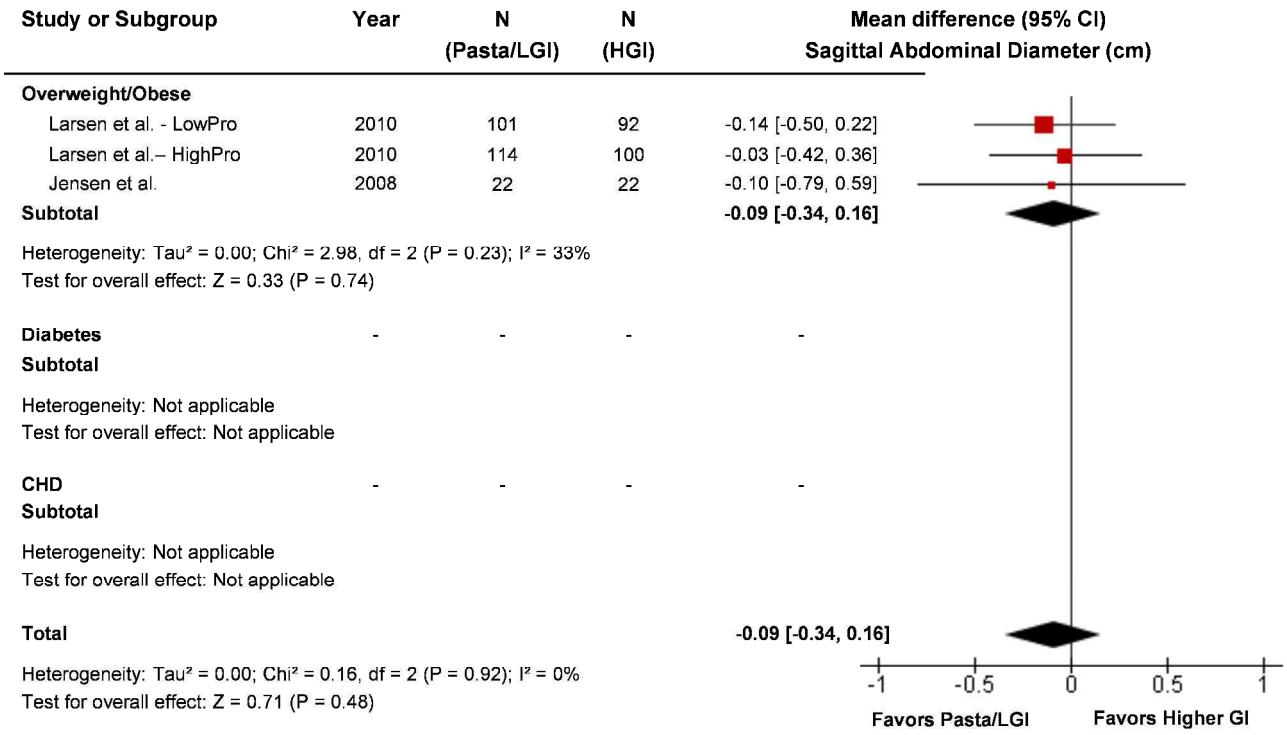
CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; Pro, protein



Supplemental Figure S6: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on waist-to-hip ratio (n = 445)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

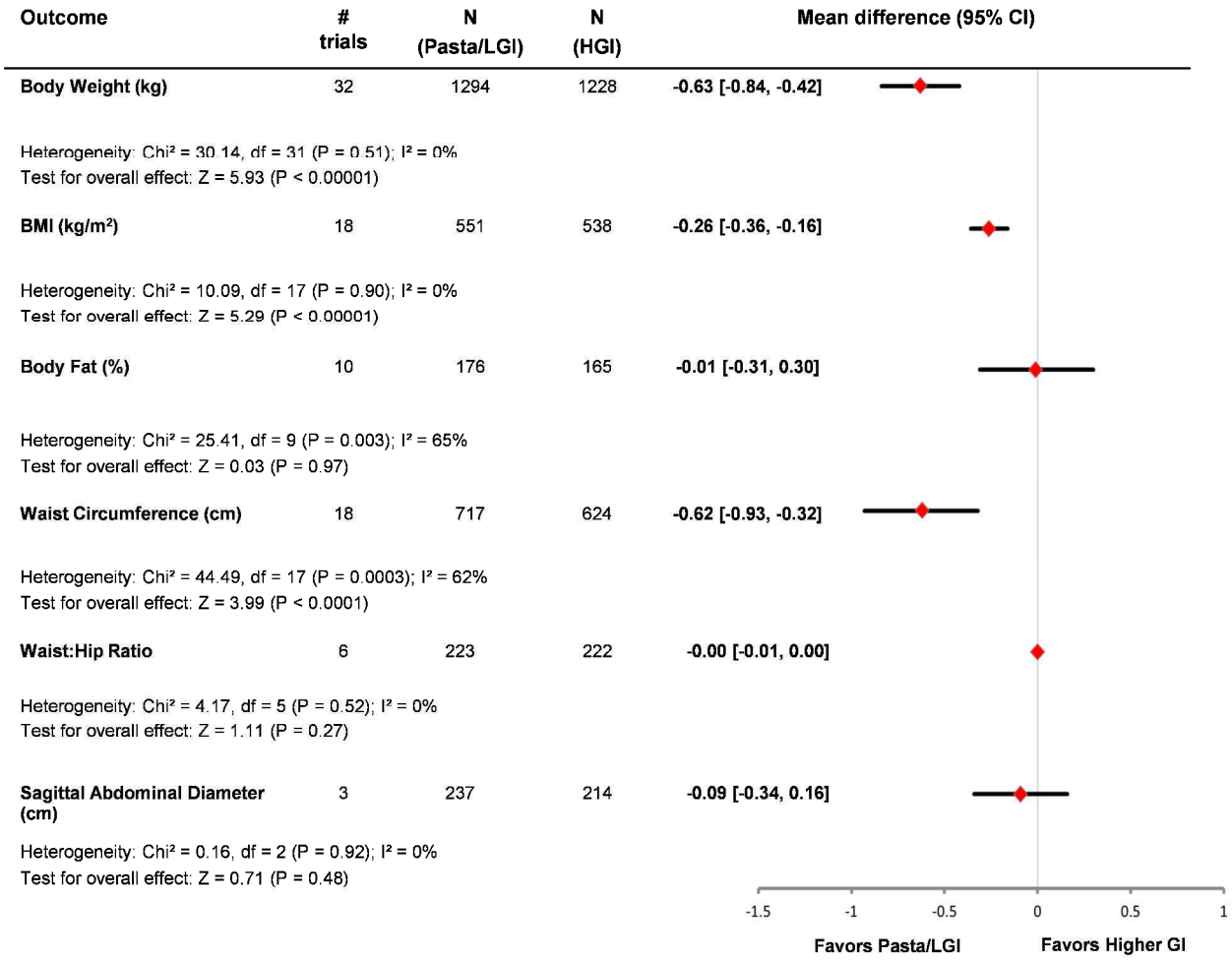
CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet



Supplemental Figure S7: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on sagittal abdominal diameter (cm) (n = 478)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

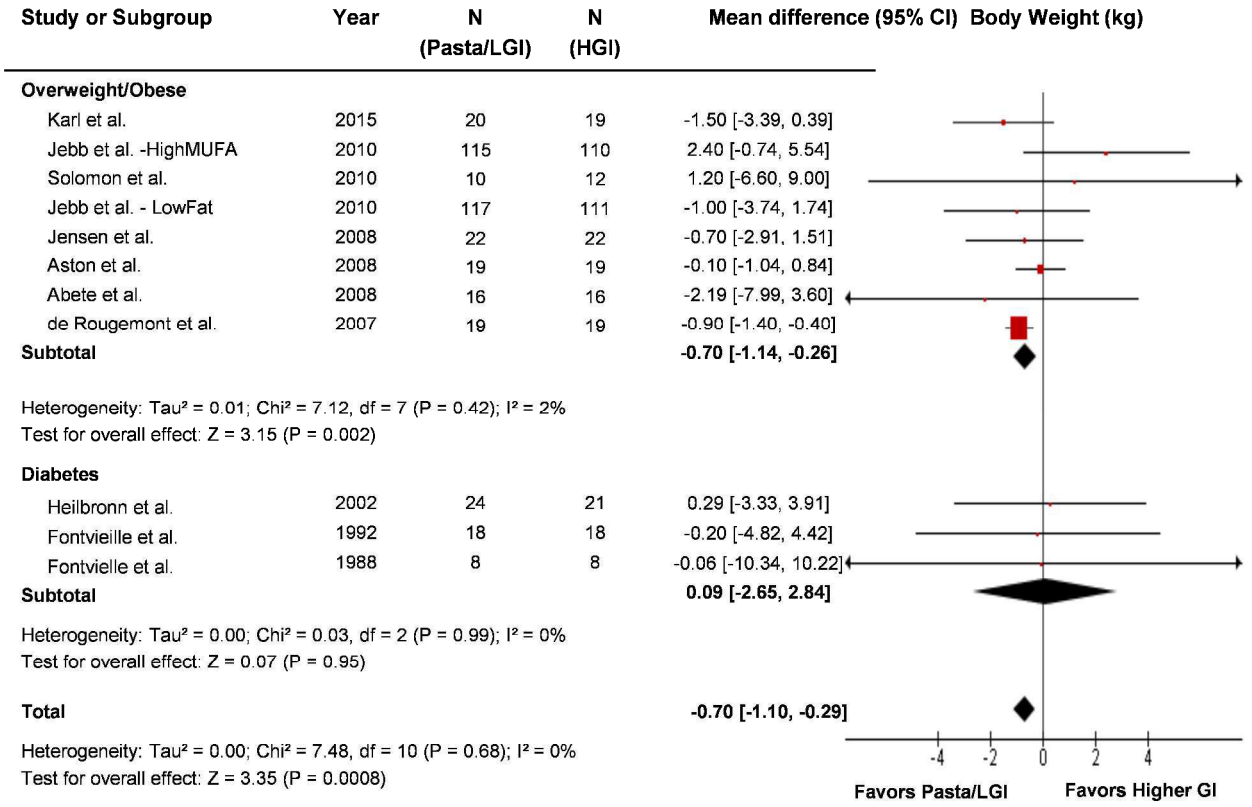
CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; Pro, protein



Supplemental Figure S8: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg) using fixed effects models (n = 2448)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance fixed effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

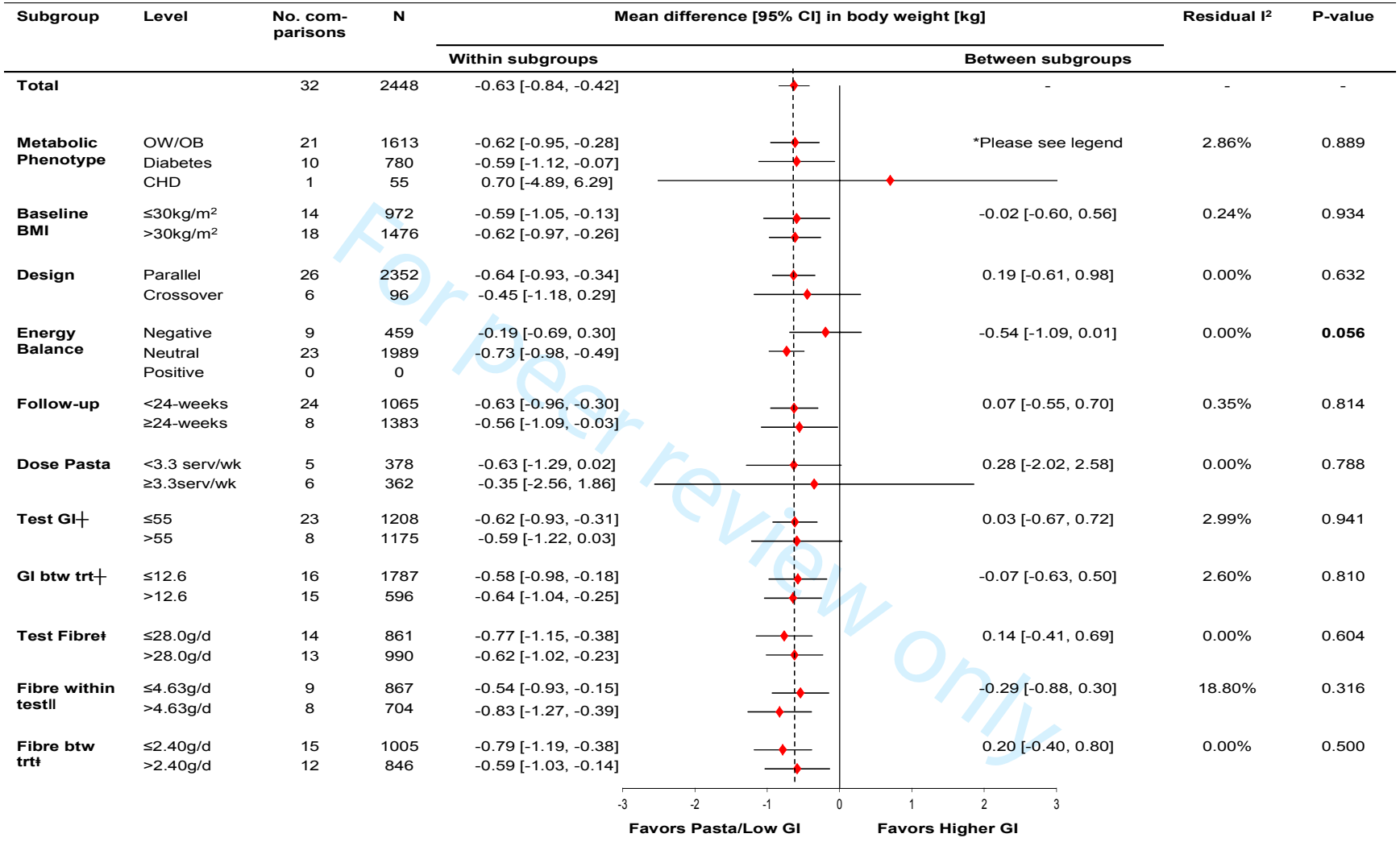
CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids; Pro, protein.



Supplemental Figure S9: Forest plot of randomized controlled trials which contain data for approximating pasta intake which are investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg) (n = 740)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids

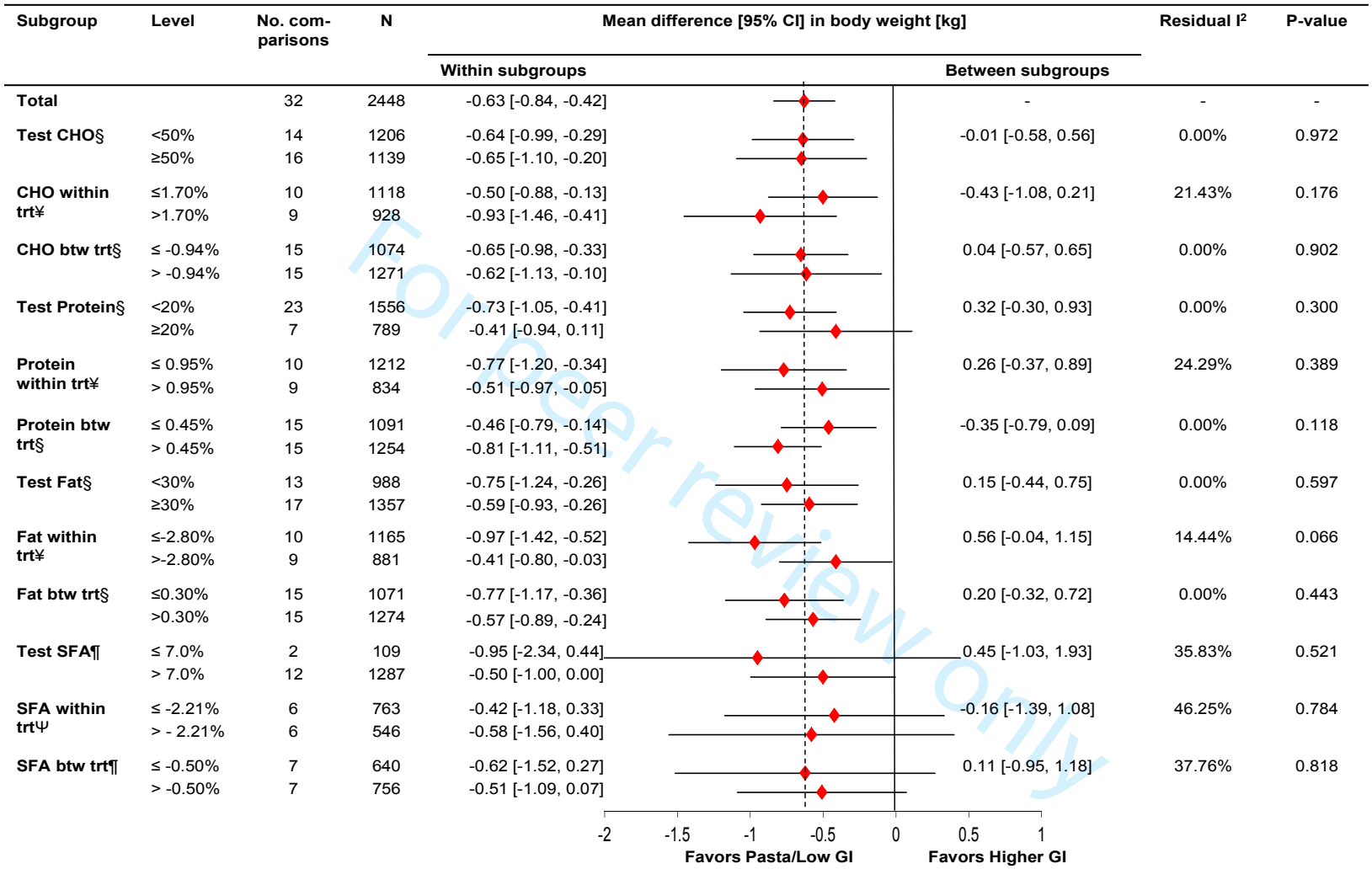


Supplemental Figure S10: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body weight (kg) (n = 2448)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body weight. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes.

BMI, body mass index; btw, between; CHD, coronary heart disease; CI, confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; serv, serving; trt, treatment; wk, week.

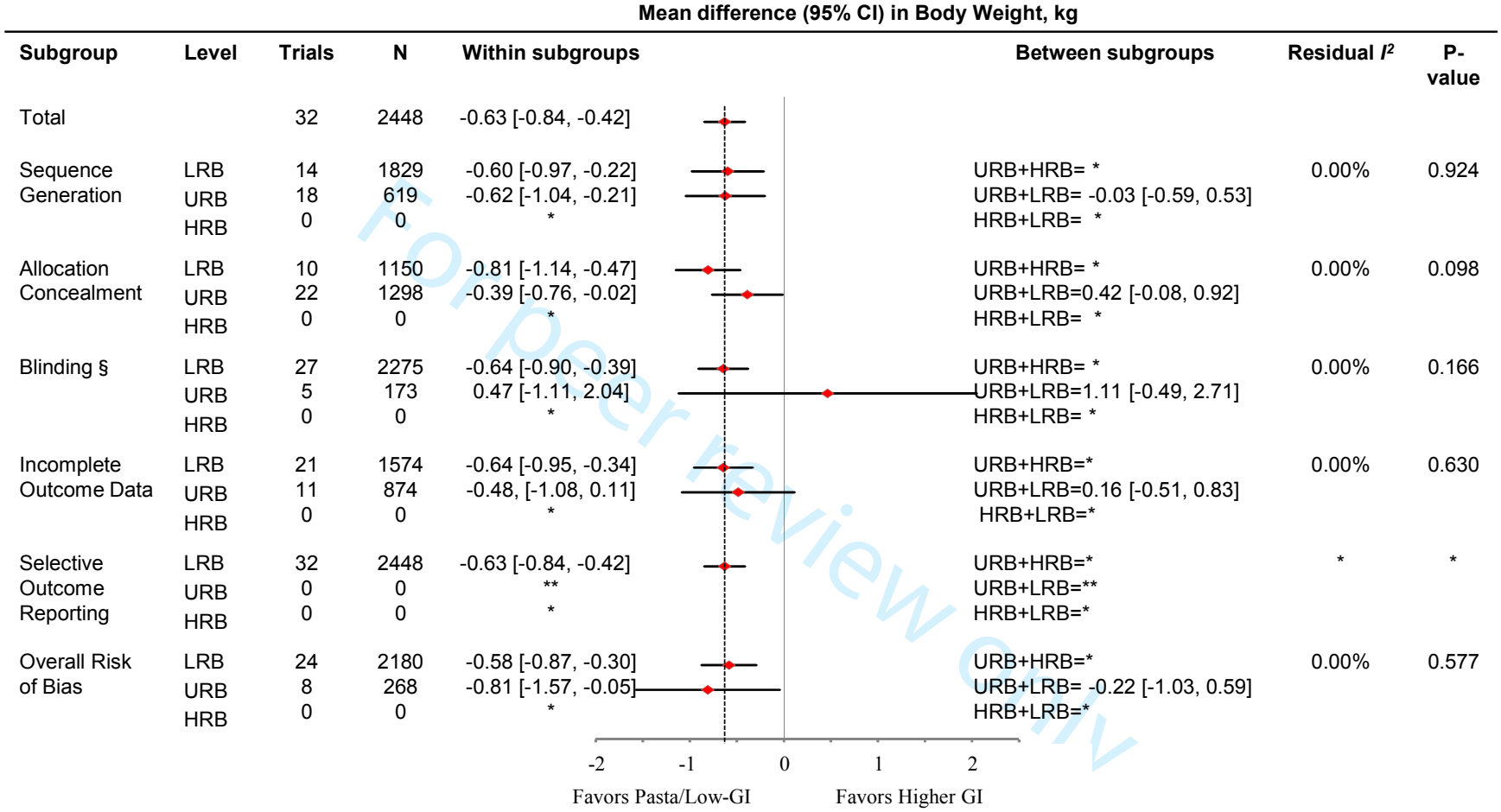
*Pairwise between-subgroup mean differences (95% CIs) for Metabolic Phenotype were as follows: 0.02kg (-0.08, 0.65) (1 vs. 2) to 1.32kg (-4.28, 6.91) (1 vs. 3) to -1.29kg (-6.91, 4.32) (2 vs. 3).
┼ data available on 31 studies
‖ data available on 17 studies
ǂ data available on 27 studies



Supplemental Figure S11: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body weight (kg) continued (n = 2448)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body weight. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes.

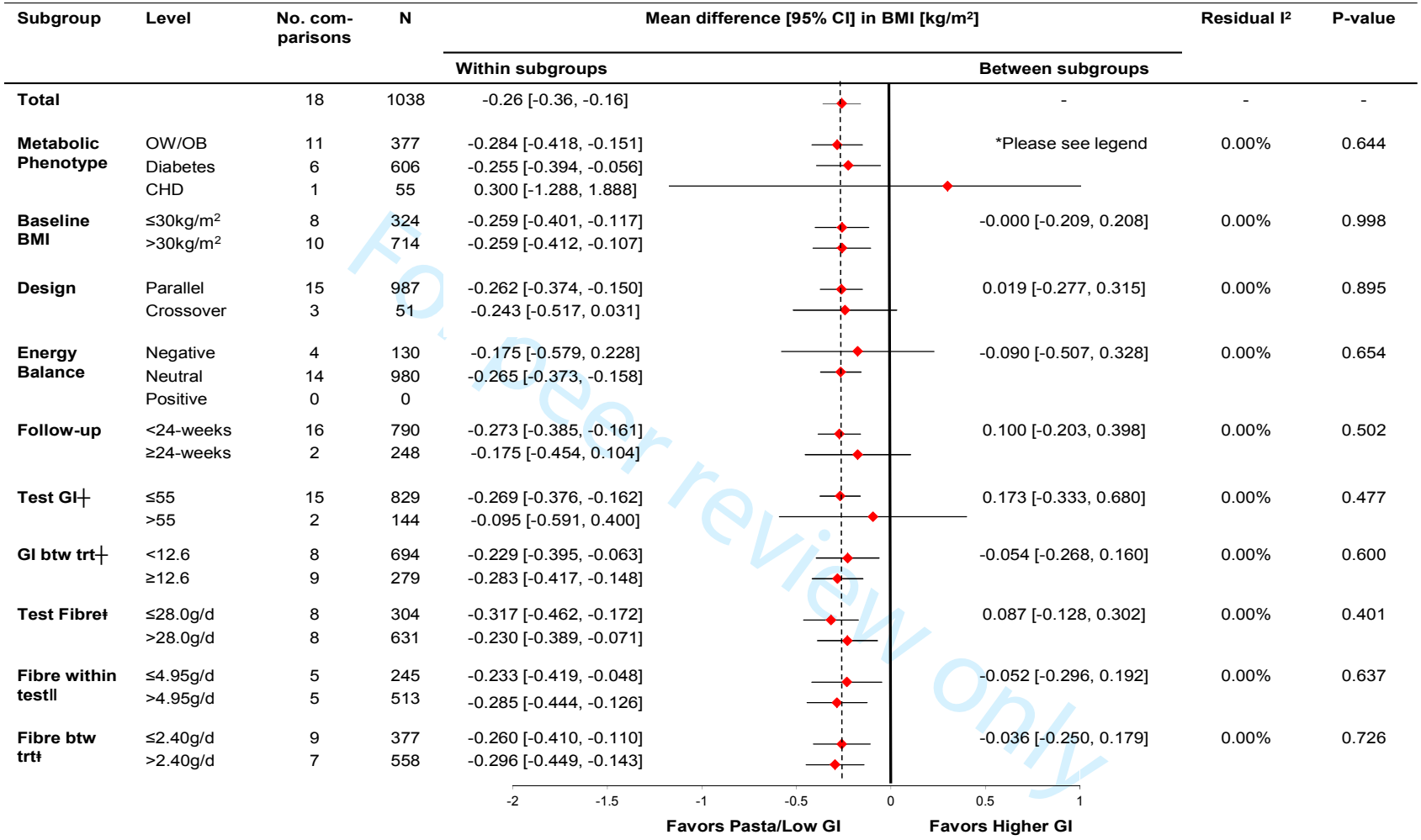
BMI, body mass index; btw, between; CI, confidence interval; GI, glycemic index (glucose scale); trt, treatment.
§ data available on 30 studies
¥ data available on 19 studies
¶ data available on 14 studies
Ψ data available on 12 studies



Supplemental Figure S12: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on body weight (kg) (n = 2448)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body weight. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

LRB=Low Risk of Bias, URB= Unclear Risk of Bias, HRB=High Risk of Bias. *Within and between subgroup analysis could not be performed against HRB since no values for HRB subgroup. ** no values for URB subgroup. § Blinding of Participants, Personnel, and Outcome Assessors.



Supplemental Figure S13: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body mass index, BMI (kg/m^2) (n = 1038)

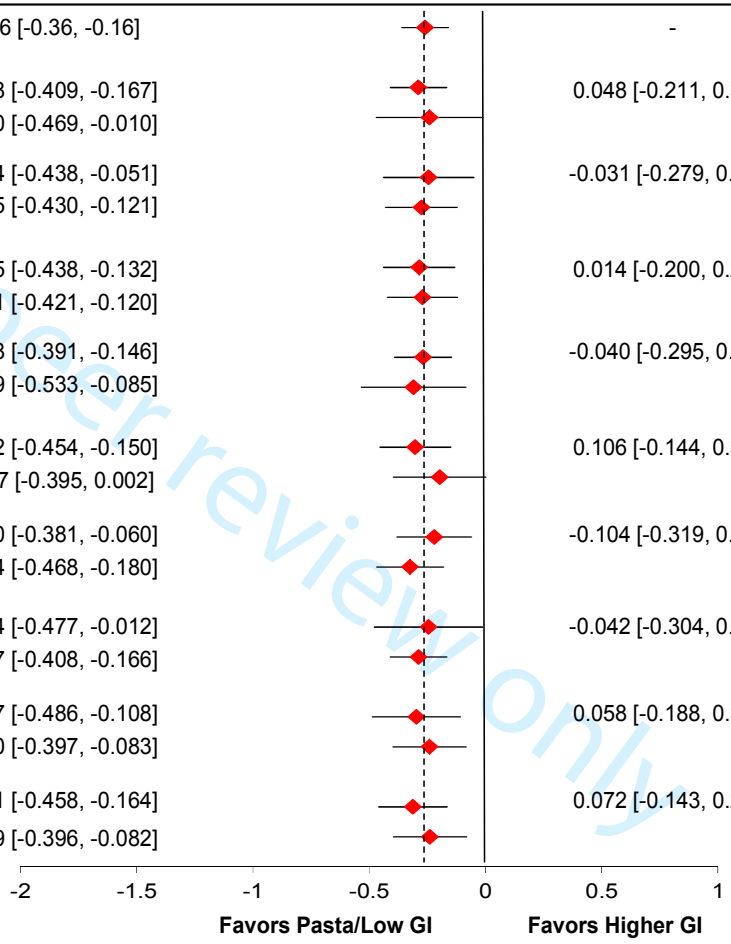
The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on BMI. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study

heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for dose (n=3 trials), therefore analyses were not performed.

*Pairwise between-subgroup mean differences (95% CIs) for Metabolic Phenotype were as follows: 0.059kg/m^2 (-0.156, 0.274) (1 vs. 2) to 0.584kg/m^2 (-1.009, 2.178) (1 vs. 3) to -0.525kg/m^2 (-2.122, 1.072) (2 vs. 3).
┼ data available on 17 studies
ǂ data available on 16 studies
‖ data available on 10 studies

BMI, body mass index; btw, between; CI, confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; trt, treatment.

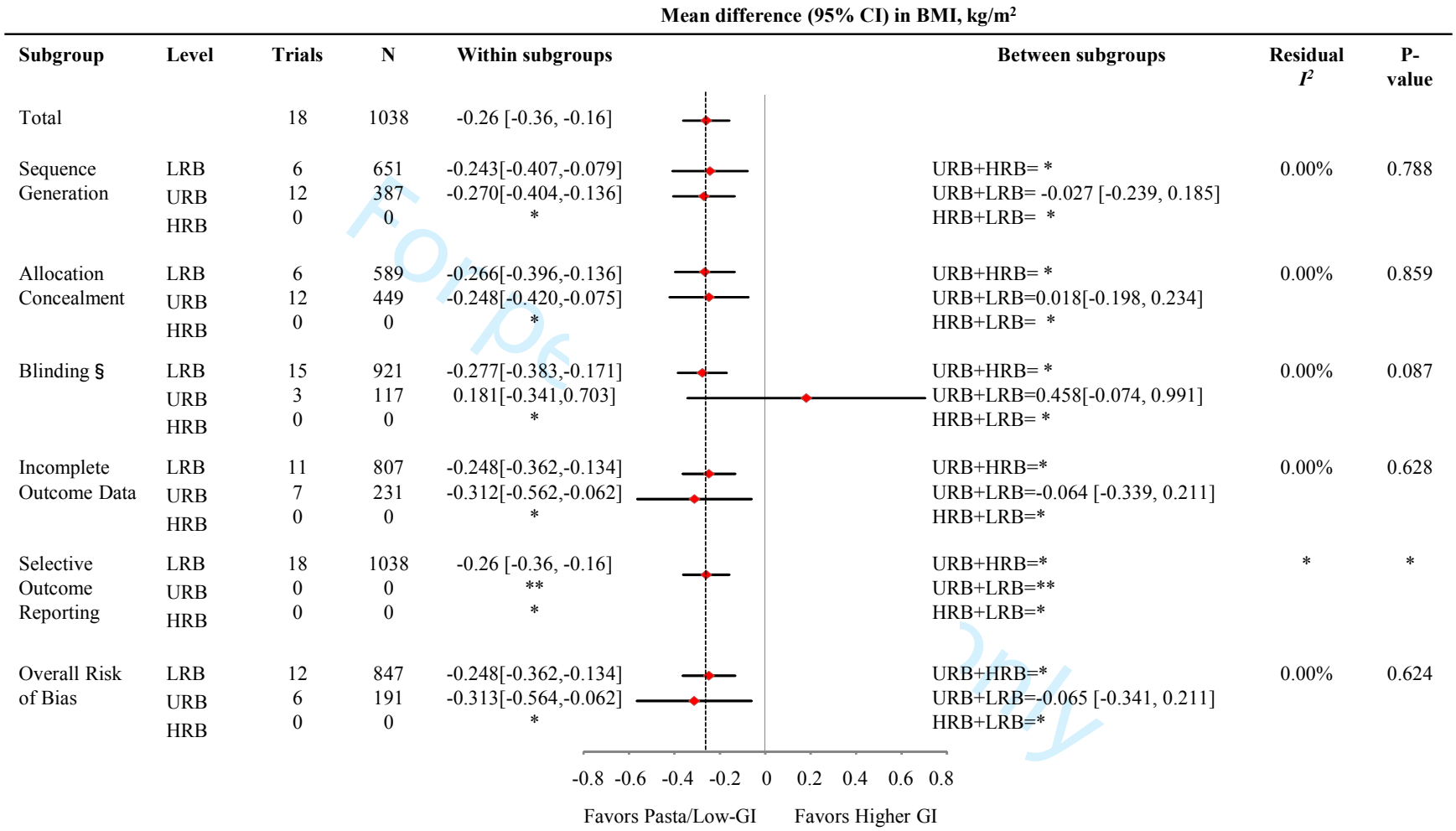
Subgroup	Level	No. comparisons	N	Mean difference [95% CI] in BMI [kg/m²]	Residual I²	P-value
				Within subgroups / Between subgroups		
Total		18	1038	-0.26 [-0.36, -0.16]	-	-
Test CHO§	< 50%	9	659	-0.288 [-0.409, -0.167]	0.00%	0.696
	≥ 50%	7	276	-0.240 [-0.469, -0.010]		
CHO within trt¥	≤ 1.35%	5	436	-0.244 [-0.438, -0.051]	0.00%	0.781
	> 1.35%	5	322	-0.275 [-0.430, -0.121]		
CHO btw trt§	≤ -1.75%	8	657	-0.285 [-0.438, -0.132]	0.00%	0.888
	> -1.75%	8	278	-0.271 [-0.421, -0.120]		
Test Protein§	< 20%	13	590	-0.268 [-0.391, -0.146]	0.00%	0.741
	≥ 20%	3	345	-0.309 [-0.533, -0.085]		
Protein within trt¥	≤ 1.04%	5	339	-0.302 [-0.454, -0.150]	0.00%	0.358
	> 1.04%	5	419	-0.197 [-0.395, 0.002]		
Protein btw trt§	≤ 0.45%	8	373	-0.220 [-0.381, -0.060]	0.00%	0.319
	> 0.45%	8	562	-0.324 [-0.468, -0.180]		
Test Fat§	< 30%	6	176	-0.244 [-0.477, -0.012]	0.00%	0.734
	≥ 30%	10	759	-0.287 [-0.408, -0.166]		
Fat within trt¥	≤ -2.60%	5	303	-0.297 [-0.486, -0.108]	0.00%	0.604
	> -2.60%	5	455	-0.240 [-0.397, -0.083]		
Fat btw trt§	≤ 0.95%	8	253	-0.311 [-0.458, -0.164]	0.00%	0.483
	> 0.95%	8	682	-0.239 [-0.396, -0.082]		



Supplemental Figure S14: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body mass index, BMI (kg/m^2) continued (n = 1038)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on BMI. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for test saturated fat (n=7 trials), saturated fat within treatment (n=6 trials) and saturated fat between treatments (n=7 trials), therefore analyses were not performed.
§ data available on 16 studies
¥ data available on 10 studies

BMI, body mass index; btw, between; CHO, carbohydrate; CI = confidence interval; GI, glycemic index (glucose scale); trt, treatment.



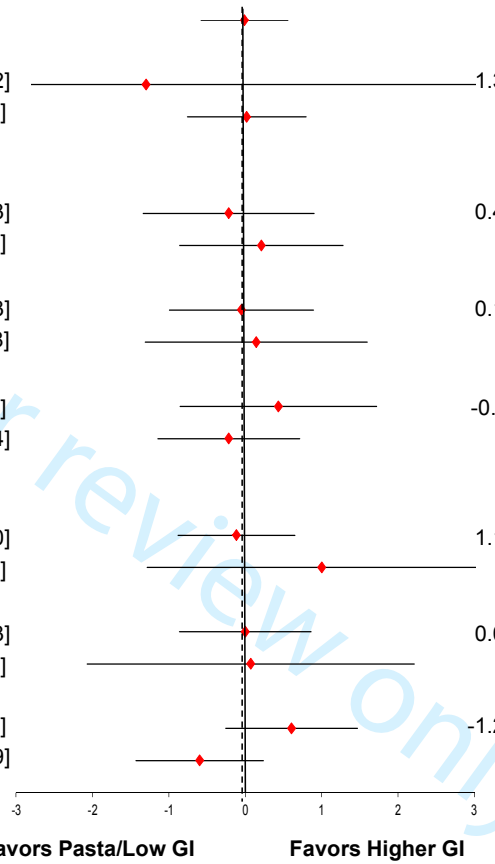
Supplemental Figure S15: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on body mass index, BMI (kg/m^2) (n = 1038)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on BMI. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

*Within and between subgroup analyses could not be performed against HRB since no values for HRB subgroup.
** no values for URB subgroup.
§ Blinding of Participants, Personnel, and Outcome Assessors.

BMI, body mass index; CI, confidence interval; HRB, High Risk of Bias; GI, glycemic index; LRB, Low Risk of Bias; URB, Unclear Risk of Bias.

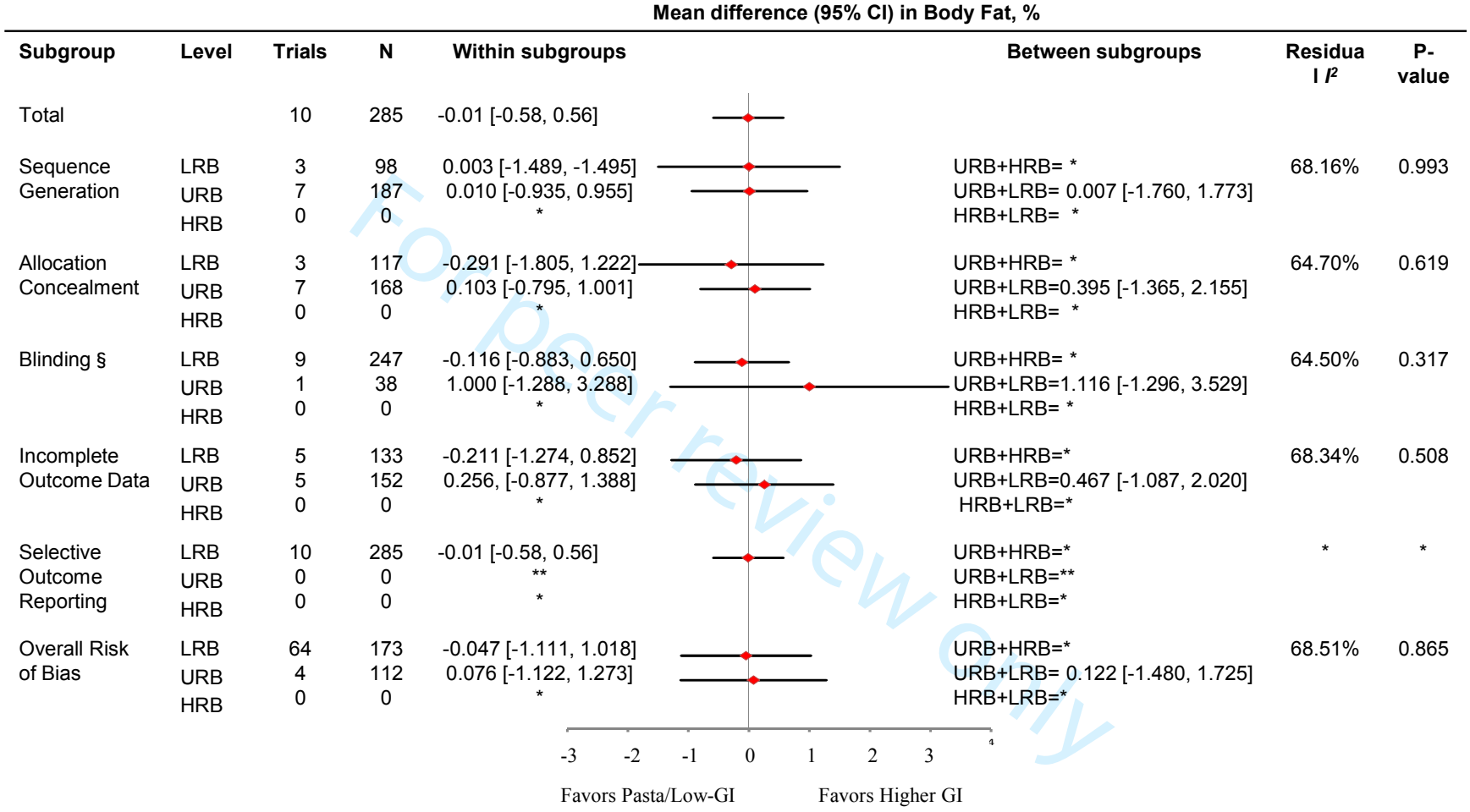
Subgroup	Level	No. comparisons	N	Mean difference [95% CI] in body fat [%]		Residual I^2	P-value
				Within subgroups	Between subgroups		
Total		10	285	-0.01 [-0.58, 0.56]	-	-	-
Metabolic Phenotype	OW/OB	9	265	-1.300 [-7.782, 5.182]	1.319 [-5.210, 7.847]	68.17%	0.654
	Diabetes	1	20	0.019 [-0.760, 0.797]			
	CHD	0	0				
Baseline BMI	≤30kg/m^2	4	112	-0.217 [-1.338, 0.903]	0.427 [-1.125, 1.978]	67.72%	0.544
	>30kg/m^2	6	173	0.210 [-0.863, 1.282]			
Design	Parallel	7	229	-0.051 [-0.996, 0.893]	0.194 [-1.540, 1.929]	66.40%	0.803
	Crossover	3	56	0.143 [-1.312, 1.598]			
Energy Balance	Negative	4	130	0.434 [-0.856, 1.723]	-0.650 [-2.241, 0.940]	68.11%	0.373
	Neutral	6	155	-0.216 [-1.147, 0.714]			
	Positive						
Follow-up	<24-weeks	9	247	-0.116 [-0.883, 0.650]	1.116 [-1.300, 3.529]	64.50%	0.317
	≥24-weeks	1	38	1.000 [-1.288, 3.288]			
Test GI	≤55	9	266	-0.000 [-0.864, 0.863]	0.070 [-2.241, 2.382]	68.43%	0.946
	>55	1	19	0.070 [-2.074, 2.214]			
GI btw trt	≤14.1	5	152	0.606 [-0.258, 1.471]	-1.204 [-2.407, -0.008]	55.49%	0.050
	>14.1	5	153	-0.597 [-1.434, 0.239]			



Supplemental Figure S16: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body fat (%) (n = 285)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body fat. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for dose (n=3), test carbohydrate, protein and fat (n=9), carbohydrate, protein and fat within treatment (n=4), carbohydrate, protein and fat between treatment (n=9), test fibre (n=8), fibre within treatment (n=4), fibre between treatment (n=8), test saturated fat (n=3 trials), saturated fat within treatment (n=2 trials) and saturated fat between treatments (n=3 trials), therefore analyses were not performed.

BMI, body mass index; btw, between; CHO, carbohydrate; CI = confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; trt, treatment.

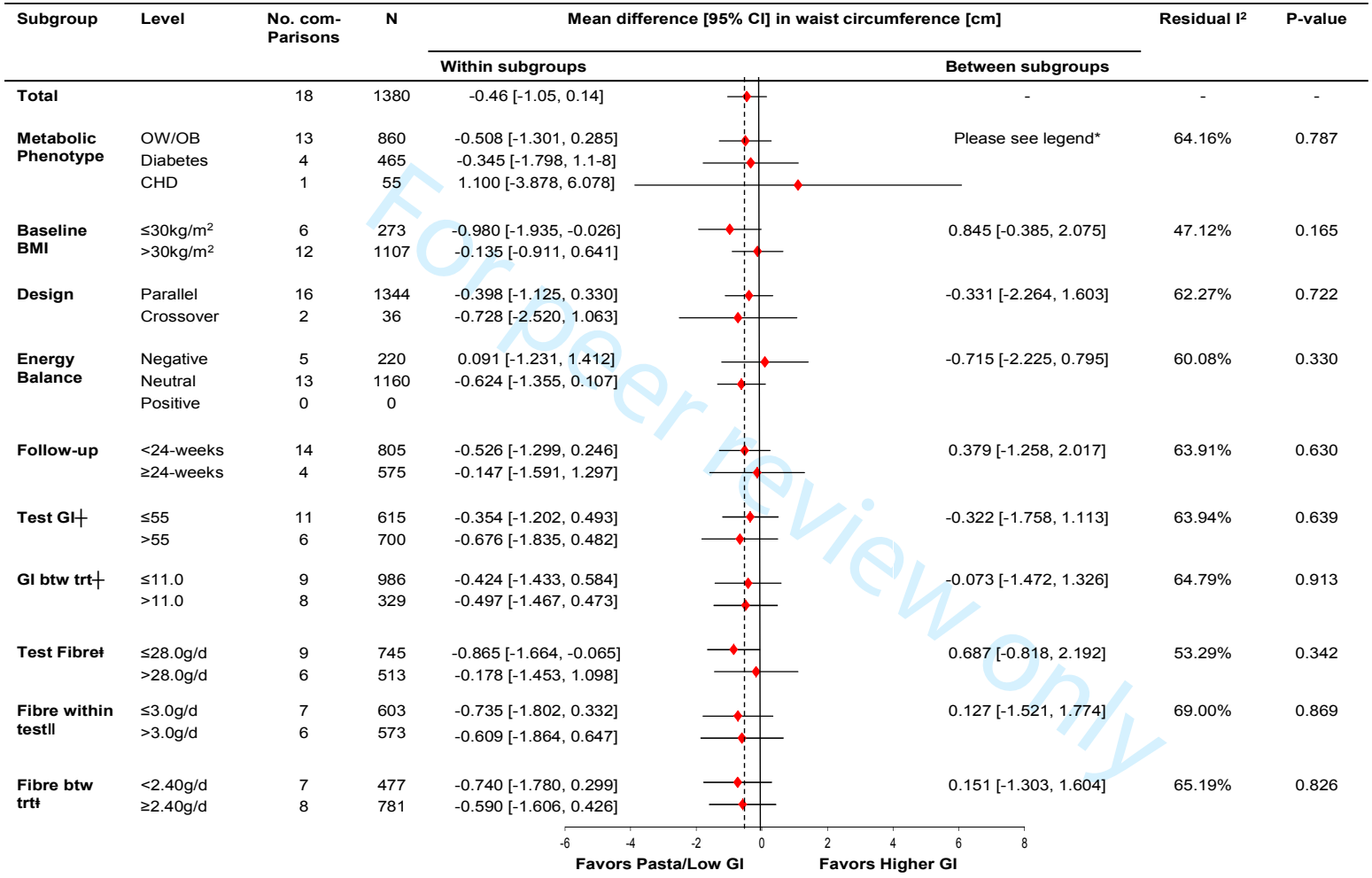


Supplemental Figure S17: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on body fat (%) (n = 285)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body fat. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

*Within and between subgroup analyses could not be performed against HRB since no values for HRB subgroup.
** no values for URB subgroup.
§ Blinding of Participants, Personnel, and Outcome Assessors.

CI, confidence interval; HRB, High Risk of Bias; GI, glycemic index; LRB, Low Risk of Bias; URB, Unclear Risk of Bias

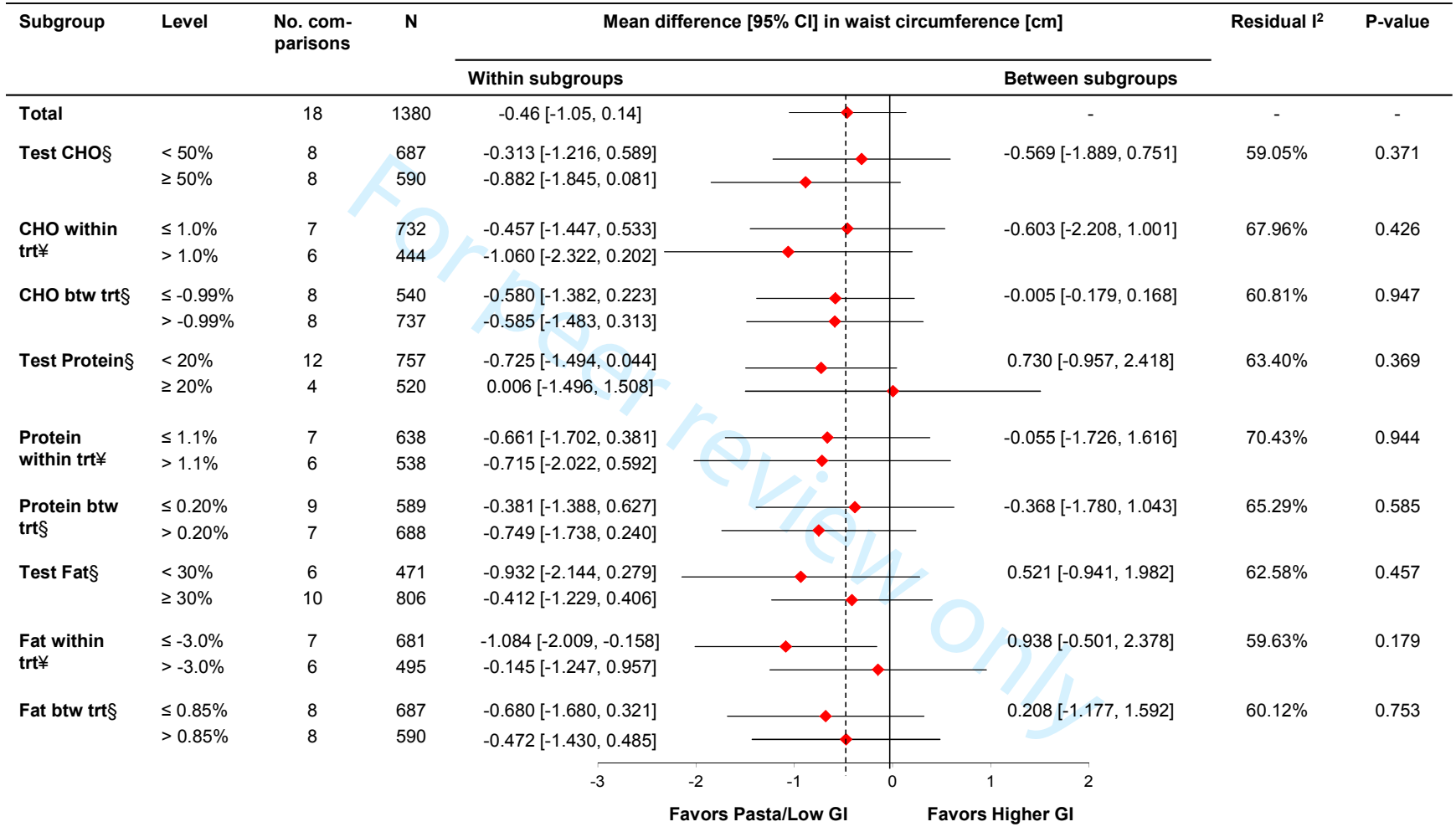


Supplemental Figure S18: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on waist circumference (cm) (n = 1380)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for dose (n=2 trials), therefore analyses were not performed.

*Pairwise between-subgroup mean differences (95% CIs) for Metabolic Phenotype were as follows: 0.163cm (-1.492, 1.818) (1 vs. 2) to 1.608cm (-3.432, 6.648) (1 vs. 3) to -1.445cm (-6.630, 3.740) (2 vs. 3).
┼ data available on 17 studies
ł data available on 15 studies

BMI, body mass index; btw, between; CI = confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; trt, treatment.

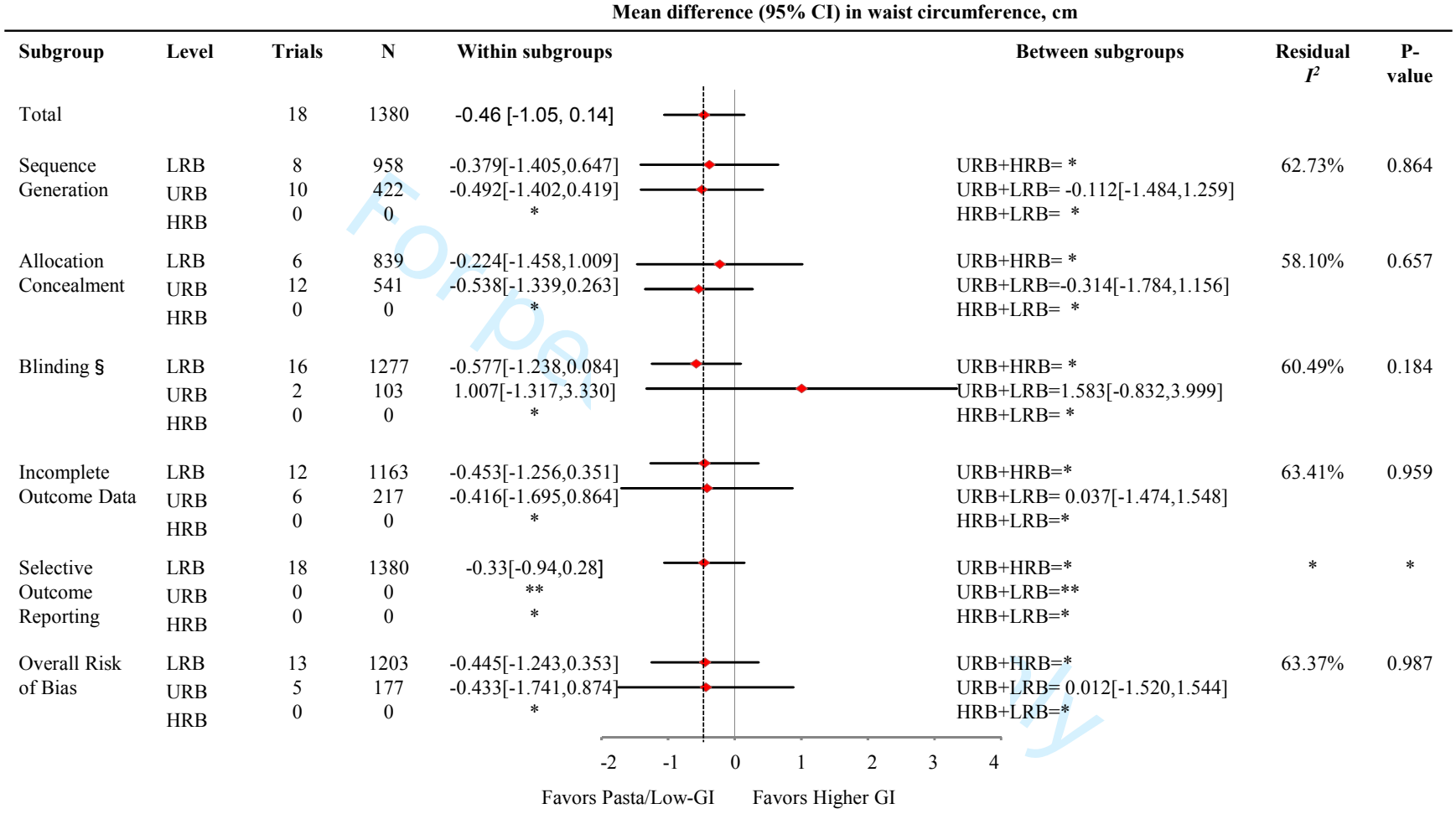


Supplemental Figure S19: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on waist circumference (cm) continued (n = 1380)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for test saturated fat (n=8 trials), saturated fat within treatment (n=7 trials) and saturated fat between treatments (n=8 trials), therefore analyses were not performed.

§ data available on 16 studies
¥ data available on 13 studies

BMI, body mass index; btw, between; CHO, carbohydrate; CI = confidence interval; GI, glycemic index (glucose scale); trt, treatment.



Supplemental Figure S20: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on waist circumference (cm) (n = 1380)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

*Within and between subgroup analyses could not be performed against HRB since no values for HRB subgroup.
** no values for URB subgroup.
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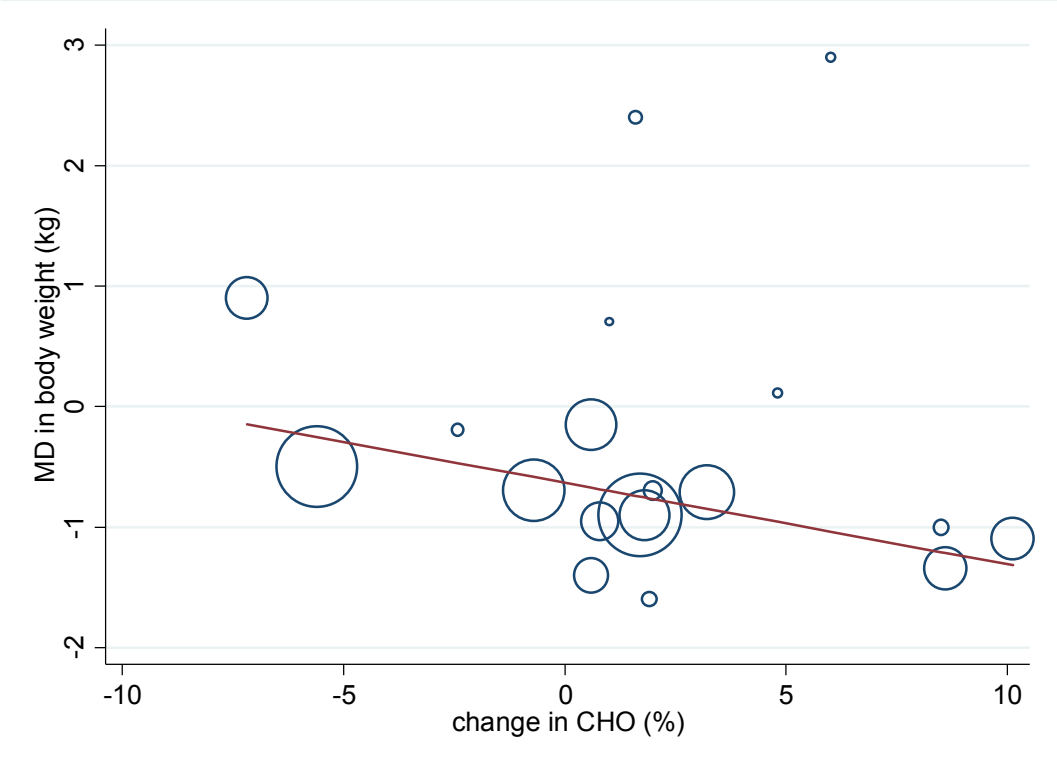
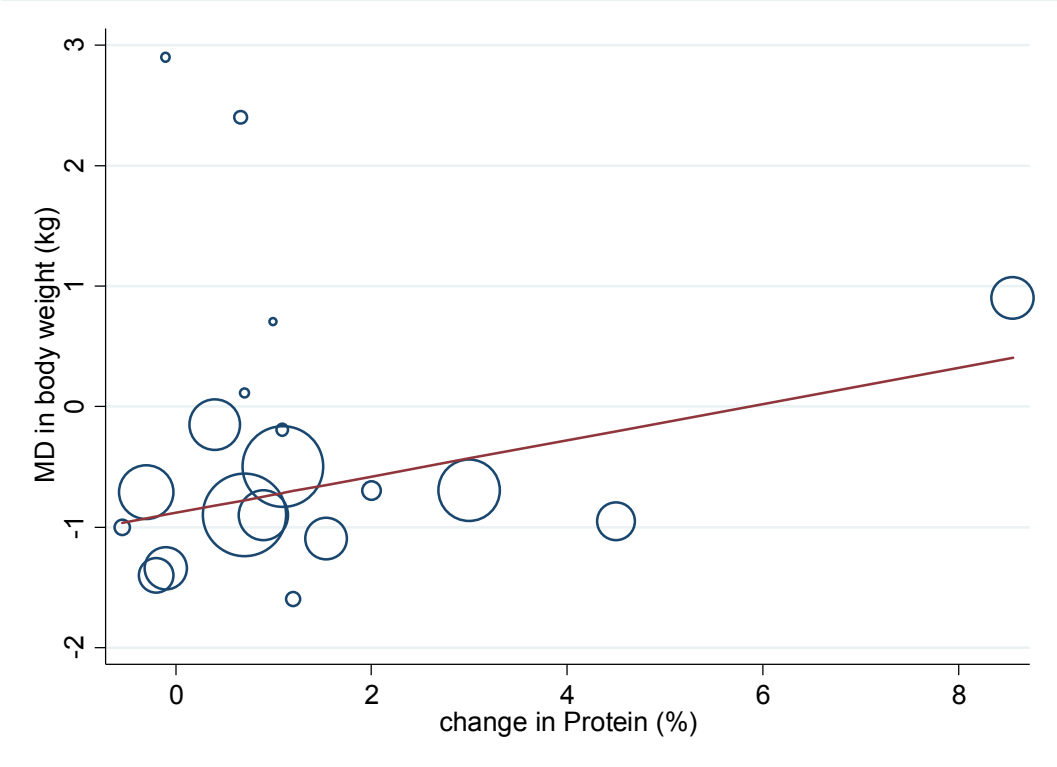
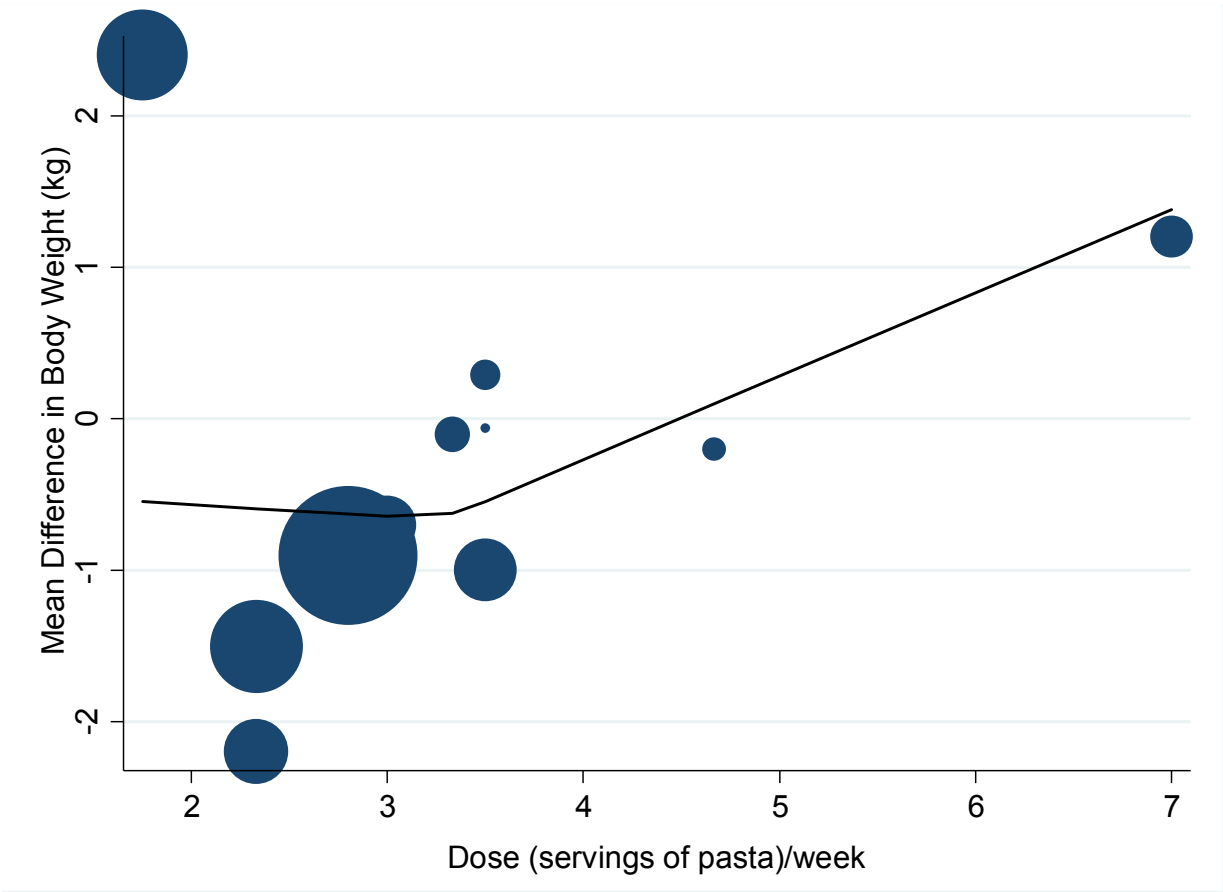
CI, confidence interval; HRB, High Risk of Bias; GI, glycemic index; LRB, Low Risk of Bias; URB, Unclear Risk of Bias



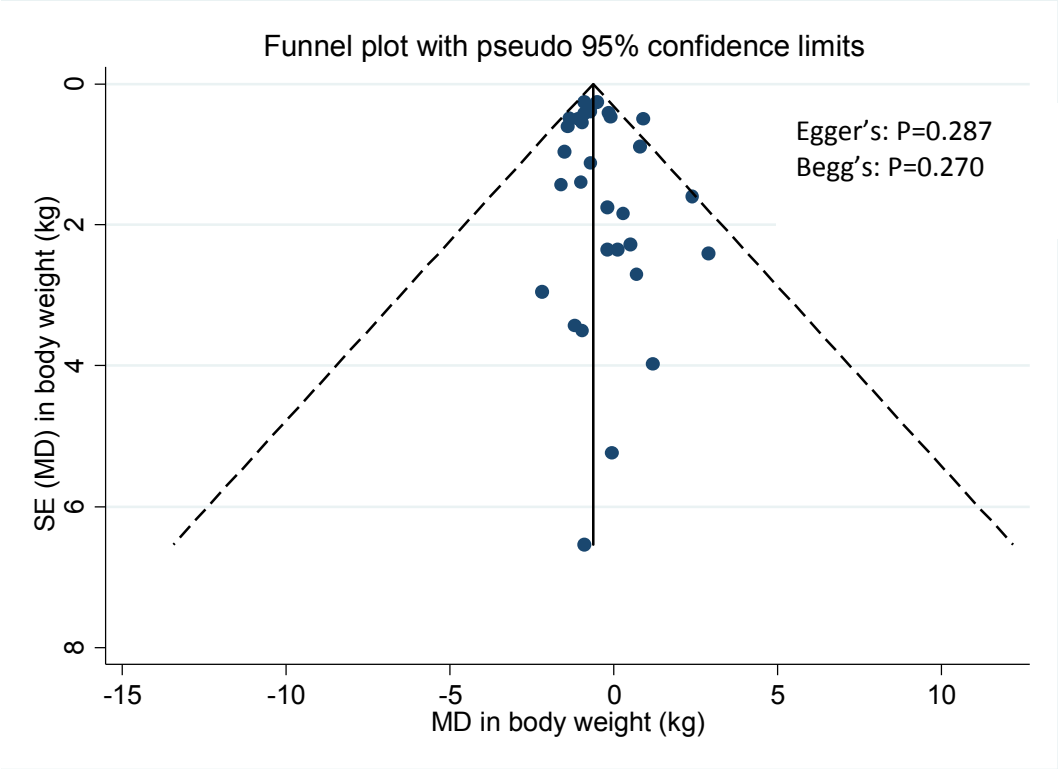
Supplemental Figure S21: Continuous meta-regression of change in carbohydrate intake in the low-GI dietary pattern intervention arms with change in body weight (n=19)
CHO, carbohydrate; MD, mean difference



Supplemental Figure S22: Continuous meta-regression of change in protein intake in the low-GI dietary pattern intervention arms with change in body weight (n=19)
MD, mean difference

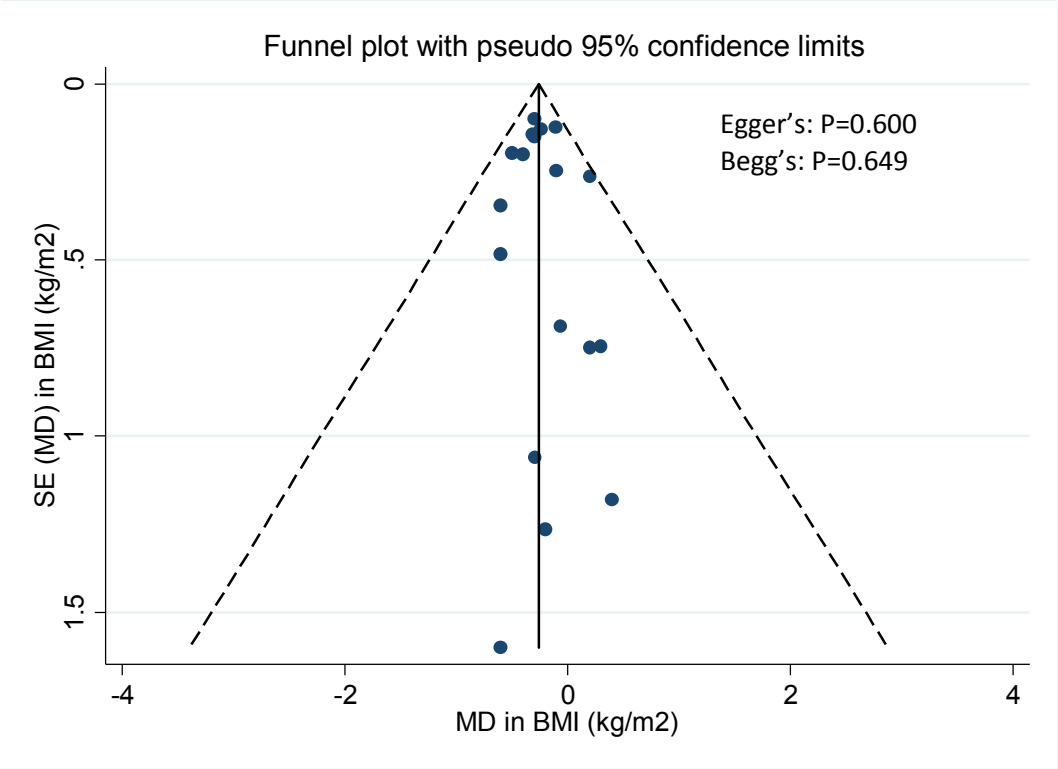


Supplemental Figure S23: Pasta intake dose-response analyses by spline curve modeling (MKSPLINE procedure)



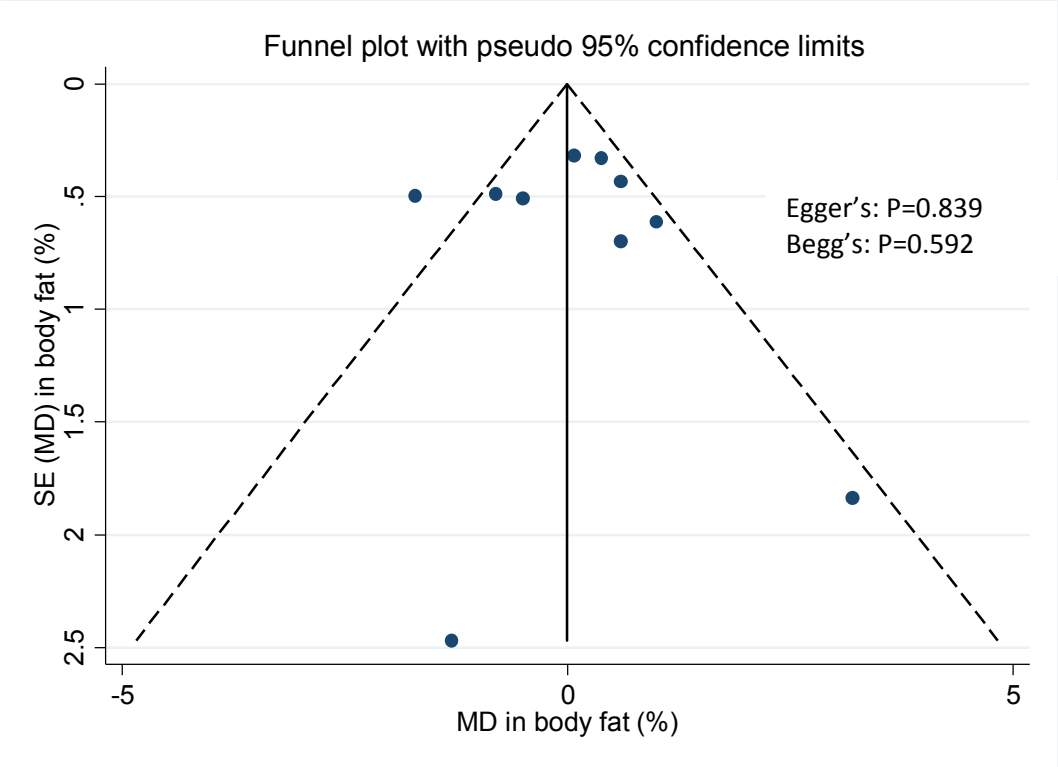
Supplemental Figure S24: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on body weight (kg)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.



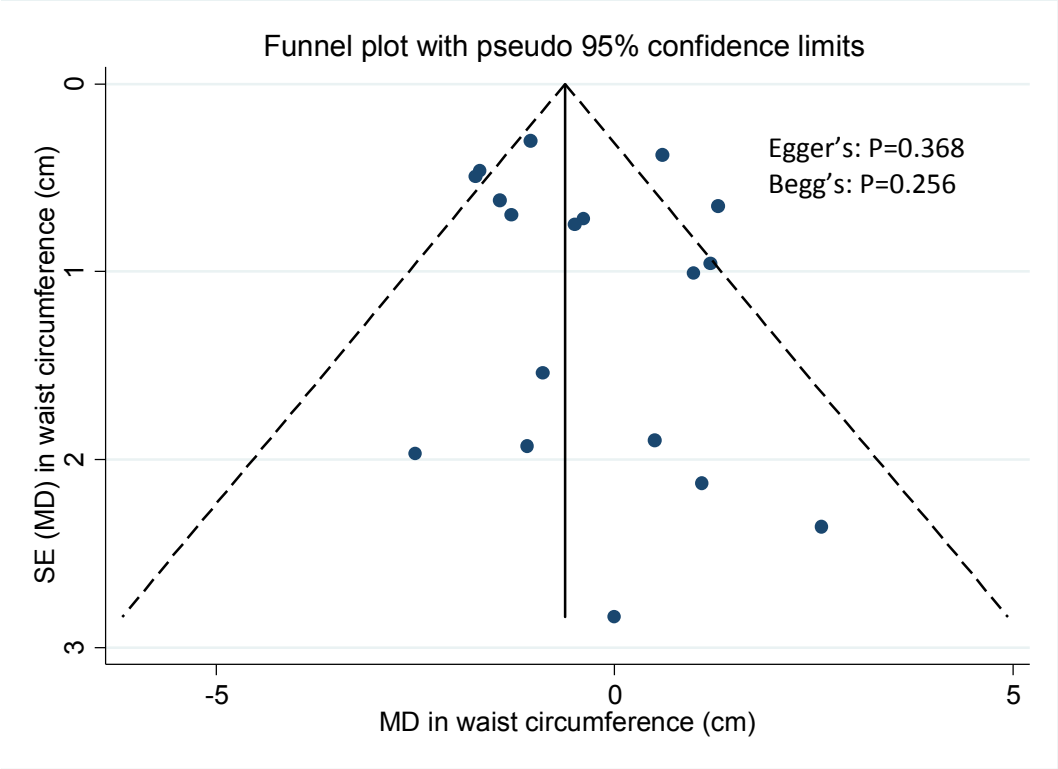
Supplemental Figure S25: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on BMI (kg/m^2)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.



Supplemental Figure S26: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on body fat (%)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.



Supplemental Figure S27: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on waist circumference (cm)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.

Supplemental Table S12: PRISMA Checklist

PRISMA 2009 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.	2-3
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	4
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	4
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 Table S2
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, Supplemental Table S1
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	Supplemental Table S1
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, Figure 1
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
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and	6, Supplemental

		simplifications made.	Table S2
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
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	7
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.	7-9

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).	9
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	7-8
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.	9-11, 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.	10-11, Table 1, Supplemental Tables S2-S3
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).	11, 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.	11-12, Figures 2-3, Supplemental Figures S3-S7
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	11-12, Figures 2-3, Supplemental Figures S3-S7
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	11, Supplemental

			Table S1, S2, S12, S15, S17, S20
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	12-15, Supplemental Tables S4-S10, Supplemental Figures S8-S26
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).	16-19
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).	19-21
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	21-22
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.	25-26

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**.

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Effect of Pasta in the Context of Low Glycemic Index Dietary Patterns on Body Weight and Markers of Adiposity: A Systematic Review and Meta-analysis of Randomized Controlled Trials in Adults

Journal:	*BMJ Open*
Manuscript ID	bmjopen-2017-019438.R1
Article Type:	Research
Date Submitted by the Author:	08-Dec-2017
Complete List of Authors:	Chiavaroli, Laura; St. Michael's Hospital, Toronto 3D Knowledge Synthesis and Clinical Trials Unit; University of Toronto, Nutritional Sciences Kendall, Cyril; University of Toronto, Nutritional Sciences Braunstein, Catherine; University of Toronto, Nutritional Sciences; St. Michael's Hospital, Toronto 3D Knowledge Synthesis and Clinical Trials Unit Blanco Mejia, Sonia; Clinical Nutrition and Risk Factor Modification Centre, St. Michael's Hospital; Toronto 3D Knowledge Synthesis and Clinical Trials Unit, St. Michael's Hospital Leiter, Lawrence; St. Michael's Hospital, Endocrinology; University of Toronto, Nutritional Sciences Jenkins, David; St. Michael's Hospital, Clinical Nutrition and Risk Factor Modification Center; University of Toronto, Nutritional Sciences Sievenpiper, John; McMaster University, Department of Pathology and Molecular Medicine; St. Michael's Hospital, Clinical Nutrition and Risk Factor Modification Centre
Primary Subject Heading:	Nutrition and metabolism
Secondary Subject Heading:	Cardiovascular medicine
Keywords:	body weight, pasta, glycemic index, glycaemic index, systematic review and meta-analysis, weight loss

Effect of Pasta in the Context of Low Glycemic Index Dietary Patterns on Body Weight and Markers of Adiposity: A Systematic Review and Meta-analysis of Randomized Controlled Trials in Adults

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Word Count: 4,968

Tables: 1

Figures: 3

Online only supplementary material included

Keywords: body weight, pasta, glycemic index, glycaemic index, systematic review and meta-analysis, weight loss

ABSTRACT

Objective: Carbohydrates have been implicated in the obesity epidemic. To assess the effect of pasta alone or pasta in the context of low glycemic index (GI) dietary patterns on adiposity, we conducted a systematic review and meta-analysis.

Design: Systematic review and meta-analysis of randomized controlled trials (RCTs) with GRADE assessment.

Eligibility criteria for selecting studies: MEDLINE, Embase, CINAHL, and the Cochrane Library were searched through 07 February 2017. We included RCTs ≥3-weeks assessing the effect of pasta alone or in the context of low-GI dietary patterns on measures of global (body weight, BMI, body fat) and regional (waist circumference [WC], waist-to-hip ratio [WHR], sagittal abdominal diameter [SAD]) adiposity in adults. Two independent reviewers extracted data and assessed risk of bias. Data were pooled using the generic inverse-variance method and expressed as mean differences (MDs) with 95% confidence intervals (95% CIs). Heterogeneity was assessed (Cochran Q statistic) and quantified (I^2-statistic). GRADE assessed the overall certainty of the evidence.

Results: We identified no trials of the effect of pasta alone and 32 trial comparisons (n=2,448 participants) of the effect of pasta in the context of low-GI dietary patterns. Pasta in the context of low-GI dietary patterns significantly reduced body weight (MD=-0.63kg [95% CIs, -0.84, -0.42kg]) and BMI (MD=-0.26kg/m^2 [95% CIs, -0.36, -0.16kg/m^2]) compared with higher-GI dietary patterns. There was no effect on other measures of adiposity. The overall certainty of the

evidence was graded as moderate for body weight, BMI, WHR, and SAD and low for WC and body fat.

Conclusions: Available RCTs demonstrate that pasta in the context of low-GI dietary patterns does not adversely affect adiposity and even reduces body weight and BMI compared to higher GI dietary patterns. Future trials should assess the effect of pasta in the context of other healthy dietary patterns**.**

Protocol registration: ClinicalTrials.gov Identifier, NCT02961088

Strengths and limitations of this study

- The present systematic review and meta-analysis was based on a comprehensive search and includes a large number of randomized controlled trials which provide the best protection against bias.
- We used the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) system to evaluate the strength and quality of the evidence.
- There was evidence of unexplained inconsistency in the intervention estimates across trials for waist circumference and body fat.
- The generalizability of our results is questionable with evidence of indirectness in the pooled estimates for all body and adiposity outcomes, as all of the trials assessed pasta in the context of low-GI dietary patterns (no trials assessed the effect of pasta alone or in the

context of other dietary patterns) and most of the available trials did not quantify the amount of pasta consumed.

INTRODUCTION

As the role of saturated fat in chronic disease has been called into question, carbohydrates have come under attack in the media[1,2], popular books[3-9], statements of health advocacy groups[10] and commentaries in leading medical journals[11,12]. Much of the attention has focused on sugars but traditional carbohydrate staples like pasta, rice, and breads are increasingly being implicated in the epidemics of overweight and obesity[1,7]. Although advantages for weight related outcomes have been shown for dietary patterns that are high in these foods but low in glycemic index (GI)[13,14], high in whole grains[15,16], and/or high in dietary fibre[17,18], there has been a general lack of recognition of the importance of carbohydrate quality.

Pasta is an important example of a food which is considered a refined carbohydrate but has a low-GI, a property that has been exploited extensively in studies of low-GI dietary patterns. It remains unclear whether pasta alone or in the context of a low-GI dietary pattern contributes to weight gain[1,7]. We undertook a systematic review and meta-analysis of randomized controlled trials (RCTs) with a full GRADE assessment to quantify the effect of pasta alone or in the context of low-GI dietary patterns on body weight and measures of adiposity relevant to the prevention and management of overweight and obesity.

METHODS

Design

Our protocol followed the guideline of the Cochrane Handbook for Systematic Reviews of Interventions[19], and findings are reported according to the guidelines of the Preferred Reporting

Items for Systematic Reviews and Meta-Analyses[20] (**Supplemental Table S1**). The protocol is registered at clinicaltrials.gov (identifier, NCT02961088).

Data sources and searches

We searched MEDLINE (http://www.nlm.nih.gov/bsd/pmresources.html), Embase (https://www.embase.com), CINAHL (https://health.ebsco.com/products/the-cinahl-database), and the Cochrane Library (http://www.cochranelibrary.com/) from inception through 07 February 2017. The full search terms used in this study are presented in **Supplemental Tables S2-S3**. Briefly, we searched using variations of the terms pasta and glycemic index and glycemic load and body weight and BMI. The search was limited to human studies and had no language restrictions. Reference lists of selected studies and reviews were also searched to identify additional articles.

Study selection

We include RCTs that investigated the effect of pasta consumed alone or in the context of low-GI dietary patterns that emphasized pasta in comparison with higher GI diets that did not include pasta on body weight or other measures of global (BMI, body fat) or abdominal (waist circumference, waist-to-hip ratio, sagittal abdominal diameter, or visceral adipose tissue assessed by imaging modalities) adiposity in participants of all health backgrounds. Trials were included if the intervention arm assessed the effect of pasta consumed alone or assessed the effect of a low GI diet which emphasized pasta as part of the low GI dietary advice. Trials were excluded if they had diet duration of <3 weeks, did not intend to use a calorie- and macronutrient-matched comparator arm that was higher in GI; included pregnant or breast-feeding women or children; or

did not provide suitable end-point data. When multiple publications existed for the same study, the article with the most information was included (n=6). Published abstracts were not included.

Data extraction

Two reviewers (LC, CB) assessed the titles and abstracts of all identified studies and independently reviewed and extracted relevant data from each report, including study design, blinding, sample size, participant characteristics, follow-up duration, identification of pasta in the low-GI diet only, comparator diet, macronutrient profile, funding source, and outcome data. The primary outcome was body weight, and secondary outcomes included markers of global (BMI, body fat) and abdominal (waist circumference, waist-to-hip ratio, sagittal abdominal diameter, or visceral adipose tissue assessed by imaging modalities) adiposity. Mean±SD differences between test and control arms were extracted for each outcome.

In those trials where the data were included in figures and not provided numerically, we used the software program Plot Digitizer (http://plotdigitizer.sourceforge.net/) to extract the data. Additional information was requested from the authors of all included trials. Disagreement were resolved by consensus or where necessary by a third author (SBM).

Risk of bias assessment

Risk of bias for each individual study was assessed using the Cochrane Risk of Bias tool[19]. The level of bias was evaluated for sequence generation, allocation concealment, blinding, incomplete outcome data, and selective reporting and determined overall as either low (proper methods taken to reduce bias), high (improper methods creating bias), or unclear (insufficient information provided to determine the bias level).

Statistical analysis

Baseline, end-of-study, and between-treatment differences in body weight, BMI, waist circumference, body fat, waist-to-hip ratio, sagittal abdominal diameter or visceral adipose tissue assessed by imaging modalities were recorded as means±SDs. If not provided, between-treatment differences in change-from-baseline or end differences were calculated by subtracting means and variance measures such as SEs were imputed with the use of published formulas[19]. Missing SDs were imputed with the use of the pooled SD from other studies included in the analysis[19].

Data analyses were conducted using Review Manager version 5.3 (RevMan) (Copenhagen, Denmark: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014) for primary analyses and Stata version 13 (College Station, TX: StataCorp LP) for subgroup analyses. A generic inverse-variance method with random-effects models was used to calculate pooled mean differences and 95% confidence intervals (CIs). Random-effects models were used even in the absence of statistically significant inter-study heterogeneity, as they yield more conservative summary effect estimates in the presence of residual heterogeneity. Change-from-baseline differences were preferred over end differences and paired analyses were applied to all crossover trials with the use of a within-individual correlation coefficient between treatments of 0.5 as described by Elbourne et al.[21]

Inter-study heterogeneity was assessed by the Cochran Q statistic, where $P<0.10$ was considered statistically significant, and quantified by the I^2 statistic, where $I^2 \geq 50\%$ indicates substantial heterogeneity[19]. Sensitivity analysis were performed which included the removal of each single study from the meta-analyses one at a time and recalculation of the summary effect. An

influential study was considered a study whose removal changed the magnitude of the pooled effect by >10%. Sensitivity analysis were also conducted using different correlation coefficient values for crossover trials (0.25 and 0.75) to test for the robustness of the effect size, conducting analyses using fixed effects models and restricting analyses to those trials for which pasta intake could be quantified.

If ≥10 trial comparisons were available, then sources of heterogeneity were explored by subgroup analyses. A priori categorical subgroups analyses were assessed by meta-regression analyses. These included patient type (normal body weight, overweight or obese [average baseline BMI >27kg/m^2]), diabetes, coronary heart disease), follow-up (<24-weeks, ≥24 weeks), baseline BMI (BMI ≤30, >30kg/m^2), design (parallel, crossover), energy balance (negative on both arms [weight loss diets], neutral on both arms [weight maintaining diets]) and dose of pasta (based on the median). A priori categorical subgroup analyses also included the following dietary factors: GI (absolute level [≤55, >55; glucose scale], within-treatment change, between-treatment change), fat intake (absolute level [<30%, ≥30% energy], within-treatment change, between-treatment change), carbohydrate intake (absolute level [<50%, ≥50% energy], within-treatment change, between-treatment change), protein intake (absolute level [<20%, ≥20% energy], within-treatment change, between-treatment change), dietary fibre intake (absolute level [<28g/day, ≥28g/day], within-treatment change, between-treatment change), and risk of bias. A priori continuous meta-regression analyses were conducted on the absolute levels and within- and between-treatment changes of these same dietary factors in the intervention arms of pasta in the context of low-GI dietary patterns. Linear and non-linear pasta intake dose-response analyses were assessed by using continuous meta-regression analyses and spline curve modeling (MKSPLINE procedure), respectively. Publication bias was assessed by visual inspection of

funnel plots and the Egger[22] and Begg[23] tests, when ≥10 trial comparisons were available. If publication bias was suspected, then adjustment for funnel plot asymmetry was done by imputing missing study data using the Duval and Tweedie trim and fill method [24].

Grading the evidence

The grading of recommendations assessment, development, and evaluation (GRADE) approach was used to assess the certainty of the evidence [25]. Evidence was graded as high, moderate, low or very low quality. The included RCTs were graded as high quality evidence by default and downgraded based on pre-specified criteria. Criteria to downgrade evidence included risk of bias (weight of studies show risk of bias assessed by the Cochrane Risk of Bias tool), inconsistency (substantial unexplained heterogeneity, I^2>50%, P<0.10), indirectness (presence of factors that limited the generalizability of the results), imprecision (the 95% CI for effect estimates were wide or crossed pre-specified minimally important differences [MIDs] for harm), and publication bias (significant evidence of small-study effects).

Patient involvement

No patients were directly involved in the development of the research question, selection of the outcome measures, design and implementation of the study, or interpretation of the results.

RESULTS

Search results

Figure 1 shows the flow of the literature. We identified 4876 reports of which 29 met eligibility criteria. No reports were identified that assessed the effect of pasta alone, while 29 reports (including 32 trial comparisons involving 2448 participants) were identified that assessed the effect of pasta in the context of low-GI dietary patterns on any adiposity outcome in adults[26-54]. Of the 32 trial comparisons that assessed the effect of pasta in the context of a low-GI dietary pattern, there were 32 trial comparisons for body weight, 18 trial comparisons for BMI[27,28,31-33,35,36,39-41,43-46,48,49,52,53], 18 trial comparisons for waist circumference[27,28,31,33,34,36,38-40,42,44-47,52,53], 10 trial comparisons for body fat[27,28,31-33,36,38,41,43,53], 6 trial comparisons for waist-to-hip ratio[31,39,40,44,45,52], and 3 trial comparisons for sagittal abdominal diameter[34,39]. There was only one trial comparison identified[35] for visceral adipose tissue assessed by imaging modalities, thus a meta-analysis could not be undertaken for this outcome.

Trial characteristics

Table 1 and **Supplemental Table S4** show the characteristics of all included trials of the effect of pasta in the context of low-GI dietary patterns. The majority of trials had a parallel design (26/32) with a median follow-up of 12 weeks (IQR: 9–21 weeks) and a median number of participants per trial of 43 (IQR: 21–112). Most participants were middle aged (median age: 50 y; IQR: 40-58 y) men and women (median ratio of men to women: 0.4:1 in available trials). The median baseline BMI across studies was 30.4kg/m^2 (IQR: 28.2–32.0). Regarding metabolic phenotype, 21 (66%) trials included participants who were overweight or obese (had a baseline BMI≥27kg/m^2), 10 (31%) had diabetes and one trial (3%) with coronary heart disease (CHD). We did not retrieve any trials where participants had a normal BMI at baseline (≤25kg/m^2),

although 6 trials did not include BMI >25 kg/m^2 as part of criteria, the average baseline BMI was ≥27 kg/m^2, therefore categorized as overweight.

Risk of bias

Supplemental Figures S1 and S2 show the individual Cochrane Risk of Bias tool assessments for each of the included trials of the effect of pasta in the context of low-GI dietary patterns. No serious risk of bias was detected.

Effect of Pasta in the Context of Low-GI dietary patterns and Body Weight

Figure 2 shows the effect of pasta in the context of low-GI dietary patterns on the primary outcome body weight. Pooled analyses showed pasta in the context of low-GI dietary patterns had the effect of reducing body weight by -0.63 kg (95% CI:-0.84, -0.42 kg; P<0.001) compared with higher GI control diets with no evidence of heterogeneity (I^2=0%, P-heterogeneity=0.51).

Effect of Pasta in the Context of Low-GI dietary patterns on Markers of Global Adiposity

Figure 3 and **Supplemental Figure S3-S4** show the effect of pasta in the context of low-GI dietary patterns on markers of global adiposity. Pooled analyses showed that pasta in the context of low-GI dietary patterns had the effect of reducing BMI by -0.26kg/m^2 (n=18 trials; MD=-0.26kg/m^2; 95% CI:-0.36, -0.16 kg/m^2; P<0.001) compared with higher GI control diets with no evidence of heterogeneity (I^2=0%, P-heterogeneity=0.90). There was no effect on body fat (n=10 trials; MD=-0.01%, 95% CI:-0.58, 0.56%; P=0.98) with evidence of substantial heterogeneity (I^2=65%, P-heterogeneity<0.01).

Effect of Pasta in the Context of Low-GI dietary patterns on Markers of Abdominal Adiposity

Figure 3 and **Supplemental Figures S5-S7** show the pooled estimates for the markers of abdominal adiposity. Pooled analyses did not show a significant effect of pasta in the context of low-GI dietary patterns compared with higher-GI control diets on waist circumference (n=18 trials; MD=-0.46cm, 95% CI:-1.05, 0.14cm; P=0.13), waist-to-hip ratio (n=6 trials; MD=-0.00, 95% CI:-0.01, 0.00; P=0.27), or sagittal abdominal diameter (n=3 trials, MD=-0.09cm, 95% CI:-0.34, 0.16cm; P=0.48) There was only evidence of substantial heterogeneity in the analyses for waist circumference (I^2=62%, P-heterogeneity<0.01).

Sensitivity analyses

We conducted four sets of sensitivity analyses (**Supplemental Tables S5-6, Supplemental Figures S8-9**). The systematic removal of each trial did not modify the direction or significance of the effect estimates or the evidence of heterogeneity for any of the outcomes with the exception of waist circumference (**Supplemental Table S5**). In the sensitivity analysis for waist circumference, two studies were influential studies in that their removal altered the magnitude of the pooled effect in the remaining studies by >10%, where the removal of the studies of McMillan-Price et al. (high protein comparison)[42] and Jenkins et al.[44] rendered the results for waist circumference statistically significant (MD= -0.62cm; 95% CI: -1.19, -0.05; P=0.03) and (MD= -0.61cm; 95% CI: -1.18, -0.04; P=0.04), respectively; forest plots not shown). Heterogeneity remained significant in both cases (I^2= 55%, P-heterogeneity<0.01 and I^2=50%, P-heterogeneity=0.01, respectively). Sensitivity analyses using correlation coefficients of 0.25 and 0.75 for paired analyses of crossover trials also did not modify the results (**Supplemental Table**

S6). In the sensitivity analyses where fixed effects models were applied (**Supplemental Figure S8**), the direction, magnitude and significance of the pooled estimates were very similar to those produced by the random effects models with the exception of the sensitivity analysis for waist circumference which was significant (MD= -0.62; 95% CI: -0.93, -0.32; P<0.001). Finally, restricting analyses to the 11 trials for which pasta intake could be quantified (median pasta intake, 3.33 servings/week [range, 1.75 – 7 servings/week]) showed a similar reduction in body weight (MD= -0.70 kg; 95% CI:-1.10, -0.29 kg; P<0.001) when pasta was consumed in the context of low-GI dietary patterns compared with the higher GI control arms without evidence of heterogeneity (I^2=0%, P-heterogeneity=0.68) (**Supplemental Figure S9)**.

Subgroup analyses

We were only able to conduct a priori categorical and continuous subgroup analyses for body weight, BMI, body fat and waist circumference. Subgroup analyses for waist-to-hip ratio and sagittal abdominal diameter could not be assessed, owing to <10 trial comparisons in each case. **Supplemental Figures S10-S12** show the categorical a priori subgroup analyses for body weight. There was no evidence of significant effect modification in any of the subgroup analyses for body weight, including no effect modification of follow-up when comparing studies less than 24 weeks duration to those greater than or equal to 24 weeks (-0.63kg vs -0.57kg, respectively) (**Supplemental Figure S10**). Neither was there evidence of significant effect modification in any of the subgroup analyses for BMI, body fat or waist circumference (**Supplemental Figures S13-20**).

Supplemental Table S7 and **Supplemental Figures S21-22** show the continuous subgroup analyses for body weight. There was evidence of significant effect modification by carbohydrate and protein intake, where an increase in carbohydrate intake in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with weight loss (β= -0.07, 95% CI: -0.12, -0.01, I^2=0.00%, P=0.02), and an increase in protein intake in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with weight gain (β=0.15, 95% CI: 0.03, 0.27, I^2=0.00%, P=0.02). None of the other continuous subgroup analyses were significant. There was no evidence of significant effect modification in any of the continuous subgroup analyses for BMI (**Supplemental Table S8**). For body fat, there was evidence of significant effect modification in the continuous meta-regression subgroup analysis of difference in GI between intervention and control groups, where greater difference in GI between the groups was associated with greater reduction in body fat in the intervention group (β=-0.09, 95% CI: -0.15, -0.03, I^2=19.39%, P=0.01) (**Supplemental Table S9**). None of the other continuous subgroup analyses were significant. For waist circumference, there was evidence of significant effect modification in the continuous meta-regression subgroup analysis of absolute carbohydrate level and absolute protein level, where greater carbohydrate level in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with greater loss in waist circumference (β=-0.11, 95% CI: -0.19, -0.04, I^2=27.06%, P<0.01) and a lower protein level in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with an increase in waist circumference (β= 0.20, 95% CI: 0.01, 0.38, I^2=43.92%, P=0.04) (**Supplemental Table S10**). None of the other continuous subgroup analyses were significant.

Dose-response analyses

Supplemental Tables S7 and S11 and **Supplemental Figure S23** show the dose-response analysis for the 11 trials for which pasta intake could be quantified. No evidence of a linear dose response was seen for pasta intake by meta-regression analyses (**Supplemental Table S7**). There was also no evidence of a non-linear dose response by MKSPLINE (P=0.85) (**Supplemental Figure S23**) or piecewise linear meta-regression analyses (**Supplemental Table S7**).

Publication Bias

Supplemental Figures S24-S27 shows the funnel plots for body weight, BMI, body fat and waist circumference. There was no evidence of funnel-plot asymmetry. Formal testing with the Egger and Begg tests did not show evidence of small-study effects (P>0.05 for both). Publication bias was not assessed for waist-to-hip ratio and sagittal abdominal diameter, owing to <10 trial comparisons.

GRADE Assessment

Supplemental Table S12 shows a summary of the GRADE assessments for the effect of pasta in the context of low-GI dietary patterns on body weight and measures of adiposity**.** The evidence was graded as moderate for body weight, BMI, waist-to-hip ratio and sagittal abdominal diameter owing to downgrades for indirectness and low for waist circumference and body fat, owing to downgrades for indirectness and inconsistency (I^2=59%, P-heterogeneity<0.001; I^2=66%, P-heterogeneity<0.01, respectively).

DISCUSSION

The present systematic review and meta-analysis quantified the effect of pasta alone and pasta in the context of low-GI dietary patterns on body weight and other markers of adiposity. We identified no trial comparisons for the effect of pasta alone and 32 trial comparisons involving 2448 participants who were predominantly middle-aged and overweight or obese for the effect of pasta in the context of low-GI dietary patterns. The primary pooled analysis demonstrated that pasta in the context of low-GI dietary patterns did not contribute to weight gain, resulting in a significant weight loss of -0.63kg when compared to diets higher in GI over a median follow-up of 12 weeks. The lack of harm was reflected in the established clinical secondary outcome measures of global (BMI and body fat) and abdominal adiposity (waist circumference, waist-to-hip ratio and sagittal abdominal diameter). These findings were robust across subgroups. The findings did not differ by metabolic phenotype in those who were overweight or obese or had diabetes, which is noteworthy since these are populations who would benefit from weight management strategies. There was also no effect modification by the energy balance of the design such that the weight loss was seen even under conditions of neutral energy balance (in which participants were instructed to consume dietary advice ad libitum), suggesting that encouragement of the consumption of pasta in the context of a low-GI dietary patterns does not cause harm and may even lead to spontaneous weight loss. There was also no effect modification by follow-up either in continuous meta-regression or categorical, where the 24 trials with <24weeks follow-up had a weight reduction similar to those 8 trials with ≥24weeks follow-up (-0.63kg vs -0.57kg, respectively). This is of relevance since many dietary studies are successful in demonstrating weight loss in the short term but not over the long term.

Findings in the context of existing studies

We are not aware of any RCTs directly assessing the effect of pasta intake on health parameters including body weight. Our findings, however, agree with earlier systematic reviews and meta-analyses of RCTs of the effect of low-GI dietary patterns irrespective of pasta intake on body weight and adiposity. The systematic review and meta-analysis by Thomas et al. in 2007 found a significant -1.1kg weight loss and -1.3kg/m^2 reduction in BMI favouring low-GI or glycemic load (GL) diets compared to control diets in 6 RCTs of 5 weeks to 6 months in duration in overweight or obese individuals[13]. Another systematic review and meta-analysis by Schwingshackl et al. in 2013 found a -0.62kg weight loss favouring low-GI/GL diets compared to higher GI/GL diets in 14 RCTs with greater than 6 months duration in overweight individuals (BMI>25kg/m^2)[14].

Our findings also agree with trials in which pasta was emphasized in the context of other healthy dietary patterns. One trial done in children in Spain given Mediterranean dietary advice which included increasing the intake of pasta found that approximately 11.3% of the participants in the Mediterranean diet group who were classified as overweight and obese changed their weight status to normal weight compared to only approximately 2.6% of the participants in the control group [55].

Other lines of evidence from observational studies have demonstrated benefits of pasta consumption on body weight and adiposity. Pasta intake was recently assessed in the Moli-sani study and the Italian Nutrition & HEalth Survey (INHES), a cross-sectional study of over 20,000 Italians from all over Italy[56]. The study demonstrated that higher pasta consumption was

associated with lower BMI, waist circumference and waist-to-hip ratio and with a lower prevalence of overweight and obesity[56]. Furthermore, greater pasta intake was associated with better adherence to the Mediterranean diet, a dietary pattern which has a demonstrated cardiovascular benefit[57]. Similar associations between greater pasta intake and lower body weight have also been observed in prospective U.S. cohorts[58]. A pooled analysis of the 3 Harvard cohorts also showed that higher GI diets (which would preclude pasta) were associated with weight gain[59].

Although the product form of pasta can vary widely, including in shape (e.g. macaroni, spaghetti, linguine), ingredients (e.g. type of wheat, egg content), and processing technique (e.g. drying temperature), studies have demonstrated that when comparing pastas varying in these parameters, despite slight variations in glycemic response among pastas, glycemic responses are still lower compared to a control, e.g. white bread[60,61]. One concern of the choice of pasta as a carbohydrate food is that is it a refined food low in fibre. Although there are whole grain pasta options available, studies have demonstrated that fiber added to pasta, does not significantly affect the glucose or insulin response, the secretion of gut hormones or satiety[62,63]. Furthermore, pasta has a similar glycemic response compared to many fiber-rich carbohydrates, including barley, legumes and steel cut oats, and still a lower glycemic response compared to other fiber-rich foods including whole wheat bread, breakfast cereals like bran flakes and potatoes with skin[64]. The typically consumed white wheat pasta also has a higher micronutrient content compared to other white wheat products like bread since it contains the aleurone layer which is preserved as a result of the use of harder wheats (durum wheat); even when durum wheats are used in breads, pasta retains a lower glycemic response primarily because of the processing

techniques used in pasta making which give pasta a compact structure and reduced starch hydrolysis[61].

The mechanism by which pasta in the context of low-GI dietary patterns lead to weight loss even under conditions of ad libitum dietary advice is unclear. Lower GI diets may result in greater body weight reduction compared to higher GI diets because lower GI foods may be more satiating [65] and have been shown to delay hunger and decrease subsequent energy intake[13]. Low-GI dietary patterns are also characterized by high fiber content[64,66] which may also contribute to improvements in satiety and hunger[17]. Furthermore, studies which have compared ad libitum low-GI dietary patterns to standard energy-restricted low-fat diets, demonstrated equivalency or better weight loss when following the low-GI diet, despite the fact that they could eat as much as they desired[13,67]. Thus, voluntary energy intake may be lower after low-GI meals, as has been previously demonstrated[68].

Strengths and limitations

The strengths of the present systematic review and meta-analysis include that it is comprehensive, includes RCTs which protects against bias and uses GRADE to evaluate the quality of evidence. Additionally, a large number of trials were identified (32 trials) for the primary outcome of body weight, the median follow-up period was 12 weeks which allows for the assessment of a moderate duration of intervention, none of the trials were rated as having a serious risk of bias, and there was no evidence of publication bias.

There are several limitations. First, we downgraded the certainty of the evidence for serious inconsistency in the treatment estimates across trials for some of the outcomes assessed. There was evidence of unexplained heterogeneity in waist circumference (I^2=62%) and in body fat (I^2=65%). Although the inconsistency in these outcomes may have related to measurement error[69] in the different techniques for measuring waist circumference and body fat, we were unable to conduct sensitivity or subgroup analyses to explore this source of heterogeneity. Second, we downgraded the certainty of the evidence for serious indirectness. Most of the available trials did not quantify the amount of pasta consumed in the context of the low-GI dietary patterns. Although sensitivity analyses in which analyses were restricted to the 11 trials that did quantify (providing a median 3.33 servings/week) pasta intake did not meaningfully alter our estimates (-0.70kg versus -0.63kg), it is difficult to quantify the effect of pasta in these diets. There is also the question of indirectness in the translation to other background diets. None of the available trials evaluated the effect of pasta alone or in the context of other dietary patterns. Whether the observed effect of pasta in the context of low-GI dietary patterns will hold in the context of other healthy dietary patterns, such as Mediterranean and Vegetarian dietary patterns, is unclear. Although there is no biological reason to doubt that the findings would hold across different dietary patterns, there was no direct evidence to support this conclusion. If the question had been asked from the perspective of benefit as opposed to that of harm, then the relatively short duration of the included trials is another reason to downgrade for serious indirectness. In the absence of long-term trials (>1 year diet duration), it is difficult to conclude with certainty that the observed lack of harm implies an actual sustainable benefit. Finally, there was some evidence of imprecision for benefit. Whereas the 95% CI of the pooled estimates did not overlap with our pre-specified MID for harm (that is, they did not contain evidence for harm) and so

were not downgraded for imprecision, the upper bound of the 95% CI did overlap with the lower bound of the same MID to assess the precision of the evidence for benefit for some outcomes.

Balancing these strengths and limitations, GRADE assessed the overall quality and strength of the available evidence of the effect of pasta in the context of low-GI dietary patterns as moderate for the primary outcome of body weight and the secondary outcomes of BMI, waist-to-hip ratio and sagittal abdominal diameter owing to downgrades for indirectness. The evidence was assessed as low for the other secondary outcomes of body fat and waist circumference owing to downgrades for indirectness and inconsistency.

Implications

These results are important considering the negative messages directed at the public regarding carbohydrates, which is influencing their food choices, as is evident in recent reductions in carbohydrate intake[70-72], and in particular reductions in pasta consumption[70,73-76]. Contrary to these concerns, the available evidence shows that when pasta is consumed in the context of low-GI dietary patterns that there is not weight gain but rather marginally clinically significant weight loss (>0.5kg)[77].

Although we were able to approximate the amount of pasta consumed in one third of included trials, it is unclear what the effect of pasta would be in the context of other dietary patterns. Low-GI dietary patterns, however, shares many similarities with a Mediterranean diet, which emphasizes many low-GI foods and has demonstrated a CVD benefit[57].

Current clinical practice guidelines already suggest the replacement of high GI foods with low-GI foods for improvement of glycemic control and cardiovascular risk factors[78,79]. The present evidence means that pasta may be highlighted as an important example of a low-GI food which can contribute to a low-GI dietary pattern, a pattern which in turn may potentially improve cardio-metabolic risk without an adverse effect on weight control.

CONCLUSIONS

In conclusion, the available evidence from RCTs does not allow us to conclude that pasta consumed in the context of low-GI dietary patterns has an adverse effect on body weight and adiposity outcomes of importance in the prevention and management of overweight and obesity. On the contrary, pasta in the context of low-GI dietary patterns reduces body weight and BMI compared with higher-GI dietary patterns. The results are generalizable in the context of a high carbohydrate dietary pattern composed of low-GI foods with or without the intention of weight loss in middle-aged individuals who are overweight or obese or have diabetes. Although the clinical significance of the observed weight loss is debatable, this finding increases our confidence that pasta in the context of low-GI dietary patterns does not result in weight gain. Further research may change our confidence in the estimates for our primary outcome body weight and several key secondary outcomes including BMI and two measures of abdominal adiposity, waist-to-hip ratio and sagittal abdominal diameter. More research is needed, to improve our estimates for the secondary outcomes, body fat and waist circumference and assess whether our findings extend to related cardio-metabolic outcomes. There is also a need for more randomized trials of >1 year diet duration to clarify whether the lack of harm for pasta in the context of low-GI dietary patterns will translate into meaningful long-term benefits. Other

randomized trials should focus on whether pasta will have similar effects in the context of other healthy dietary patterns such as a Mediterranean diet.

Figure Legend

Figure 1: Literature Search

Figure 2: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg). n = 2448. Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.
CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids; Pro, protein.

Figure 3: Plot of pooled effect estimates from randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on global and abdominal markers of adiposity. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.
BMI; body mass index; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet.

Acknowledgements

We wish to thank Teruko Kishibe, an information specialist in the Scotiabank Health Sciences Library, St Michael's Hospital, for her help in the development of search terms used. Aspects of this work were presented at the Società Italiana di Nutrizione Umana (SINU) Congress, in Parma, Italy, on April 12, 2017 and at the 35th International Symposium on Diabetes and Nutrition, of the Diabetes and Nutrition Study Group (DNSG) of the European Association for the Study of Diabetes (EASD) in Skagen, Denmark, on June 20 and 21, 2017.

Data Sharing

No additional data available.

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Ethics Approval

Not required.

Authorship:

All authors had full access to all of the data (including statistical reports and tables) in this study and take full responsibility for the integrity of the data and the accuracy of the data analysis.

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Transparency declaration: As guarantor, J.L. Sievenpiper affirms that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

Funding:

This work was funded by the Canadian Institutes of Health Research (funding reference number, 129920) through the Canada-wide Human Nutrition Trialists' Network (NTN). The Diet, Digestive tract, and Disease (3-D) Centre, funded through the Canada Foundation for Innovation (CFI) and the Ministry of Research and Innovation's Ontario Research Fund (ORF),

provided the infrastructure for the conduct of this project. **L Chiavaroli** was funded by a Toronto 3D Postdoctoral Fellowship Award. **DJA Jenkins** was funded by the Government of Canada through the Canada Research Chair Endowment. **JL Sievenpiper** was funded by a PSI Graham Farquharson Knowledge Translation Fellowship, Canadian Diabetes Association (CDA) Clinician Scientist award, CIHR INMD/CNS New Investigator Partnership Prize, and Banting & Best Diabetes Centre Sun Life Financial New Investigator Award. None of the sponsors had a role in any aspect of the present study, including design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, approval of the manuscript or decision to publish.

Conflicts of Interest:

All authors have completed the Unified Competing Interest form (available on request from the corresponding author) and declare: **CWC Kendall** has received research support from the Advanced Foods and Material Network, Agrifoods and Agriculture Canada, the Almond Board of California, the American Pistachio Growers, Barilla, the California Strawberry Commission, the Calorie Control Council, CIHR, the Canola Council of Canada, the Coca-Cola Company (investigator initiated, unrestricted grant), Hain Celestial, the International Tree Nut Council Nutrition Research and Education Foundation, Kellogg, Kraft, Loblaw Companies Ltd., Orafti, Pulse Canada, Saskatchewan Pulse Growers, Solae and Unilever. He has received travel funding, consultant fees and/or honoraria from Abbott Laboratories, the Almond Board of California, the American Peanut Council, the American Pistachio Growers, Barilla, Bayer, the Canola Council of Canada, the Coca-Cola Company, Danone, General Mills, the International Tree Nut Council Nutrition Research and Education Foundation, Kellogg, Loblaw Companies Ltd., the Nutrition Foundation of Italy, Oldways Preservation Trust, Orafti, Paramount Farms, the Peanut Institute,

PepsiCo, Pulse Canada, Sabra Dipping Co., Saskatchewan Pulse Growers, Solae, Sun-Maid, Tate and Lyle, and Unilever. He is on the Clinical Practice Guidelines Expert Committee for Nutrition Therapy of the European Association for the Study of Diabetes (EASD) and has served on the scientific advisory board for the Almond Board of California, the International Tree Nut Council, Oldways Preservation Trust, Paramount Farms and Pulse Canada. **DJAJ** has received research grants from Saskatchewan Pulse Growers, the Agricultural Bioproducts Innovation Program through the Pulse Research Network, the Advanced Foods and Material Network, Loblaw Companies Ltd., Unilever, Barilla, the Almond Board of California, Agriculture and Agri-food Canada, Pulse Canada, Kellogg's Company, Canada, Quaker Oats, Canada, Procter & Gamble Technical Centre Ltd., Bayer Consumer Care, Springfield, NJ, Pepsi/Quaker, International Nut & Dried Fruit (INC), Soy Foods Association of North America, the Coca-Cola Company (investigator initiated, unrestricted grant), Solae, Haine Celestial, the Sanitarium Company, Orafti, the International Tree Nut Council Nutrition Research and Education Foundation, the Peanut Institute, the Canola and Flax Councils of Canada, the Calorie Control Council (CCC), the CIHR, the Canada Foundation for Innovation and the Ontario Research Fund. He has received in-kind supplies for trial as a research support from the Almond board of California, Walnut Council of California, American Peanut Council, Barilla, Unilever, Unico, Primo, Loblaw Companies, Quaker (Pepsico), Pristine Gourmet, Bunge Limited, Kellogg Canada, WhiteWave Foods. He has been on the speaker's panel, served on the scientific advisory board and/or received travel support and/or honoraria from the Almond Board of California, Canadian Agriculture Policy Institute, Loblaw Companies Ltd, the Griffin Hospital (for the development of the NuVal scoring system, the Coca-Cola Company, EPICURE, Danone, Diet Quality Photo Navigation (DQPN), FareWell, Verywell, True Health Initiative, Saskatchewan Pulse Growers,

Sanitarium Company, Orafti, the Almond Board of California, the American Peanut Council, the International Tree Nut Council Nutrition Research and Education Foundation, the Peanut Institute, Herbalife International, Pacific Health Laboratories, Nutritional Fundamental for Health, Barilla, Metagenics, Bayer Consumer Care, Unilever Canada and Netherlands, Solae, Kellogg, Quaker Oats, Procter & Gamble, the Coca-Cola Company, the Griffin Hospital, Abbott Laboratories, the Canola Council of Canada, Dean Foods, the California Strawberry Commission, Haine Celestial, PepsiCo, the Alpro Foundation, Pioneer Hi-Bred International, DuPont Nutrition and Health, Spherix Consulting and WhiteWave Foods, the Advanced Foods and Material Network, the Canola and Flax Councils of Canada, the Nutritional Fundamentals for Health, Agri-Culture and Agri-Food Canada, the Canadian Agri-Food Policy Institute, Pulse Canada, the Saskatchewan Pulse Growers, the Soy Foods Association of North America, the Nutrition Foundation of Italy (NFI), Nutra-Source Diagnostics, the McDougall Program, the Toronto Knowledge Translation Group (St. Michael's Hospital), the Canadian College of Naturopathic Medicine, The Hospital for Sick Children, the Canadian Nutrition Society (CNS), the American Society of Nutrition (ASN), Arizona State University, Paolo Sorbini Foundation and the Institute of Nutrition, Metabolism and Diabetes. He received an honorarium from the United States Department of Agriculture to present the 2013 W.O. Atwater Memorial Lecture. He received the 2013 Award for Excellence in Research from the International Nut and Dried Fruit Council. He received funding and travel support from the Canadian Society of Endocrinology and Metabolism to produce mini cases for the Canadian Diabetes Association (CDA). He is a member of the International Carbohydrate Quality Consortium (ICQC). His wife, ALJ, is a director and partner of Glycemic Index Laboratories, Inc., and his sister received funding through a grant from the St. Michael's Hospital Foundation to develop a cookbook for

one of his studies. **JL Sievenpiper** has received research support from the Canadian Institutes of health Research (CIHR), Canadian Diabetes Association (CDA), PSI Foundation, Banting and Best Diabetes Centre (BBDC), Canadian Nutrition Society (CNS), American Society for Nutrition (ASN), Calorie Control Council, INC International Nut and Dried Fruit Council Foundation, National Dried Fruit Trade Association, The Coca-Cola Company (investigator initiated, unrestricted), Dr. Pepper Snapple Group (investigator initiated, unrestricted), The Tate and Lyle Nutritional Research Fund at the University of Toronto, and The Alberta Pulse Growers Glycemic Control and Cardiovascular Disease in Type 2 Diabetes Fund at the University of Toronto. He has received in-kind research support from the Almond board of California, Walnut Council of California, American Peanut Council, Barilla, Unilever, Unico, Primo, Loblaw Companies, Quaker (Pepsico), Pristine Gourmet, Bunge Limited, Kellogg Canada, WhiteWave Foods. He has received reimbursement of travel expenses, speaker fees, and/or honoraria from the Canadian Diabetes Association (CDA), Canadian Nutrition Society (CNS), University of Alabama at Birmingham, Oldways Preservation Trust, Nutrition Foundation of Italy (NFI), Diabetes and Nutrition Study Group (DNSG) of the European Association for the Study of Diabetes (EASD), International Life Sciences Institute (ILSI) North America, Dr. Pepper Snapple Group, Corn Refiners Association, World Sugar Research Organization, Dairy Farmers of Canada, Società Italiana di Nutrizione Umana (SINU), III World Congress of Public Health Nutrition, C3 Collaborating for Health, Sprim Brasil, WhiteWave Foods, Rippe Lifestyle, mdBriefcase, Federation of European Nutrition Societies (FENS), New York Academy of Sciences, International Diabetes Federation, American Heart Association (AHA), American Society for Nutrition (ASN), FoodMinds LLC, Memac Ogilvy & Mather LLC, Pulse Canada, Pepsico, BCFN Foundation, The Ginger Network LLC, and Dietitians of Canada. He has ad hoc

consulting arrangements with Winston & Strawn LLP, Perkins Coie LLP, and Tate & Lyle. He is a member of the European Fruit Juice Association Scientific Expert Panel. He is on Clinical Practice Guidelines Expert Committees of the Canadian Diabetes Association (CDA) European Association for the study of Diabetes (EASD), and Canadian Cardiovascular Society (CCS), as well as an expert writing panel of the American Society for Nutrition (ASN). He serves as an unpaid scientific advisor for the Food, Nutrition, and Safety Program (FNSP) and the Technical Committee on Carbohydrates of the International Life Science Institute (ILSI) North America. He is a member of the International Carbohydrate Quality Consortium (ICQC), Executive Board Member of the Diabetes and Nutrition Study Group (DNSG) of the EASD, and Director of the Toronto 3D Knowledge Synthesis and Clinical Trials foundation. His wife is an employee of Unilever Canada. No competing interests were declared by **CR Braunstein**, **S** Blanco Mejia**, and LA Leiter.**

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Table 1: Summary of trial characteristics

Trial Characteristics*	ALL	Neutral Energy Balance	Negative Energy Balance
Trial Number (n)	32	23	9
Trial Size (total, range)	2448 (8 - 250)	1989 (8 - 250)	459 (13 - 123)
Male: Female[a] (%)	40:60	47:53	27:73
Age (years)	50 (40 - 58)	52.0 (42.1 – 59.5)	49.5 (34.2 – 53.0)
Metabolic Phenotype (OW/OB:DM:CHD) (%)	66:31:3	57:39:4	89:11:0
Setting (IP:OP) (%)	3:97	4:96	0:100
Baseline Body Weight (kg)[b]	85.5 (80.0 - 91.9)	84.1 (79.5 – 87.5)	92.5 (86.1 – 93.9)
Baseline BMI (kg/m^2)[c]	30.4 (28.2 - 32.0)	29.5 (27.4 – 31.4)	31.7 (30.1 – 32.9)
Study Design (C:P) (%)	19:81	26:74	0:100
Dose Pasta (servings/week)[d]	3.3 (2.3 - 3.5)	3.4 (2.9 – 4.1)	2.3 (2.3-3.5)
GI in Pasta/LGI group	49.0 (44.0 - 55.1)	46.5 (49.9 – 55.5)	44.0 (42.3 - 49.4)
GI in Higher GI group	62.5 (61.6 - 63.2)	63.3 (60.1 – 64.4)	61.0 (59.2 – 66.6)
Calorie reduction in Pasta/LGI group (kcal)[e]	-179 (-90 - -448)	-165 (-74 - -313)	-447 (-134 - -594)
Calorie reduction in Higher GI group (kcal)[e]	-181 (-93 - -401)	-160 (-40 - -248)	-470 (-172 - -561)
Feeding Control (Met:Supp:DA) (%)	6:44:50	4:48:48	11:33:56
Follow-Up Duration (Weeks)	12 (9-21)	12 (6-24)	12 (10 - 21)
Funding Sources (A:I:AI:NR) (%)	47:9:25:19	44:13:26:17	56:0:22:22

* median (inter quartile range), unless otherwise indicated
[a]24/32 trials provided data on sex
[b] 30/32 trials reported baseline body weight
[c] 28/32 trials reported baseline BMI
[d] 11/32 trials provided data from which dose could be approximated
[e] 20/32 trials provided data from which to approximate changes in caloric intake
A, agency; AI, agency and industry; BMI, body mass index; C, crossover design; CHD, coronary heart disease; DA, dietary advice; DM, diabetes; GI, glycemic index; I, industry; IP, inpatient; LGI, low glycemic index; Met, metabolic; NR, not reported; OB, obese; OP, outpatient; OW, overweight; P, parallel design; Suppl, supplemented/provision of certain food

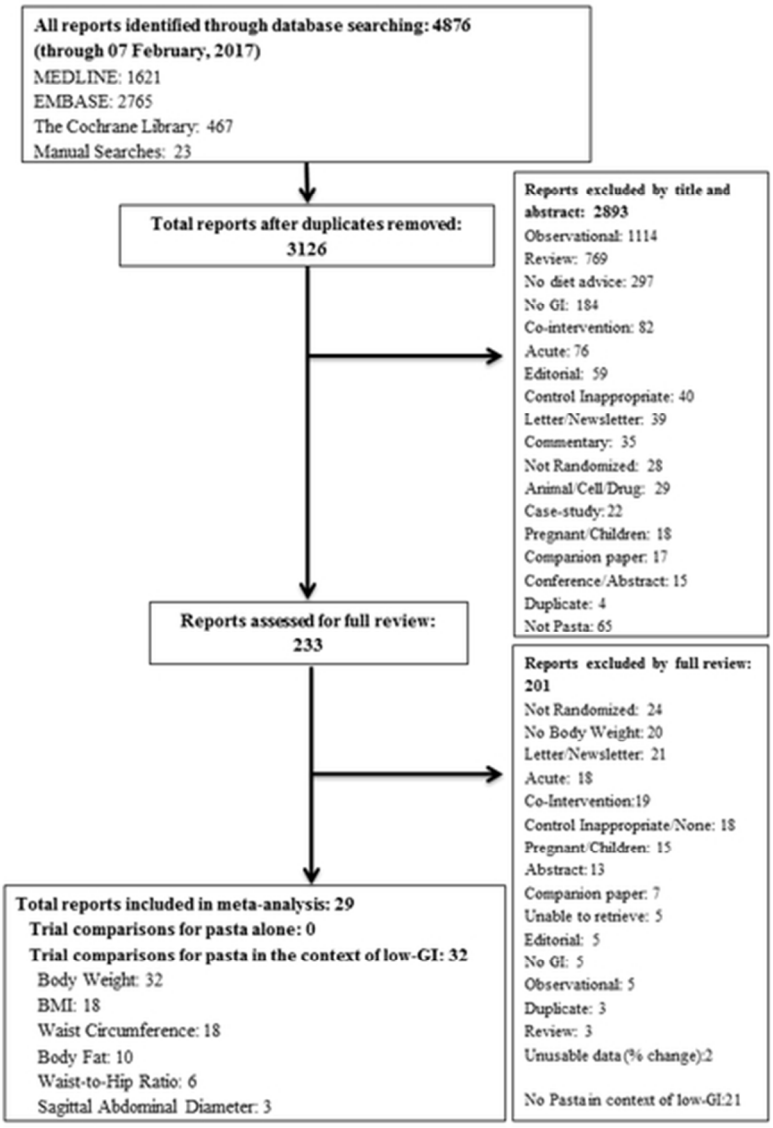

Figure 1: Literature Search

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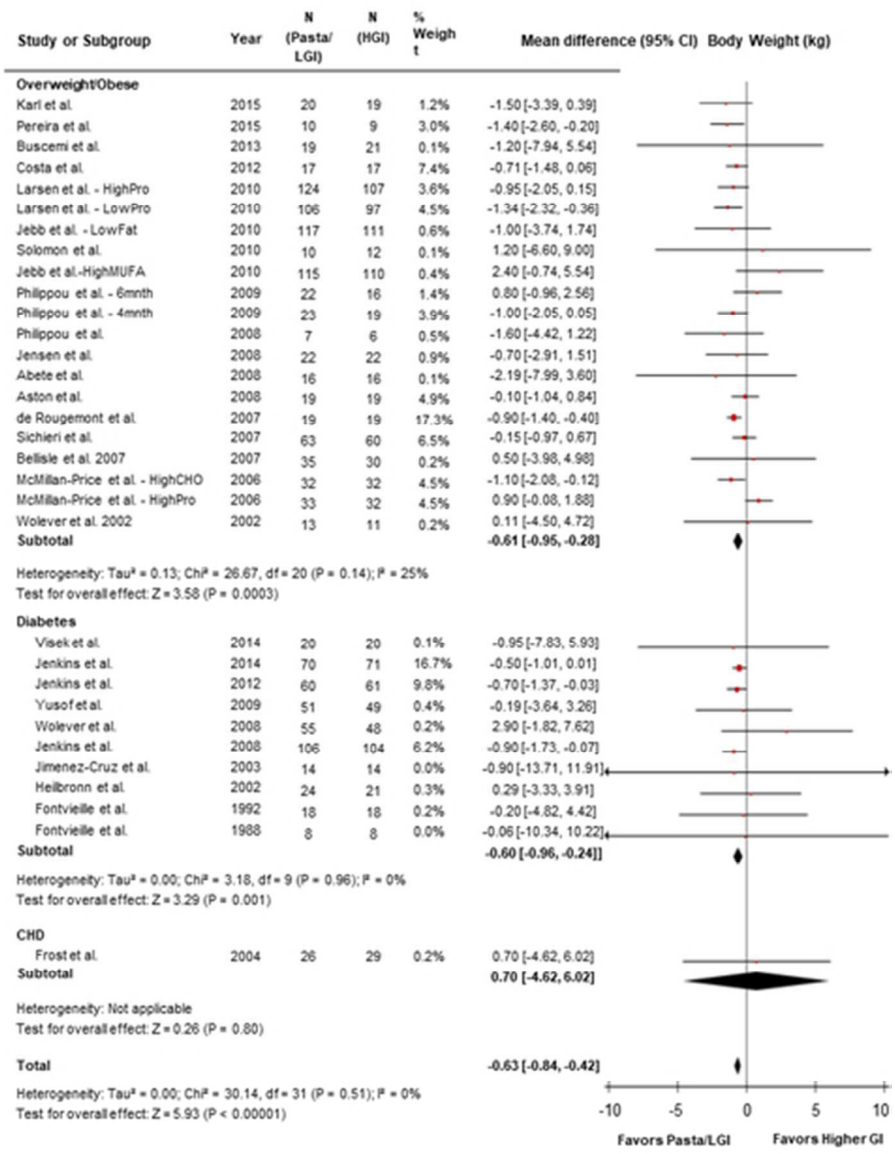

Figure 2: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg). n = 2448. Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I2 statistic where I2≥50% is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.

CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids; Pro, protein.

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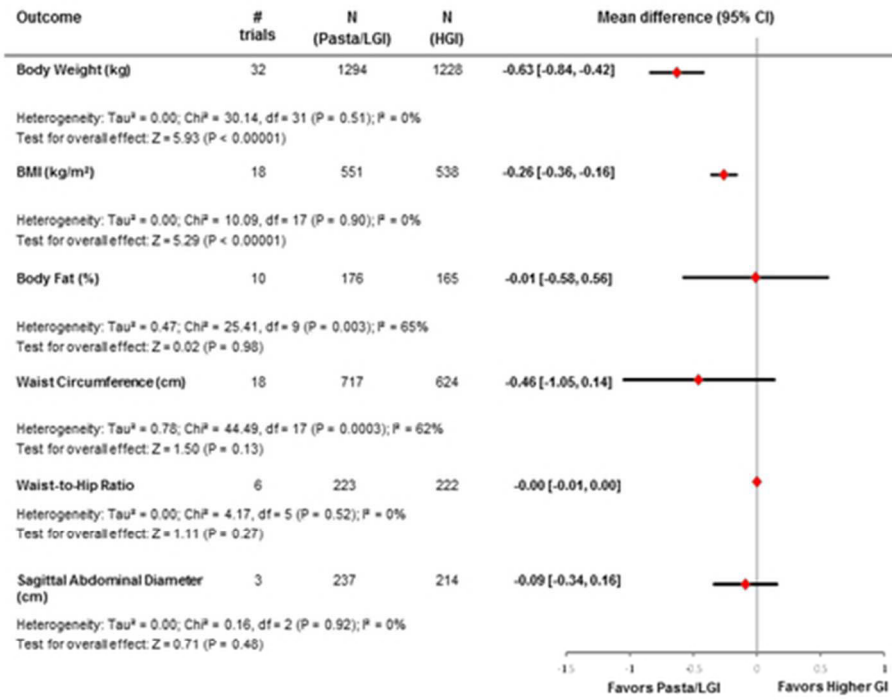


Figure 3: Plot of pooled effect estimates from randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on global and abdominal markers of adiposity. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I2 statistic where I2≥50% is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.
BMI; body mass index; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet.

40x54mm (300 x 300 DPI)

Online Supplemental Information

Table of Contents

Supplemental Tables

Supplemental Table S1: PRISMA Checklist

PRISMA 2009 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.	2-3
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.	6
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.	6-7, Supplemental Table S2
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.	6, Supplemental Table S1
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	Supplemental Table S1
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).	6-7, Figure 1
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.	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.	7, Supplemental Table S2
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.	7
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	8
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.	8-10

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).	10
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	8-10
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.	11, 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.	11-12, Table 1, Supplemental Tables S2-S3
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).	12, 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.	12-13, Figures 2-3, Supplemental Figures S3-S7
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	12-13, Figures 2-3, Supplemental Figures S3-S7
Risk of bias	22	Present results of any assessment of risk of bias across studies	12,16

across studies		(see Item 15).	Supplemental Table S1, S2, S12, S15, S17, S20
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	13-16, Supplemental Tables S4-S10, Supplemental Figures S8-S26
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).	17-20
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).	20-22
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	22-24
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.	27-28

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 studies assessing the effect of pasta in the context of low-GI dietary patterns on body weight in randomized controlled trials

Database	Search Period	Search Terms
Medline	1946 to February 07, 2017	1. pasta/ 2. spaghetti/ 3. macaroni/ 4. lasagna/ 5. fusilli/ 6. noodle/ 7. glycaemic index.tw. 8. glycemic index.tw. 9. glycaemic ind*.tw. 10. glycemic ind*.tw. 11. glycemic load*.tw. 12. glycaemic load*.tw. 13. glycemic index/ 14. body mass index/ 15. body mass index.tw. 16. BMI.tw. 17. overweight.tw. 18. weight*.tw. 19. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 20. 14 or 15 or 16 or 17 or 18 21. 19 and 20 22. limit 21 to animals 23. 21 not 22
Embase	1946 to February 07, 2017	1. pasta/ 2. spaghetti/ 3. macaroni/ 4. lasagna/ 5. fusilli/ 6. noodle/ 7. glycaemic index.tw. 8. glycemic index.tw. 9. glycaemic ind*.tw. 10. glycemic ind*.tw. 11. glycemic load*.tw. 12. glycaemic load*.tw. 13. glycemic index/ 14. body mass index/ 15. body mass index.tw. 16. BMI.tw. 17. overweight.tw. 18. weight*.tw.

		19. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 20. 14 or 15 or 16 or 17 or 18 21. 19 and 20 22. limit 21 to animals 23. 21 not 22
The Cochrane Library	1946 to February 07, 2017	1. pasta/ 2. spaghetti/ 3. macaroni/ 4. lasagna/ 5. fusilli/ 6. noodle/ 7. glycemic index/ 8. glycaemic ind*.tw. 9. glycemic ind*.tw. 10. glycemic load*.tw. 11. glycaemic load*.tw. 12. exp body weight/ 13. body weight*.tw. 14. BMI.tw. 15. body mass index/ 16. body mass index.tw. 17. weight*.tw. 18. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 19. 12 or 13 or 14 or 15 or 16 or 17 20. 18 and 19 21. limit 20 to animals 22. 20 not 21

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**
Adult men and women excluding pregnant or breastfeeding women	Low glycemic index interventions where pasta is included as part of the intervention	Higher glycemic index diets where pasta is not included as part of the intervention	Body weight Body mass index (BMI) Body Fat (%) Waist circumference Waist-to-hip ratio

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

Supplement Table S4a: Trial characteristics

Overweight/Obese Trials

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Karl et al. 2015 ‡‡‡	39 (19M:20F)**	OB, OP					2.33	USA	P	Negative+ Neutral	17		CR to 2/3kcal; Metabolic	Agency
Low GI	20		56 (5)*	92.9 (13.6)*	32.3 (3.4)*	42:133						68:15:16		
Higher GI	19		56 (5)*	94 (9.7)*	33.4 (2.6)*	61:201						70:16:14		
Pereira et al. 2015	19 (4M:15F)**	OW, IP/OP					NR	Brazil	P	Neutral	6.4		Ad libitum	Unknown
Low GI	10		28(5)	80.0(12.6)	29.9 (2.1)	41.2(2.2) ¥ **						48.3:16.1:32.8		
Higher GI	9		26(3)	79.1(12.2)	29.1 (2.0)	74.1(2.9) ¥ **						54.6:12.7:34.4		
Buscemi et al. 2013	40 (19M:21F)**	OW/OB, high CVD risk, OP					NR	Italy	P	Negative	12		CR to 20kcal/kg/d; Ad libitum	Unknown
Low GI	19		51 (8)	93.8 (17.3)	34.3 (6.6)	48.1: 138						56:18:26		
Higher GI	21		49 (8)	93.2 (14.4)	34.5 (5.1)	59.3: 174						57:16:27		
Costa et al. 2012	17 (7M:10F)	OW, IP/OP	25.4 (5.8)	84.1(16.3)	26.3(3.2)		NR	Brazil	C	Neutral	4		Ab libitum,2 meals+3 fruit/d provided	NR
Low GI						47.5(3.8)						58.6:13.9:25.5		
Higher GI						61.6(2.8)						55.4:14.2:30.3		
Jebb et al. 2010 - High MUFA	225**	OH, some CV risk factors, OP	~52		~28.5+		1.75	UK	P	Neutral	24		Ad Libitum, key foods provided	Agency, foods by industry
Low GI	115			83.7 (69.6-93.1)¶¶		~55.2						~44.6:16.4:35.7****		
Higher GI	110			80.5 (70.0-92.1)¶¶		~63.3						~44.9:15.3:35.6****		
Jebb et al. 2010 - Low Fat	250**	OH, some CV risk factors, OP	~52		~28.5+		3.5	UK	P	Neutral	24		Ad Libitum, key foods provided	Agency, foods by industry
Low GI	117			79.4 (70.1- 91.8)¶¶		~56.3						~51.5:14.2:26.1****		
Higher GI	111			80.7 (71.4- 91.4)¶¶		~64.4						~51.1:15.7:27.5****		
Larsen et al. 2010-High Pro	231**	OW/OB, OP			NR		NR	Europe	P	Neutral	26		Ad libitum	Agency
Low GI	124		42.1 (6.5)	88.5 (15.6)		~56.5: 108.9						~43:22:32		
Higher GI	107		42 (5.7)	89.5 (17.1)		~61.4: 113.1						~45:23:31		
Larsen et al. 2010 - Low Pro	203 **	OW/OB, OP			NR		NR	Europe	P	Neutral	26		Ad libitum	Agency
Low GI	106		42.2 (5.7)	88.4 (15.7)		~56.2: 121						~51:18:30		
Higher GI	97		42 (5.9)	86.6 (13.8)		~61.6: 137.9						~51:17:31		

Supplement Table S4b: Trial characteristics continued

Overweight/Obese Trials continued

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Solomon et al. 2010	22 (8M:14F)**	OB, Pre-T2DM, OP					7	USA	P	Neutral	12		Metabolic plus excerise program	Agency
Low GI	10 (3M:7F)		67 (6)	97.4 (12.0)	34.9 (1.1)	39.8 (0.9)						54.7(0.3):28.3(0.3):17.0(0.3)		
Higher GI	12 (5M:7F)		64 (3)	94.7 (15.2)	34.1 (1.1)	80.0 (2.1)						55.6(0.7):27.8(0.7):16.6(0.3)		
Philippou et al. 2009- 6 mo	38**	≥1 CHD risk factors, OP	(35-65) ¶				NR	UK	P	Negative (for all but n=2)	24		500kcal CR; Ad libitum	Agency
Low GI	22			91.3(14.8)***	29.1 (3.6)***	50.6 (4.6): 114.4(31.5)								
Higher GI	16			97.5(16.4)***	30.5 (3.5)***	63.2(5.6): 175.0(45.6)								
Philippou et al. 2009- 4 mo	42**	OW, OP	(18-65)¶				NR	UK	P	Neutral	16		Ad libitum	Unknown
Low GI	23			87.2 (15.3)	32.5 (4.8)	49.7(5.7):89.7(27.5)						47.6(6.7):19.5(4.2):31.8(5.8)		
Higher GI	19			83.6 (13.4)	31.3 (4.8)	63.7(9.4):136.8(56.3)						48.9(7):19.3(4.9):30.9(9)		
Abete et al. 2008	32 (18M:14F)	OB, OP	36(7)				2.33	Spain	P	Negative	8		30% CR; Ad libitum, 3-day menus	Agency
Low GI	16			94.3(16.1)	32.8 (4.3)	(40-45)¶						50.2 (1.8);18.3(1.6);31.5(1.6)		
Higher GI	16			94.4(13.1)	32.2 (4.4)	(60-65)¶						47.8(6.8);19.6(5.6);32.6(4.3)		
Aston et al. 2008	19 (0M:19F)**	OW/OB, OP	51.9(7.6)	87.5 (15)	33.1 (4.9)		3.33	UK	C	Neutral	12		Ad libitum, key CHO foods provided	Agency
Low GI						55.5(3.8): 133.8(22.9)****						51.4(6.0):17.0(2.4):32.2(5.1)***		
Higher GI						63.9(3): 138.8(30.5)****						47.6(6.1):17.6(3.3):34.1(5.7)***		
Jensen et al. 2008	44 (0M:44F)**	OW, OP	(20-40)¶				3	Denmark	P	Neutral	10		Ad libitum, partial provision, menu plans	Agency, Industry
Low GI	22 (0M:22F)			77.9(6.9)	27.4 (1.5)	72¥						~57(5):17(0):23(5) ‡		
Higher GI	22 (0M:22F)			80.2(1.4)	27.6 (0.3)	95¥						~57(5):17(0):22(5) ‡		
Philippou et al. 2008	13 (5M:8F)**	≥1 CHD risk factors, OP					NR	UK	P	Negative	12		500kcal CR; Ad libitum	Agency
Low GI	7 (3M:4F)		54 (49-58)¶¶	81.5 (4.7)***	28.7 (2.1)***	51.3(51.0-52.0): 105.6 (76.9-110.1)¶¶						46.0(37.8-51.0): 17.1(15.7-17.4): 32.8(31.3-37.1)¶¶		
Higher GI	6 (2M:4F)		45 (39-50)¶¶	89.7 (12.8)***	31.5 (4.4)***	59.3(59.2-64.0): 114.7(98.5-134.9)¶¶						49.4(47.8-51.7):19.6(14.0-23.1):29.2(25.2-34.5)¶¶		

Supplement Table S4c: Trial characteristics continued

Overweight/Obese Trials continued

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Bellisle et al. 2007	65 (0M:65F)**	OW/OB, OP					NR	France	P	Neutral	12		Ad libitum	Industry
Low GI	35		46.1 (13.6)	80 (13.2)	30.2 (4.1)	na								
Higher GI	30		45.3 (12.0)	79 (13.1)	30.4 (4.4)	na								
de Rougemont et al. 2007	38 (22M:16F)**	OW, OP					2.8	France	P	Neutral	5		Ad libitum, some foods provided	Agency, Industry
Low GI	19		36.3 (8.7)	77.2 (9.6)	27.5 (1.3)	46.5 (1.3)						42.6 (3.9):19.8 (1.7):37.7 (4.4)		
Higher GI	19		40.4 (9.6)	77.3 (9.2)	27.2 (1.3)	66.3 (2.6)						44.1 (3.5):17.6 (1.7):38.4 (3.1)		
Sichieri et al. 2007	123 (OM:123F)**	OW, OP					NR	Brazil	P	Negative	72		100-300kcal CR; 6-d menu and exchange lists provided	Agency
Low GI	63		37.2 (5.4)*	67.7 (6.6)*	na	21(38): 74(84)						59.5 (6.3): 13.3: 27.2(4.6)		
Higher GI	60		37.5 (5.6)*	68.5 (7.5)*	na	51(28): 199(43)						61.6 (6.2): 12.3: 26.1(4.7)		
McMillian-Price et al. 2006-High Carb	64(16M:48F)	OW/OB, OP					NR	Australia	P	Negative	12		Ad libitum, key foods and meals provided	Agency-Industry
Low GI	32		30.5 (7.9)	87.1 (15.3)	30.6 (4.5)	45 (6):89 (28)						56 (6):19 (0):22 (6)		
Higher GI	32		31.8 (9.6)	86 (10.7)	30.9 (3.4)	70 (6):129 (45)						60 (6):18 (6):19 (6)		
McMillian-Price et al. 2006-High Protein	65(15M:50F)	OW/OB, OP					NR	Australia	P	Negative	12		Ad libitum, key foods and meals provided	Agency-Industry
Low GI	33		34.6 (8.6)	88.4 (17.2)	32.1 (5.2)	44 (6):59 (23)						40 (11):26 (6):28 (6)		
Higher GI	32		30.2 (8.5)	87.7 (16.4)	31.3 (4.5)	59 (6):75 (17)						42 (6):28 (6):27 (6)		
Wolever et al. 2002	24 (5M:19F)**	IGT, OP					NR	Canada	P	Neutral	16		Ad libitum, partial provision	Agency
Low GI	13(3M:10F)		55.2 (10.8)	79.7 (13.1)***	29.7 (4.3)	54.4 (2.5):91.8 (9.4)						54.8 (6.1):19.4 (1.8):24.7 (5.8)		
Higher GI	11(2M:9F)		58.8 (13.3)	76.4 (20.4)***	29.3 (7.3)	59.3 (2.0):96.8 (11.6)						52.8 (6.6):17.4 (2.3):27.9 (6.3)		

Supplement Table S4d: Trial characteristics continued

Diabetes Trials

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Jenkins et al. 2014	141(77M:64F)	T2DM, OP					NR	Canada	P	Neutral	12		Ad libitum, bread supplement	Industry Association
Low GI	70 (38M:32F)		59 (10)	85 (20)	30 (5)	~51:53						~38.5:19.8:37.2		
Higher GI	71 (39M:32F)		59 (10)	84 (19)	31 (6)	~62:89						~49.2:19.8:27.4		
Visek et al. 2014	20 (12M:8F)	T2DM, OP	62.7 (5.8)	91.9 (14.1)	32 (4.2)		NR	Czech Republic	C	Neutral	12		Ad libitum	Agency
Low GI						49 (48-51)¶ ¶						~37.2:18.0:36.0		
Higher GI						68 (61-72)¶ ¶						~36.2:17.3:40.0		
Jenkins et al. 2012	121 (61M: 60F)	T2DM, OP					NR	Canada	P	Neutral	12		Ad libitum	Agency
Low GI	60		58 (10.1)	85.6 (20.1)	31.4 (7.0)	47: 80						45.4:22.8:30.5		
Higher GI	61		61 (7.8)	82.5 (17.2)	29.9 (5.5)	58: 100						48.3:21.4:28.5		
Yusof et al. 2009	100**	T2DM, OP	NR				NR	Malaysia	P	Neutral	12		Ad libitum, key foods provided to lowGI group	Agency
Low GI	51			69.12 (13.33)	27.05 (4.91)	57(6): 108(32)						52(4):18(3):30(4)		
Higher GI	49			66.83 (11.50)	26.79 (4.65)	64(5): 131(30)						54(4):17(3):28(5)		
Jenkins et al 2008	210 (125M:82F)	T2DM, OP					NR	Canada	P	Neutral	24		Ad libitum	Agency
Low GI	106 (65M:41F)		60 (10)	87.0 (20.0)	30.6 (6.0)	49.4: 91.5						44.0:21.2:33.3		
Higher GI	104 (63M:41F)		61 (9)	87.8 (19.4)	31.2 (5.8)	59.3: 117.9						47.5:20.7:30.5		
Wolever et al. 2008	103	T2DM, OW/OB, OP					NR	Canada	P	Neutral	52		Ad libitum, key foods provided	Agency
Low GI	55		60.6 (7.5)*	81.1 (18.7)*	31.6 (4.5)*	55.1 (3.0): 133 (14.8)						51.9 (6.7):20.6(3.0):26.5 (5.9)		
Higher GI	48		60.4 (7.9)*	84.4(18.0)*	30.1 (4.3)*	63.2 (2.8): 135 (20.8)						46.5 (6.2):20.4 (2.8):30.8 (4.8)		

Supplement Table S4e: Trial characteristics continued

Diabetes Trials continued

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Jimenez-Cruz et al. 2003	14 (6M:8F)**	T2DM, OP	59 (34)				NR	Mexico	C	Neutral	6		Ad libitum	Industry
Low GI				91.6 (24.3)	32.4 (6.0)	44(3.4): 86(19.8)						60:21:23		
Higher GI				92.6 (25.4)	32.3 (6.0)	56(4.9): 139(27.3)						64:18:20		
Heilbronn et al. 2002	45 (23M:22F)**	T2DM, OW, OP			NR		3.5	Australia	P	Negative	8		CR to1500kcal/d; Ad libitum, partial provision	Unknown
Low GI	24 (11M:13F)		56.0(9.4)	91.7(16.2)		43						58.9 (2.9):22.2 (1.5):17.9 (3.9)		
Higher GI	21 (12M:9F)		57.5(9.6)	93.2 (13.3)		75						60.8 (2.3):21.7 (0.9):17.1 (2.3)		
Fontvieille et al. 1992	18 (12M:6F)	T1DM/T2DM, OP	47.2(11.6)	NR	24.8(2.6)		4.7	France	C	Neutral	5		Ad libitum	Agency, Industry
Low GI						38.1(5.3)						45.8(7.2):18.0(2.5):36.2(6.8)		
Higher GI						64.2(3.1)						44.9(7.3):18.8(1.6):36.3(6.0)		
Fontvielle et al. 1988	8 (4M:4F)	T1DM, OP	43.5 (9.9)	NR	24.1 (6.8)		3.5	France	C	Neutral	3		Ad libitum, partial provision	Agency, Industry
Low GI						46.5(2.5)						46.1 (4.5):17.4 (1.4):35.0 (2.8)		
Higher GI						60.1 (5.1)						45.4 (4.5):16.9 (1.7):36.0 (2.8)		
CHD Trial														
Frost et al. 2004	55 (48M:7F)**	CHD, OP					NR	UK	P	Neutral §§§	12		Ad Libitum	Unknown
Low GI	26 (23M:3F)		63.6 (9.4)	81.2 (12.2)	26.9 (3.3)	50(4):115(39)						49 (5):18 (5):31 (5)		
Higher GI	29 (25M: 4F)		61.8 (9)	81.7 (16.7)	28.7 (4.6)	57(4):106(34)						47 (10):18 (5):32 (10)		

Abbreviations: BMI=Body Mass Index; C=Crossover design; Carb=Carbohydrate; CED= carbohydrate exchange diet; CHD= Coronary Heart Disease; CR= calorie restriction; CV=cardiovascular; GI= glycemic index; GL=glycemic load; IGT= impaired glucose tolerance; IP=inpatient; IR= insulin resistant; High Carb= high carbohydrate; HI= hyperinsulinemic; M = male; mo=months; MUFA=monounsaturated fatty acids; na=not available; T1DM=type 1 diabetes mellitus; T2DM = type 2 diabetes mellitus; OB= obese; OH=otherwise healthy; OP = outpatient; OW= overweight; P=Parallel design; SD= standard deviation; F = female; UK= United Kingdom; USA= United States of America; wks=weeks; y= years

§Negative energy balance diets were designed for weight loss, Neutral energy balance diets for weight maintenance, where all diets are isocaloric between test and control groups; §§Energy from carbohydrate: protein: fat for the planned diet or if not planned; §§§ Participants were advised to lose weight if they had a BMI>28kg/m2.

¶ Range of values; ¶¶ median and interquartile range (IQR); ¶ ¶ ¶ Actual GI/GL or if not available, planned;

* Calculated before dropout; **Completer Analysis, as used in data analysis; *** completer analysis, data obtained from authors; **** based on less participants than in analysis;

+ approximate based on all study arms; ¥ approximate based on test meals; ‡ approximate based on n=45 from Sloth et al. 2004, the original publication of this study;

‡‡‡ analysis includes weight loss and weight maintenance phases

Supplemental Table S5: Sensitivity analysis of the systematic removal of each trial*

	MD [95% CI], P-value I^2, P-value					
	Body Weight (kg) N=32	**BMI (kg/m^2) N=18**	**Body Fat (%) N=10**	**Waist Circumference (cm) N=18**	**Waist-to-hip Ratio N=6**	**Sagittal Abdominal Diameter (cm) N=3**
All studies	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.51	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.91	-0.01 [-0.58, 0.56], P=0.98 65%, P=0.003	-0.46 [-1.05, 0.14], P=0.13 62%, P<0.01	-0.00 [-0.01, 0.00], P=0.27 0.00%, P=0.52	-0.09 [-0.34, 0.16], P=0.48 0.00%, P=0.92
Removal of:						
OW/OB						
Pereira et al. 2015	-0.61 [-0.82, -0.39], P<0.01 0.00%, P=0.54	-0.24 [-0.34, -0.14], P<0.01 0.00%, P=0.93	0.19 [-0.25, 0.63], P=0.39 36%, P=0.13	-0.37 [-1.04, 0.31], P=0.28 62%, P<0.01	-0.00 [-0.01, 0.01], P=0.67 0.00%, P=0.58	n/a
Karl et al. 2015	-0.62 [-0.83, -0.41], P<0.01 0.00%, P=0.50	-0.25 [-0.35, -0.16], P<0.01 0.00%, P=0.91	0.10 [-0.51, 0.71], P=0.74 64%, P<0.01	n/a	n/a	n/a
Buscemi et al. 2013	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.86	-0.08 [-0.63, 0.48], P=0.79 64%, P<0.01	-0.46 [-1.06, 0.15], P=0.14 64%, P<0.01	n/a	n/a
Costa et al. 2012	-0.62 [-0.84, -0.41], P<0.01 0.00%, P=0.46	-0.26 [-0.37, -0.16], P<0.01 0.00%, P=0.86	-0.06 [-0.73, 0.61], P=0.86 66%, P<0.01	-0.33 [-0.94, 0.28], P=0.29 59%, P<0.01	n/a	n/a
Jebb et al. 2010 - LowFat	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
Larsen et al. 2010 - LowPro	-0.60 [-0.81, -0.38], P<0.01 0.00%, P=0.57	n/a	n/a	-0.45 [-1.09, 0.18], P=0.16 64%, P<0.01	n/a	-0.05 [-0.39, 0.29], P=0.79 0.00%, P=0.86
Jebb et al. 2010- HighMUFA	-0.64 [-0.85, -0.43], P<0.01 0.00%, P=0.65	n/a	n/a	n/a	n/a	n/a
Larsen et al. 2010 - HighPro	-0.62 [-0.83, -0.41], P<0.01 0.00%, P=0.48	n/a	n/a	-0.37 [-1.00, 0.26], P=0.26 62%, P<0.01	n/a	-0.13 [-0.45, 0.19], P=0.42 0.00%, P=0.92
Solomon et al. 2010	-0.63 [-0.84, -0.42], P<0.01	-0.26 [-0.36, -0.16], P<0.01	n/a	n/a	n/a	n/a

	0.00%, P=0.47	0.00%, P=0.88				
Philippou et al. 2009 - 6mnth	-0.65 [-0.86, -0.44], P<0.01 0.00%, P=0.59	-0.28 [-0.37, -0.18], P<0.01 0.00%, P=0.97	-0.12 [-0.71, 0.48], P=0.70 65%, P<0.01	-0.55 [-1.15, 0.04], P=0.07 61%, P<0.01	n/a	n/a
Philippou et al. 2009 - 4mnth	-0.61 [-0.83, -0.40], P<0.01 0.00%, P=0.48	-0.25 [-0.35, -0.15], P<0.01 0.00%, P=0.89	-0.09 [-0.72, 0.54], P=0.78 66%, P<0.01	-0.44 [-1.08, 0.19], P=0.17 64%, P<0.01	n/a	n/a
Abete et al. 2008	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	n/a	n/a	n/a	n/a	n/a
Philippou et al. 2008	-0.62 [-0.83, -0.42], P<0.01 0.00%, P=0.48	-0.26 [-0.35, -0.16], P<0.01 0.00%, P=0.89	-0.06 [-0.68, 0.55], P=0.84 68%, P<0.01	-0.41 [-1.02, 0.19], P=0.18 63%, P<0.01	n/a	n/a
Aston et al. 2008	-0.66 [-0.87, -0.44], P<0.01 0.00%, P=0.52	n/a	-0.00 [-0.70, 0.69], P=0.99 68%, P<0.01	-0.54 [-1.14, 0.07], P=0.08 62%, P<0.01	n/a	n/a
Jensen et al. 2008	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.27 [-0.36, -0.17], P<0.01 0.00%, P=0.88	n/a	-0.44 [-1.05, 0.18], P=0.16 64%, P<0.01	-0.00 [-0.01, 0.00], P=0.24 0.00%, P=0.41	-0.09 [-0.35, 0.18], P=0.51 0.00%, P=0.69
de Rougemont et al. 2007	-0.57 [-0.80, -0.34], P<0.01 0.00%, P=0.53	-0.25 [-0.36, -0.14], P<0.01 0.00%, P=0.87	0.06 [-0.57, 0.70], P=0.84 67%, P<0.01	n/a	n/a	n/a
Sichieri et al. 2007	-0.66 [-0.88, -0.45], P<0.01 0.00%, P=0.53	n/a	n/a	n/a	n/a	n/a
Bellisle et al. 2007	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.88	n/a	-0.47 [-1.08, 0.13], P=0.13 64%, P<0.01	-0.00 [-0.01, 0.00], P=0.14 0.00%, P=0.70	n/a
McMillan-Price et al. 2006 - HighCHO	-0.61 [-0.82, -0.39], P<0.01 0.00%, P=0.51	n/a	n/a	-0.38 [-1.01, 0.25], P=0.23 63%, P<0.01	n/a	n/a
McMillan-Price et al. 2006 - HighPro	-0.70 [-0.91, -0.49], P<0.01 0.00%, P=0.91	n/a	n/a	**-0.62 [-1.19, -0.05], P=0.03 55%, P<0.01**	n/a	n/a
Wolever et al. 2002	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
Diabetes:						
Visek et al. 2014	-0.63 [-0.84, -0.42], P<0.01	-0.26 [-0.36, -0.16], P<0.01	0.01 [-0.57, 0.60], P=0.96	n/a	n/a	n/a

	0.00%, P=0.46	0.00%, P=0.86	68%, P<0.01			
Jenkins et al. 2014	-0.66 [-0.88, -0.43], P<0.01 0.00%, P=0.47	-0.29 [-0.39, -0.18], P<0.01 0.00%, P=0.94	n/a	**-0.61 [-1.18, -0.04], P=0.04 50%, P=0.01**	-0.00 [-0.01, 0.00], P=0.21 0.00%, P=0.43	n/a
Jenkins et al. 2012	-0.62 [-0.84, -0.40], P<0.01 0.00%, P=0.46	-0.25 [-0.35, -0.15], P<0.01 0.00%, P=0.87	n/a	-0.44 [-1.05, 0.17], P=0.16 64%, P<0.01	-0.00 [-0.01, 0.01], P=0.64 0.00%, P=0.53	n/a
Yusof et al. 2009	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.87	n/a	-0.33 [-0.95, 0.28], P=0.29 58%, P<0.01	n/a	n/a
Jenkins et al. 2008	-0.61 [-0.83, -0.40], P<0.01 0.00%, P=0.48	-0.25 [-0.36, -0.15], P<0.01 0.00%, P=0.87	n/a	n/a	n/a	n/a
Wolever et al. 2008	-0.64 [-0.84, -0.43], P<0.01 0.00%, P=0.57	n/a	n/a	-0.50 [-1.10, 0.09], P=0.10 62%, P<0.01	n/a	n/a
Jimenez-Cruz et al. 2003	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.26 [-0.35, -0.16], P<0.01 0.00%, P=0.86	n/a	n/a	n/a	n/a
Heilbronn et al. 2002	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	n/a	n/a	n/a	n/a	n/a
Fontvieille et al. 1992	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
Fontvielle et al. 1988	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
CHD						
Frost et al. 2004	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	-0.26 [-0.36, -0.17], P<0.01 0.00%, P=0.89	n/a	-0.48 [-1.09, 0.12], P=0.12 63%, P<0.01	-0.00 [-0.01, 0.00], P=0.25 1%, P=0.40	n/a

*Sensitivity analysis included the removal of each single study from the meta-analyses one at a time and the summary effect was recalculated. An influential outlier was considered a study whose removal changed the magnitude of the pooled effect by >10%.

BMI, body mass index; CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; MD, mean difference; mnth, month; MUFA, monounsaturated fatty acids; n/a, not applicable; OB, obese; OW, overweight; Pro, protein

Supplemental Table S6: Sensitivity analyses of the use of correlation coefficients of 0.25 and 0.75 for crossover trials

	MD (95% CI), P-value I^2, P-value		
	Correlation Coefficient used in the Primary Analysis	Correlation Coefficient used in Sensitivity Analyses	
Outcome (no. crossover trials/total)	**0.5**	**0.25**	**0.75**
Body Weight, kg (6*/32)	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.51	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.51	-0.63 [-0.84, -0.43], P<0.01 0.00%, P=0.51
BMI, kg/m^2 (3/18)	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.91	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.90	-0.26 [-0.35, -0.17], P<0.01 0.00%, P=0.90
Body Fat, % (3*/10)	-0.01 [-0.58, 0.56], P=0.98 65%, P<0.01	-0.00 [-0.58, 0.58], P=0.99 64%, P<0.01	-0.02 [-0.57, 0.54], P=0.96 66%, P<0.01
Waist Circumference, cm (2*/18)	-0.46 [-1.05, 0.14], P=0.13 62%, P<0.01	-0.44 [-1.04, 0.15], P=0.14 60%, P<0.01	-0.46 [-1.07, 0.14], P=0.13 65%, P<0.01
Waist-to-hip Ratio (0/6)	-0.00 [-0.01, 0.00], P=0.27 0.00%, P=0.52	n/a	n/a
Sagittal Abdominal Diameter, cm (0/6)	-0.09 [-0.34, 0.16], P=0.48 0.00%, P=0.92	n/a	n/a

* one of these crossover trials, however, did not require the use of a correlation coefficient as complete data was available

BMI, body mass index; CI, confidence interval; MD, mean difference; no., number

Supplement Table S7. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on body weight (kg)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	24.1 – 37.1 kg/m^2	32	2448	-0.04 (-0.13, 0.06)	0.00%	0.454
Follow-up	3 – 72 wks	32	2448	0.01 (-0.01, 0.02)	0.00%	0.314
Dose Pasta	1.75 – 7.0 serv/wk	11	740	0.29 (-0.91, 1.50)	0.00%	0.595
GI[3]	21 - 72	31	2383	-0.01 (-0.04, 0.02)	0.45%	0.482
Difference in GI[2]	4.9 - 40.2	31	2383	-0.00 (-0.04, 0.03)	2.75%	0.811
Fiber[3]	8.0 - 39.4g/d	27	1851	0.02 (-0.01, 0.06)	0.00%	0.221
Change in Fiber[4]	-7.5 - +13.8g/d	17	1571	-0.02 (-0.07, 0.04)	23.13%	0.595
Difference in Fiber[2]	-8.5 - +15.3g/d	27	1851	-0.01 (-0.06, 0.04)	0.00%	0.701
Saturated Fat[3]	5.1 - 12.5%	14	1396	-0.05 (-0.30, 0.20)	34.32%	0.648
Change in Saturated Fat[4]	-8.2 - -1.2%	12	1309	-0.01 (-0.29, 0.28)	46.27%	0.965
Difference in Saturated Fat[2]	-2.0 - +2.3%	14	1396	-0.34 (-1.08, 0.39)	32.80%	0.329
CHO[3]	37.2 - 68.0%	30	2345	-0.01 (-0.05, 0.03)	0.00%	0.541
Change in CHO[4]	-7.2 - +10.1%	19	2046	-0.07 (-0.12, -0.01)	0.00%	**0.016**
Difference in CHO[2]	-11.1 - +5.4%	30	2345	0.02 (-0.05, 0.10)	0.00%	0.547
Protein[3]	13.3 - 26.1%	30	2345	0.04 (-0.04, 0.12)	0.00%	0.332

Change in Protein[4]	-0.5 - +8.6%	19	2046	0.15 (0.03, 0.27)	0.00%	**0.017**
Difference in Protein[2]	-2.5 - +3.4%	30	2345	-0.18 (-0.38, 0.02)	0.00%	0.069
Fat[3]	16.0 - 37.7%	30	2345	0.01 (-0.05, 0.06)	0.00%	0.852
Change in Fat[4]	-12.2 - +5.4%	19	2046	0.05 (-0.02, 0.12)	17.73%	0.122
Difference in Fat[2]	-4.4 - +10.6%	30	2345	0.02 (-0.05, 0.09)	0.00%	0.610

1 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 body weight on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in body weight. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.

2 Difference in diet variable between the intervention and control arms

3 Intake at the end of study in the intervention arm

4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index

Supplemental Table S8. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on BMI (kg/m^2)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	26.3 – 37.1 kg/m^2	18	1038	-0.01 (-0.06, 0.03)	0.00%	0.559
Follow-up	4 – 24 wks	18	1038	0.00 (-0.01, 0.02)	0.00%	0.559
Dose Pasta*	2.33 – 7.0 serv/wk	4	143			
GI[3]	39.8 - 72	17	973	0.01 (-0.01, 0.03)	0.00%	0.153
Difference in GI[2]	7.0 - 40.2	17	973	-0.01 (-0.03, 0.01)	0.00%	0.275
Fiber[3]	8.0 - 39.4g/d	16	935	0.01 (-0.01, 0.02)	0.00%	0.366
Change in Fiber[4]	-1.8 - +12.4g/d	10	758	0.00 (-0.03, 0.03)	0.00%	0.846
Difference in Fiber[2]	-4.9 - +13.0g/d	16	935	-0.00 (-0.02, 0.02)	0.00%	0.821
Saturated Fat*[3]	7.6 - 12.5%	7				
Change in Saturated Fat*[4]	-2.4 - -1.2%	6				
Difference in Saturated Fat*[2]	-1.0 - +2.3%	7				
CHO[3]	37.2 - 68.0%	16	935	-0.00 (-0.02, 0.01)	0.00%	0.594
Change in CHO[4]	-5.6 - +3.2%	10	758	-0.02 (-0.06, 0.02)	0.00%	0.325
Difference in CHO[2]	-11.1 - +2.0%	16	935	-0.01 (-0.03, 0.02)	0.00%	0.531
Protein[3]	13.9 – 22.8%	16	935	0.00 (-0.04, 0.04)	0.00%	0.905

Change in Protein[4]	-0.2 - +3.0%	10	758	0.01 (-0.11, 0.14)	0.00%	0.786
Difference in Protein[2]	-2.5 - +3.4%	16	935	-0.03 (-0.12, 0.06)	0.00%	0.441
Fat[3]	16.0 - 37.7%	16	935	0.00 (-0.02, 0.02)	0.00%	0.717
Change in Fat[4]	-4.8 - +5.4%	10	758	0.02 (-0.02, 0.06)	0.00%	0.240
Difference in Fat[2]	-4.4 - +10.6%	16	935	0.01 (-0.01, 0.04)	0.00%	0.299

*For Dose, there were <10 trials so subgroup analyses were not performed.

1 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 BMI on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in BMI. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.

2 Difference in diet variable between the intervention and control arms

3 Intake at the end of study in the intervention arm

4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index; wk, week

Supplemental Table S9. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on body fat (%)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	26.3 – 36.0 kg/m^2	10	285	-0.02 (-0.26, 0.23)	67.69%	0.890
Follow-up	4 – 24 wks	10	285	0.06 (-0.06, 0.17)	66.00%	0.303
Dose Pasta*	2.33 - 3.33 serv/wk	3	96			
GI[3]	39.8 - 72	10	285	0.12 (-0.00, 0.25)	52.21%	0.053
Difference in GI[2]	7.0 - 40.2	10	285	-0.09 (-0.15, -0.03)	19.39%	**0.008**
Fiber*[3]	8.0 - 39.4g/d	8	228			
Change in Fiber*[4]	-1.8 - +12.4g/d	4	87			
Difference in Fiber*[2]	-4.9 - +13.0g/d	8	228			
Saturated Fat*[3]	7.6 - 12.5%	3	93			
Change in Saturated Fat*[4]	-2.4 - -1.2%	2	51			
Difference in Saturated Fat*[2]	-1.0 - +2.3%	3	93			
CHO*[3]	37.2 - 68.0%	9	247			
Change in CHO*[4]	-5.6 - +3.2%	4	87			
Difference in CHO*[2]	-11.1 - +2.0%	9	247			
Protein*[3]	13.9 – 22.8%	9	247			

Change in Protein*[4]	-0.2 - +3.0%	4	87			
Difference in Protein*[2]	-2.5 - +3.4%	9	247			
Fat*[3]	16.0 - 37.7%	9	247			
Change in Fat*[4]	-4.8 - +5.4%	4	87			
Difference in Fat*[2]	-4.4 - +10.6%	9	247			

*There were <10 trials so subgroup analyses were not performed.

1 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 body fat on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in body fat. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.
2 Difference in diet variable between the intervention and control arms
3 Intake at the end of study in the intervention arm
4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index; wk, week

Supplemental Table S10. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on waist circumference (cm)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	26.3 – 36.0 kg/m^2	18	1380	0.10 (-0.13, 0.32)	59.02%	0.372
Follow-up	4 – 52 wks	18	1380	0.05 (-0.03, 0.13)	60.50%	0.225
Dose Pasta*	3.0 – 3.33 serv/wk	2	63			
GI[3]	41.2 – 72.0	17	1315	-0.01 (-0.12, 0.10)	66.00%	0.841
Difference in GI[2]	4.9 – 32.9	17	1315	-0.02 (-0.10, 0.06)	64.51%	0.596
Fiber[3]	8.0 - 39.4g/d	15	1258	0.05 (-0.05, 0.14)	59.60%	0.342
Change in Fiber[4]	-1.8 - +13.8g/d	13	1176	0.01 (-0.17, 0.20)	70.59%	0.861
Difference in Fiber[2]	-4.9 - +15.3g/d	15	1258	-0.00 (-0.14, 0.13)	64.86%	0.939
Saturated Fat*[3]	6.1 – 10.1%	8	604			
Change in Saturated Fat*[4]	-7.6 - -1.2%	7	562			
Difference in Saturated Fat*[2]	-2.0 - +2.3%	8	604			
CHO[3]	38.5 - 58.6%	16	1277	-0.11 (-0.19, -0.04)	27.06%	**0.007**
Change in CHO[4]	-7.2 - +10.1%	13	1176	-0.09 (-0.23, 0.04)	57.19%	0.148
Difference in CHO[2]	-11.1 - +5.4%	16	1277	-0.01 (-0.18, 0.17)	60.81%	0.947
Protein[3]	13.9 – 26.1%	16	1277	0.20 (0.01, 0.38)	43.92%	**0.038**

Change in Protein[4]	-0.3 - +8.6%	13	1176	0.19 (-0.09, 0.46)	63.38%	0.165
Difference in Protein[2]	-2.5 - +3.4%	16	1277	-0.14 (-0.66, 0.38)	62.79%	0.570
Fat[3]	22.0 - 37.2%	16	1277	0.10 (-0.05, 0.26)	53.48%	0.185
Change in Fat[4]	-12.2 - +5.4%	13	1176	0.08 (-0.07, 0.24)	61.15%	0.274
Difference in Fat[2]	-4.4 - +10.6%	16	1277	0.09 (-0.06, 0.25)	47.17%	0.220

*There were <10 trials so subgroup analyses were not performed.

1 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 waist circumference on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in waist circumference. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.

2 Difference in diet variable between the intervention and control arms

3 Intake at the end of study in the intervention arm

4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index; wk, week

Supplementary Table S11. Post-hoc piecewise linear continuous dose-response meta-regression analyses for the effect of pasta intake in the context of low-GI dietary patterns on body weight (kg)

Dose threshold of servings of pasta consumed per week	Dose ranges of servings of pasta consumed per week	β (95% CIs) *	Residual I^2 †	*p*-value
3.0	≤3.0	-0.70 (–3.27, 1.86)	0.00%	0.890
	>3.0	0.91 (–0.89, 2.70)		
3.33	≤3.33	0.05 (–1.80, 1.89)	0.00%	0.518
	>3.33	0.44 (–1.75, 2.63)		
3.5	≤3.5	0.09 (–1.65, 1.82)	0.00%	0.888
	>3.5	0.46 (–1.89, 2.81)		

* β is the slope derived from the piecewise linear meta-regression analyses and represents the treatment effect on body weight for doses above and below each dose-threshold representing servings of pasta consumption per week; † The residual I^2 value indicates heterogeneity unexplained by each dose-threshold.

Supplementary Table S12: GRADE assessment of study quality

Quality assessment*							№ of patients		Effect	Quality Importance
№ of studies	Design	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Pasta/LGI diet	Higher GI diet	Absolute (95% CI)	
Body Weight (follow up: median 12 weeks)										
32	randomised trials	not serious	not serious	serious [a]	not serious	none	1294	1228	MD **-0.63 kg** (-0.84 to -0.42)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness
BMI (follow up: median 12 weeks)										
18	randomised trials	not serious	not serious	serious [a]	not serious	none	551	538	MD **-0.26 kg/m^2** (-0.36 to -0.16)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness
Waist Circumference (follow up: median 12 weeks)										
18	randomised trials	not serious	serious [b]	serious [a]	not serious	none	717	624	MD **-0.46 cm** (-1.05 to 0.14)	⊕⊕◯◯ LOW [a,b] Due to downgrade for inconsistency and indirectness

Quality assessment*							№ of patients		Effect	Quality Importance
№ of studies	**Design**	**Risk of bias**	**Inconsistency**	**Indirectness**	**Imprecision**	**Publication Bias**	**Pasta/LGI diet**	**Higher GI diet**	**Absolute (95% CI)**	
Body Fat (follow up: median 12 weeks)										
10	randomised trials	not serious	serious [c]	serious [a]	not serious	none	176	165	MD **-0.01 %** (-0.58 to 0.56)	⊕⊕◯◯ LOW [a,c] Due to downgrade for inconsistency and indirectness
Waist-to-hip Ratio (follow up: median 12 weeks)										
6	randomised trials	not serious	not serious	serious [a]	not serious	none [d]	223	222	MD **-0.00** (-0.01 to 0.00)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness
Sagittal Abdominal Diameter (follow up: median 26 weeks)										
3	randomised trials	not serious	not serious	serious [a]	not serious	none [d]	237	214	MD **-0.09 cm** (-0.34 to 0.16)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness

CI, Confidence interval; GI, glycemic index; LGI, low glycemic index; MD, Mean difference

*All outcomes started with high quality evidence since all studies were randomized controlled trials. Risk of Bias –We rated down for risk of bias if the majority of studies were considered to be at high risk of bias. Inconsistency – We assessed inconsistency using I^2 estimates where an I^2 of 50% or higher indicates substantial heterogeneity. I^2 is the percentage of variability in the treatment estimates that is attributable to heterogeneity between studies. We rated down for inconsistency if there was substantial heterogeneity that was unexplained by any a priori sensitivity or subgroup analyses. Indirectness – We rated down for indirectness if there were factors present relating to the population, interventions, and outcomes that limited the generalizability of the results. Imprecision – We rated down for imprecision if the 95% confidence interval (95% CI) crossed the minimally important difference (MID) for harm. MIDs used for each outcome are: 0.5kg for body weight (based on Johnston et al. JAMA 2014;312(9):923-33); 0.2kg/m^2 for BMI; 2.0cm for waist circumference; 2.0% for body fat; 0.02 for waist-to-hip ratio; 2.0cm for sagittal abdominal diameter.

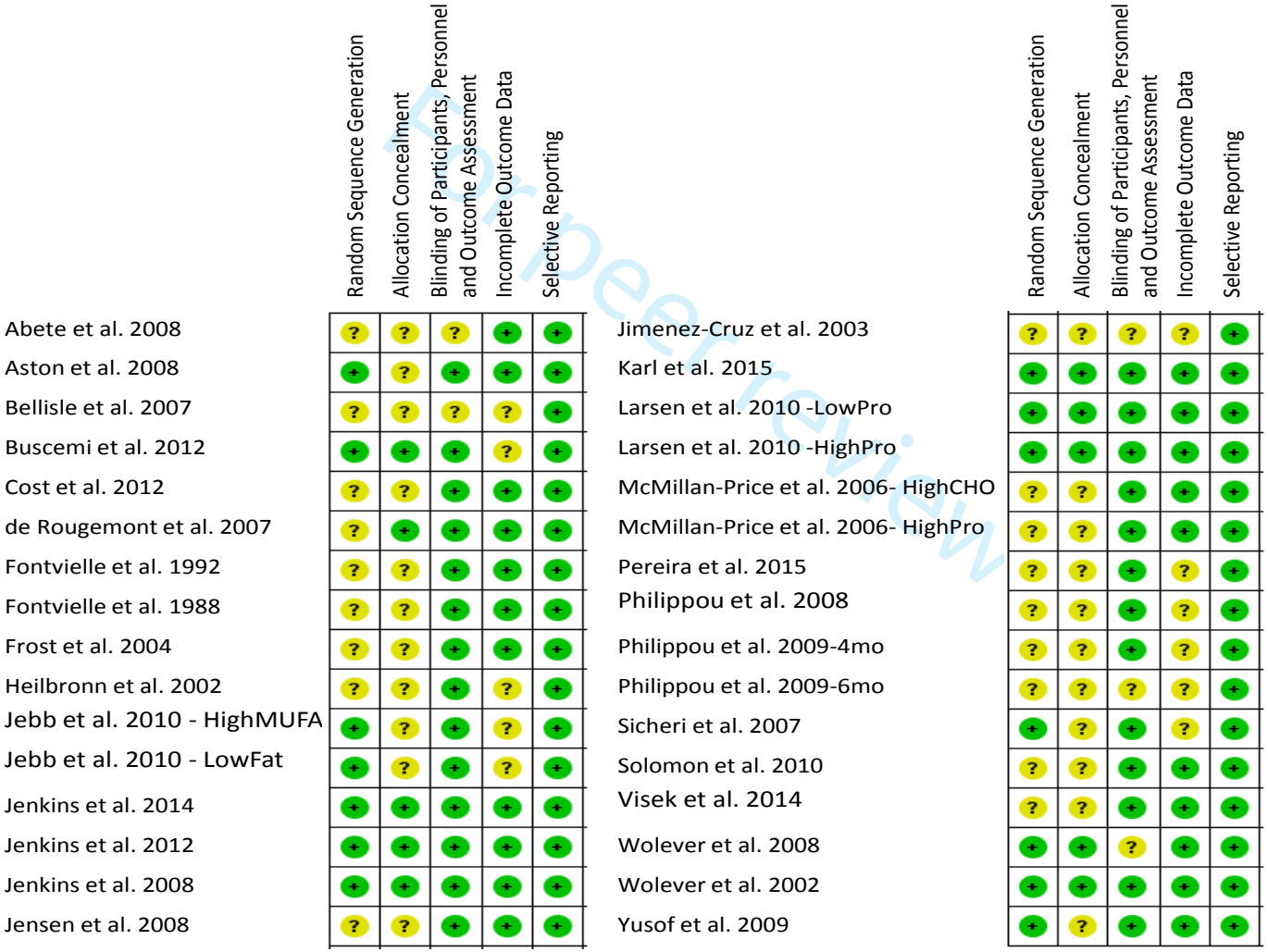
a. Downgrade for serious indirectness, since most of the available trials did not quantify the amount of pasta consumed in the context of the low-GI dietary patterns

b. Downgrade for serious inconsistency, as there was evidence of substantial inter-study (I^2=62%, P-heterogeneity<0.001), which could not be explained

c. Downgrade for serious inconsistency, as there was evidence of substantial inter-study (I^2= 65%, P-heterogeneity=0.003), which could not be explained

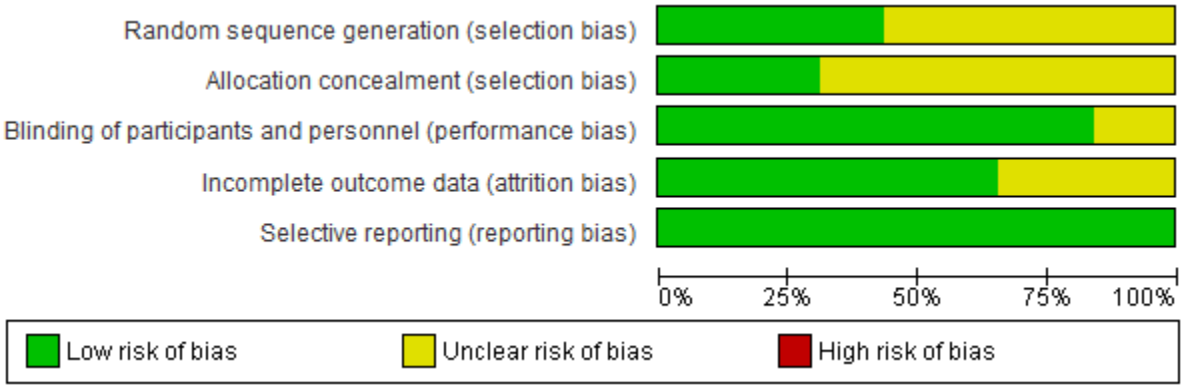
d. No downgrade for publication bias, as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (<10 trials included in the meta-analysis)

Supplemental Figures

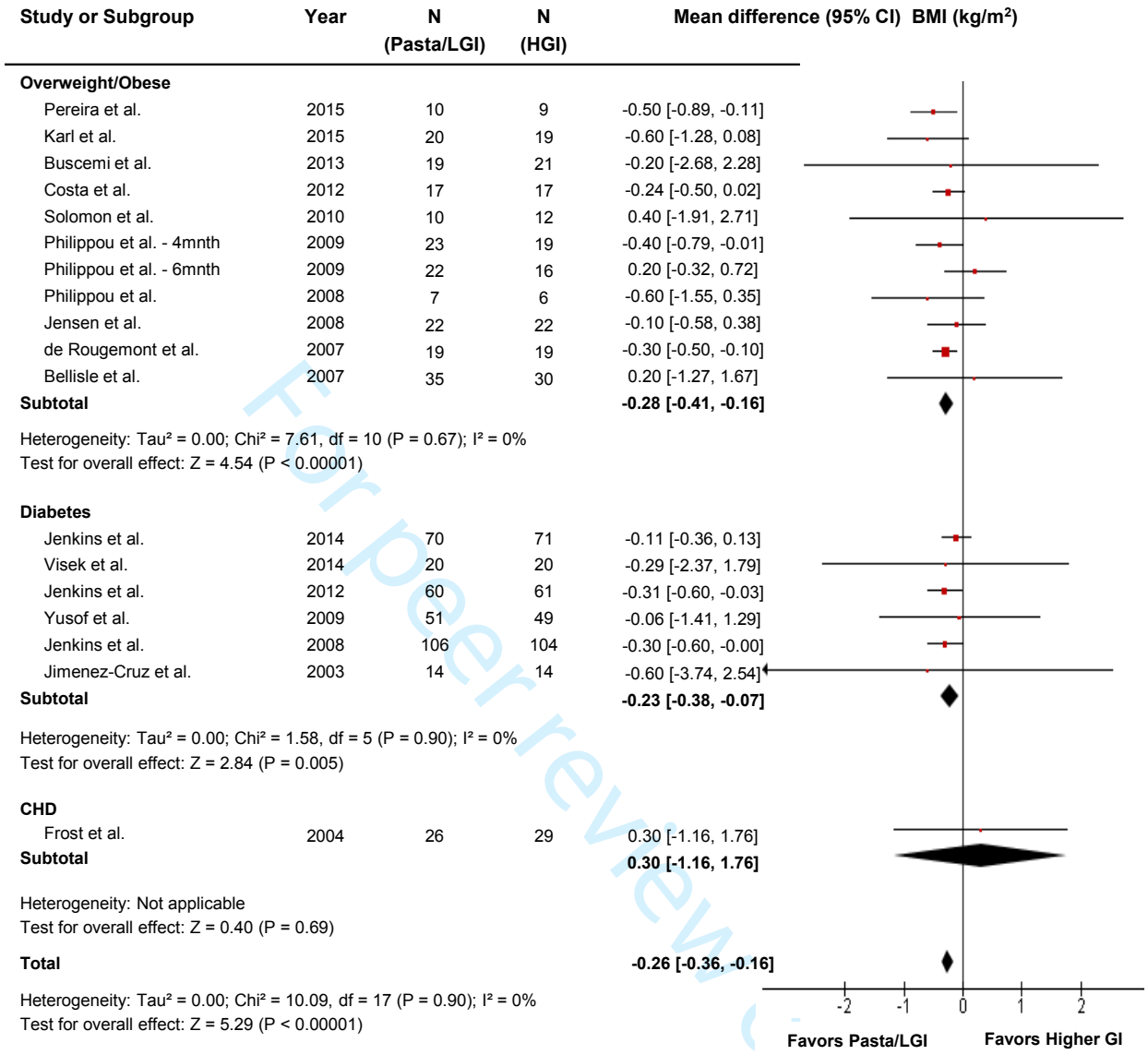


Supplemental Figure S1: Cochrane risk of bias summary for all included trials

Summary of risk of bias ratings for each individual study included in the meta-analysis.



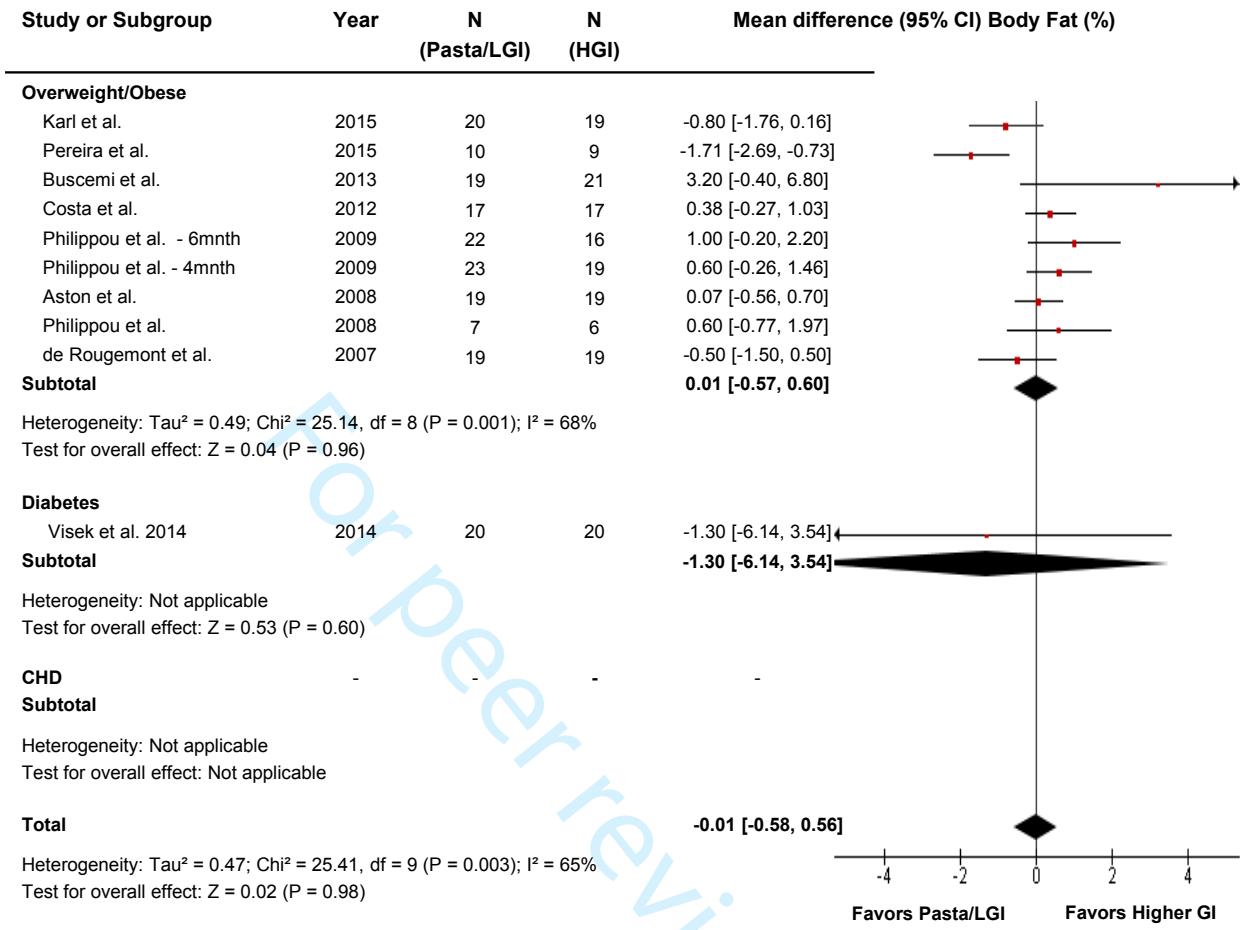
Supplemental Figure S2: Risk of bias proportion graph for all included trials



Supplemental Figure S3: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on BMI (kg/m^2) (n= 1038)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.

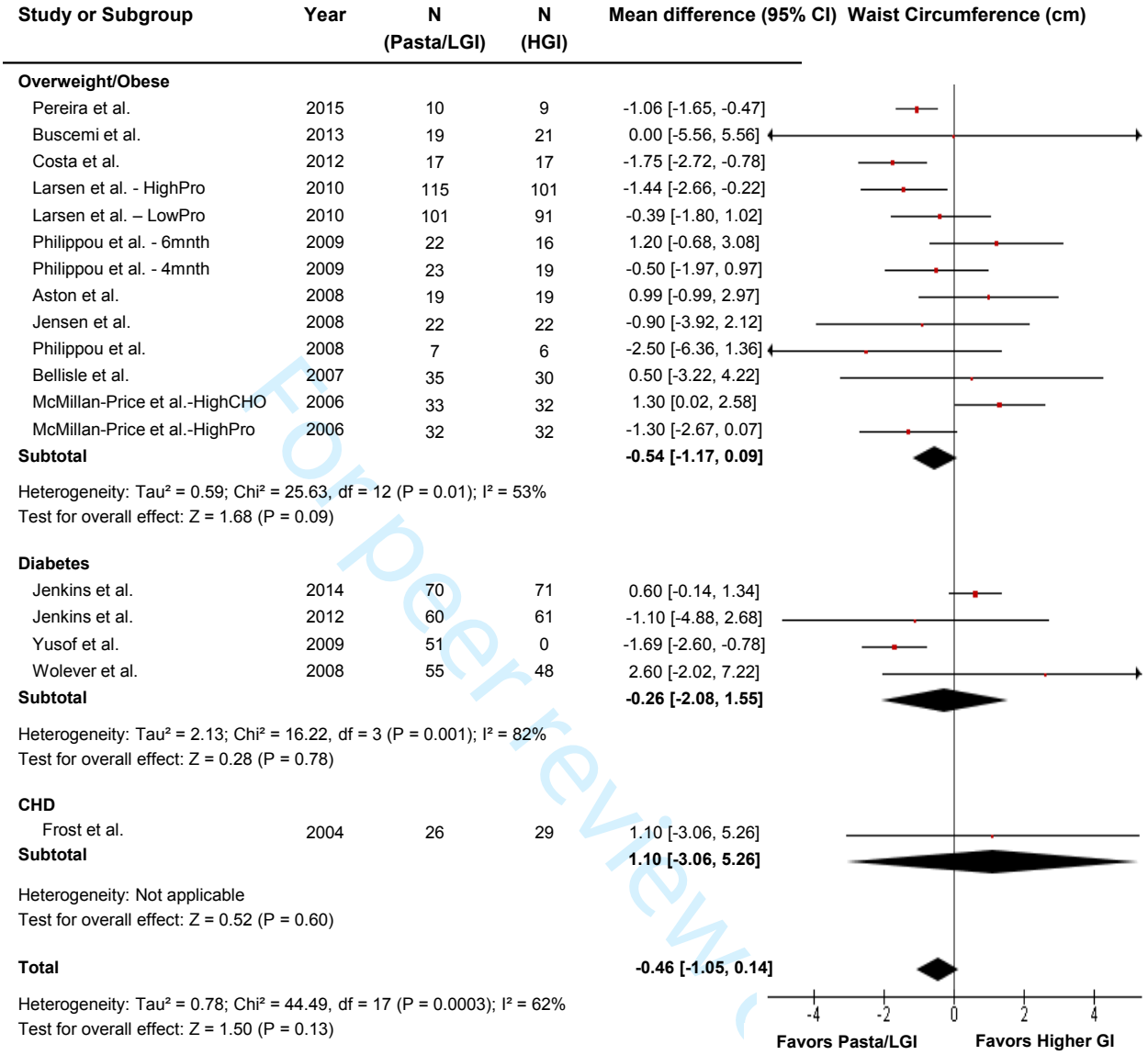
BMI, body mass index; CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet



Supplemental Figure S4: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body fat (%) (n= 285)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

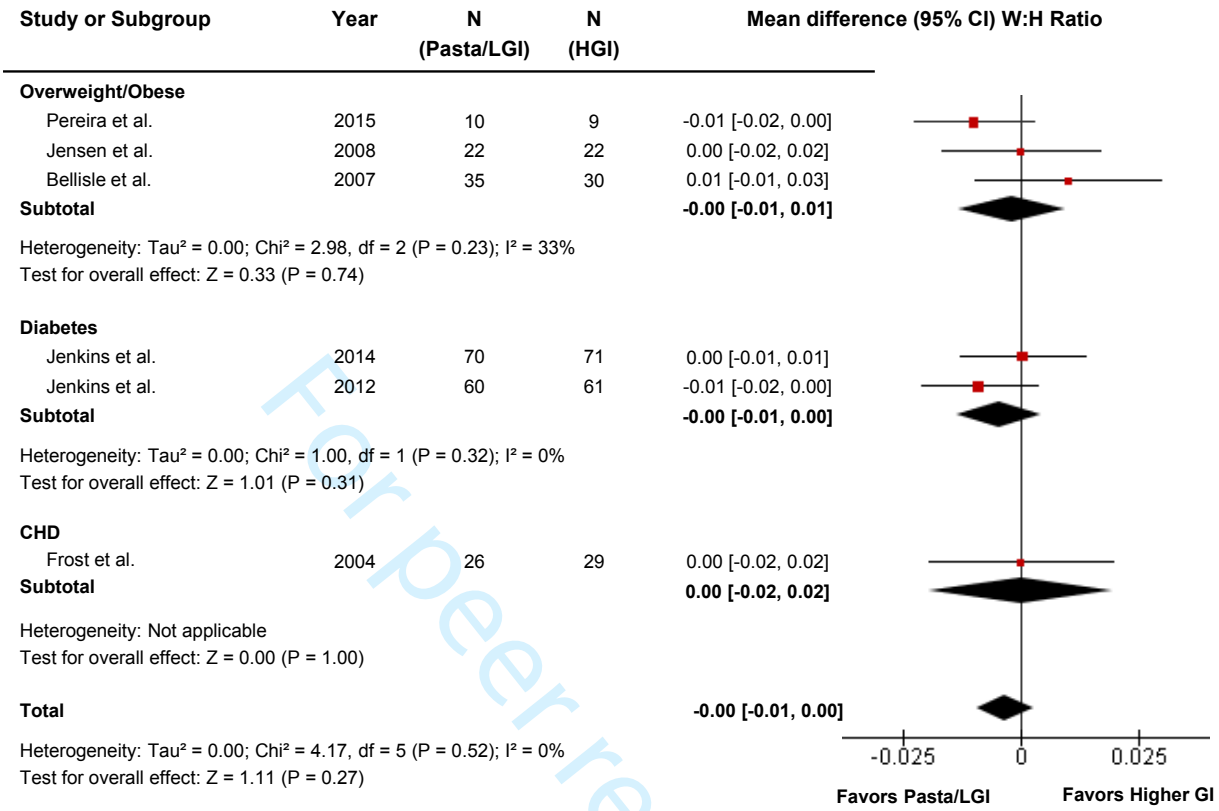
CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet



Supplemental Figure S5: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on waist circumference (cm) (n = 1380)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

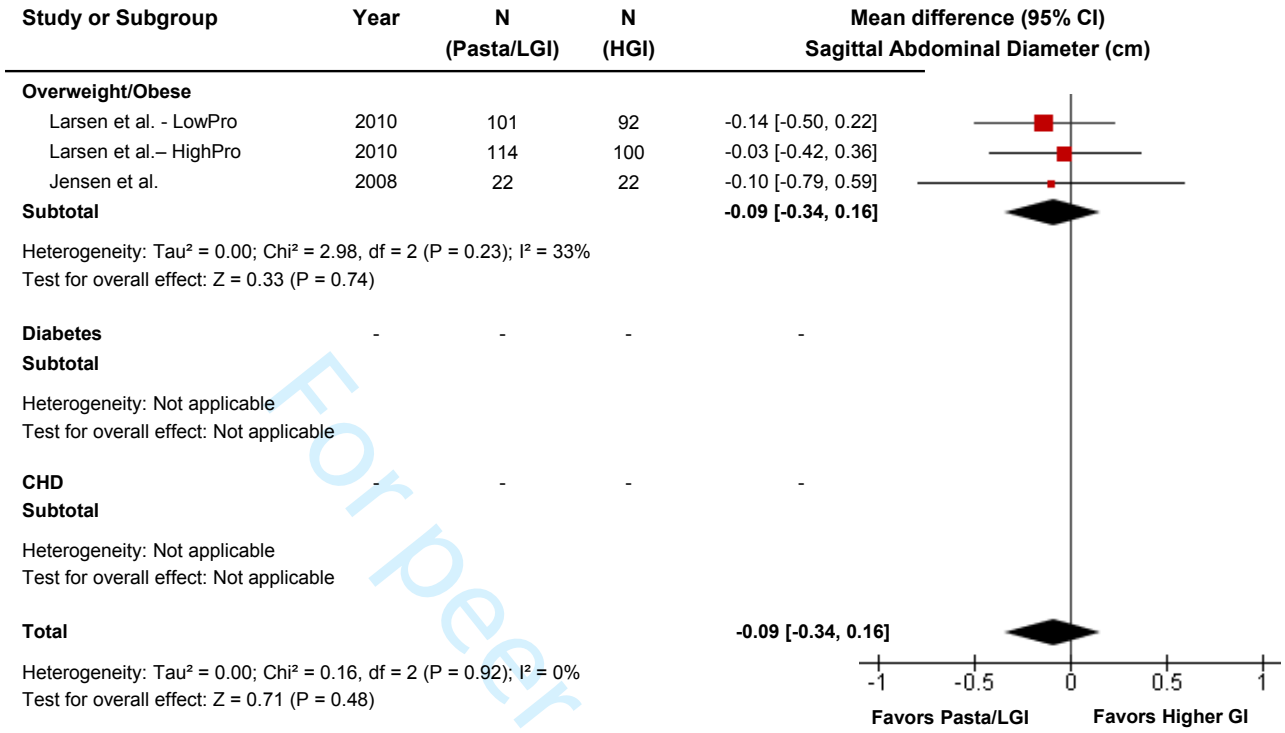
CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; Pro, protein



Supplemental Figure S6: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on waist-to-hip ratio (n = 445)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

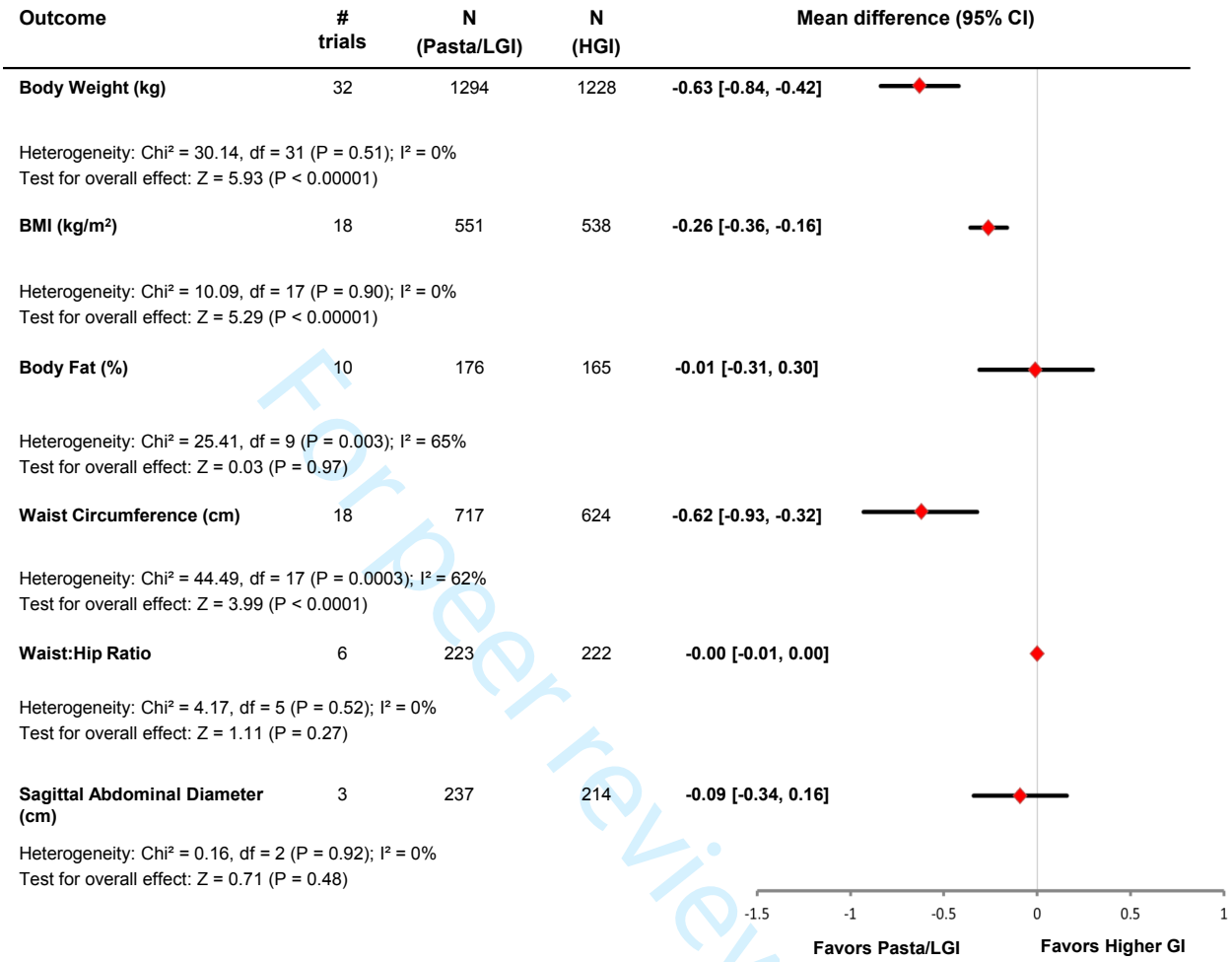
CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet



Supplemental Figure S7: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on sagittal abdominal diameter (cm) (n = 478)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

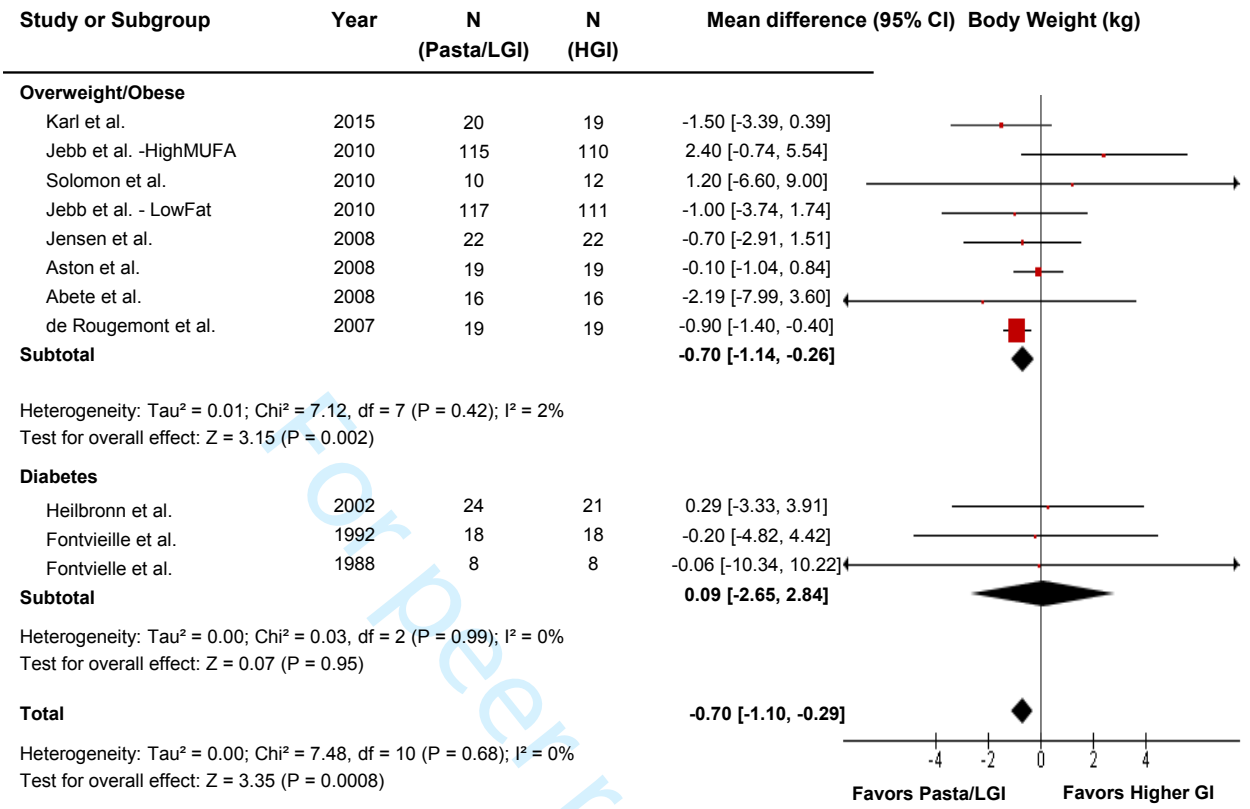
CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; Pro, protein



Supplemental Figure S8: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg) using fixed effects models (n = 2448)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance fixed effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

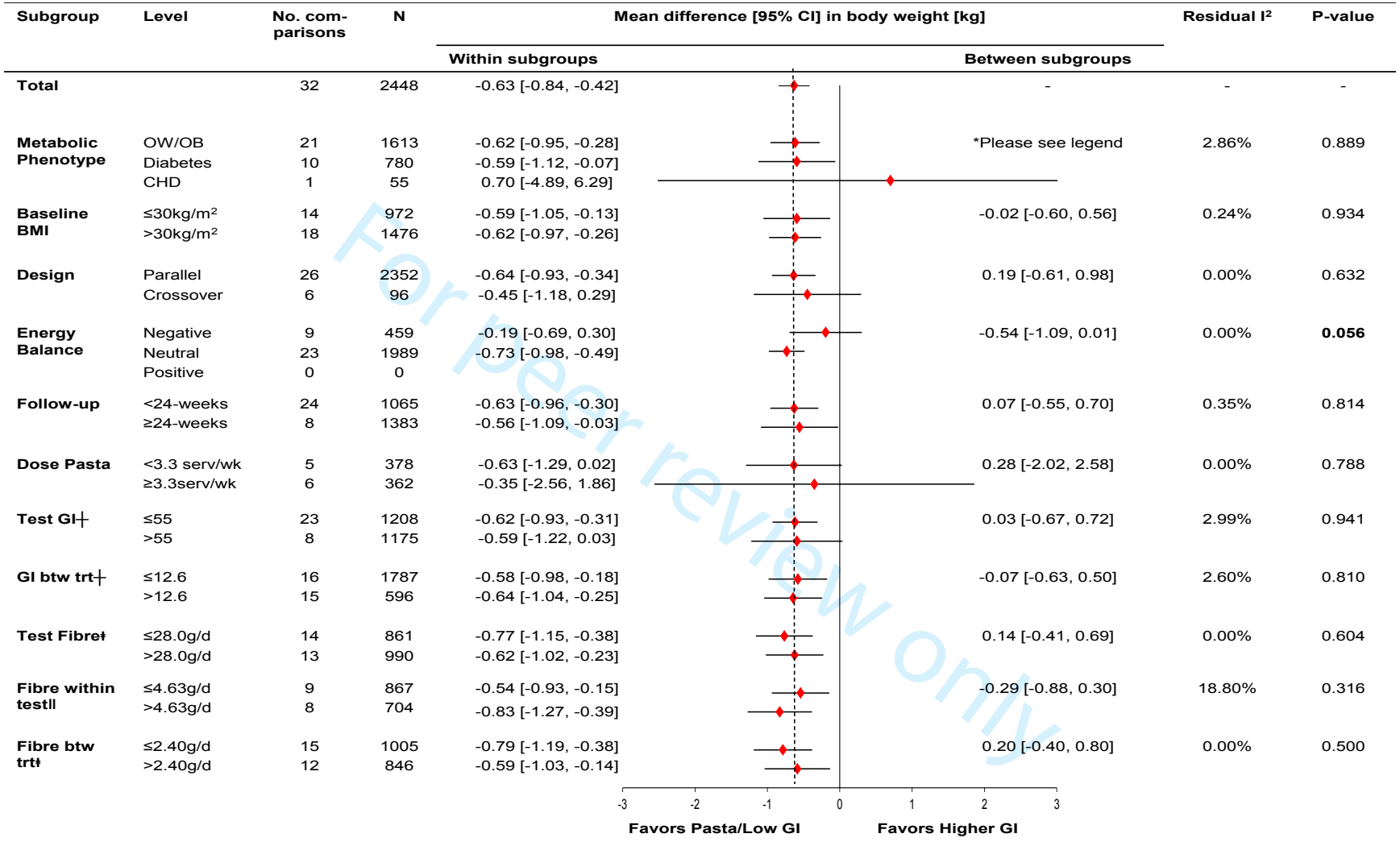
CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids; Pro, protein.



Supplemental Figure S9: Forest plot of randomized controlled trials which contain data for approximating pasta intake which are investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg) (n = 740)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids

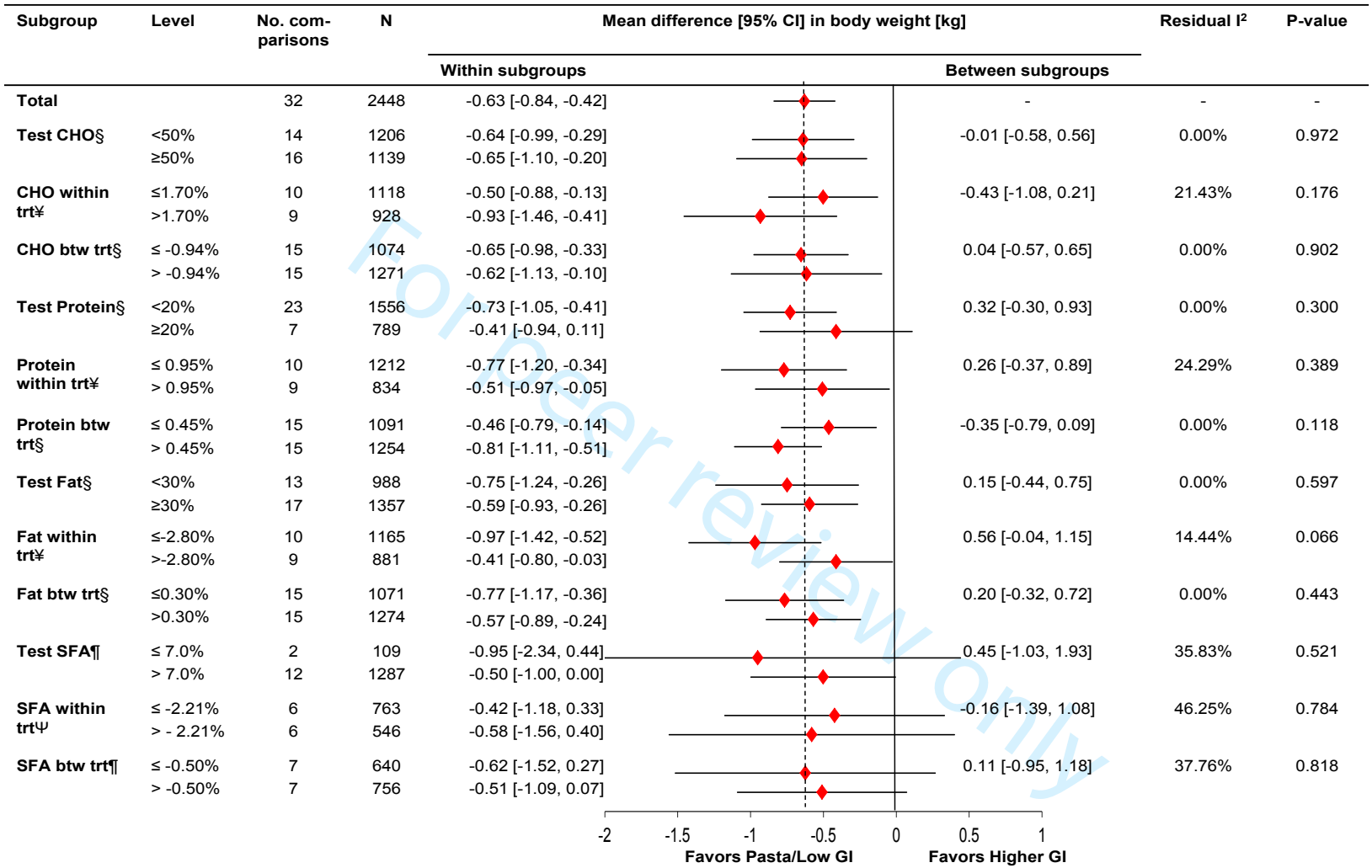


Supplemental Figure S10: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body weight (kg) (n = 2448)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body weight. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes.

BMI, body mass index; btw, between; CHD, coronary heart disease; CI, confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; serv, serving; trt, treatment; wk, week.

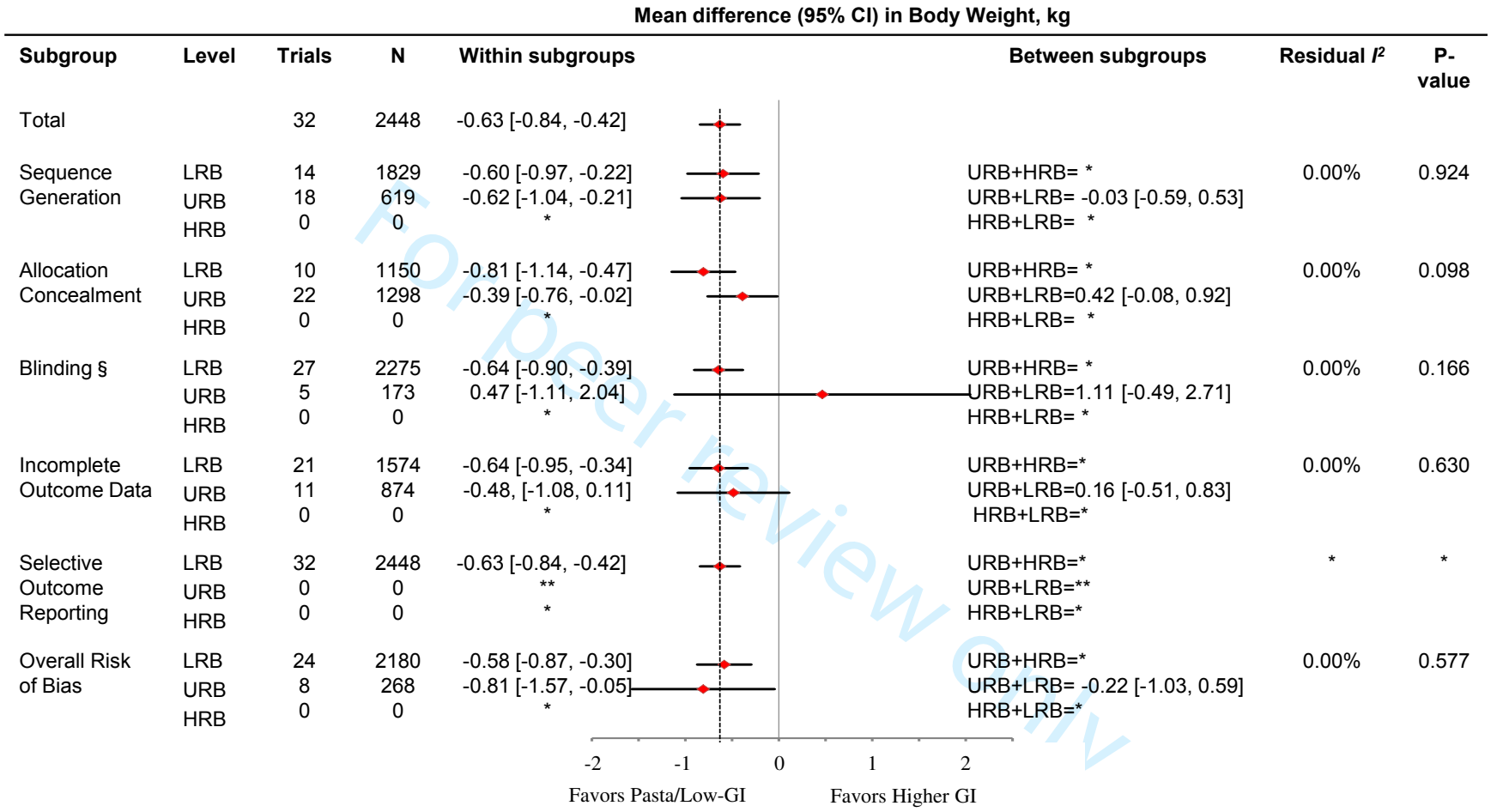
*Pairwise between-subgroup mean differences (95% CIs) for Metabolic Phenotype were as follows: 0.02kg (-0.08, 0.65) (1 vs. 2) to 1.32kg (-4.28, 6.91) (1 vs. 3) to -1.29kg (-6.91, 4.32) (2 vs. 3).
┼ data available on 31 studies
‖ data available on 17 studies
ǂ data available on 27 studies



Supplemental Figure S11: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body weight (kg) continued (n = 2448)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body weight. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes.

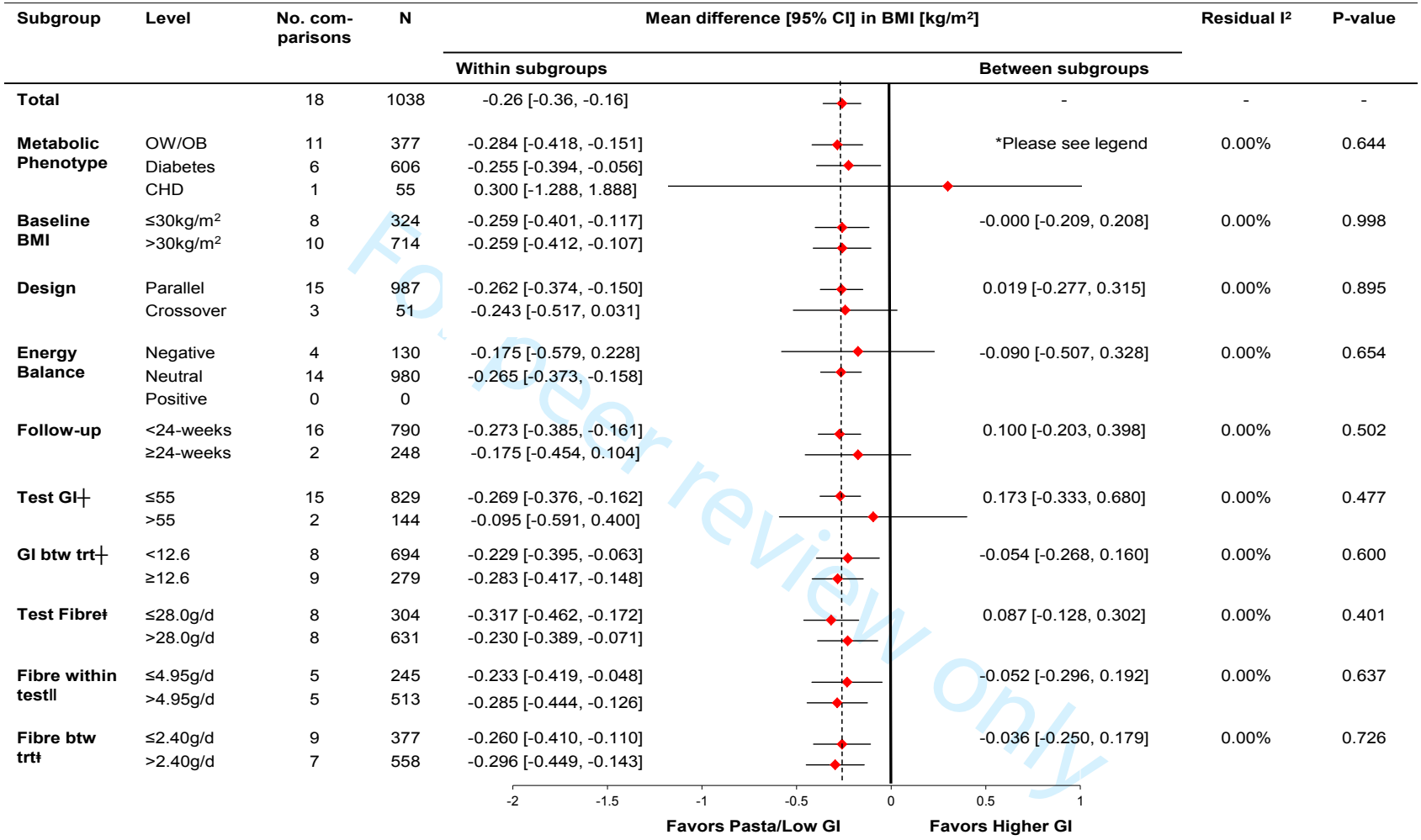
BMI, body mass index; btw, between; CI, confidence interval; GI, glycemic index (glucose scale); trt, treatment.
§ data available on 30 studies
¥ data available on 19 studies
¶ data available on 14 studies
Ψ data available on 12 studies



Supplemental Figure S12: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on body weight (kg) (n = 2448)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body weight. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

LRB=Low Risk of Bias, URB= Unclear Risk of Bias, HRB=High Risk of Bias. *Within and between subgroup analysis could not be performed against HRB since no values for HRB subgroup. ** no values for URB subgroup. § Blinding of Participants, Personnel, and Outcome Assessors.



Supplemental Figure S13: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body mass index, BMI (kg/m^2) (n = 1038)

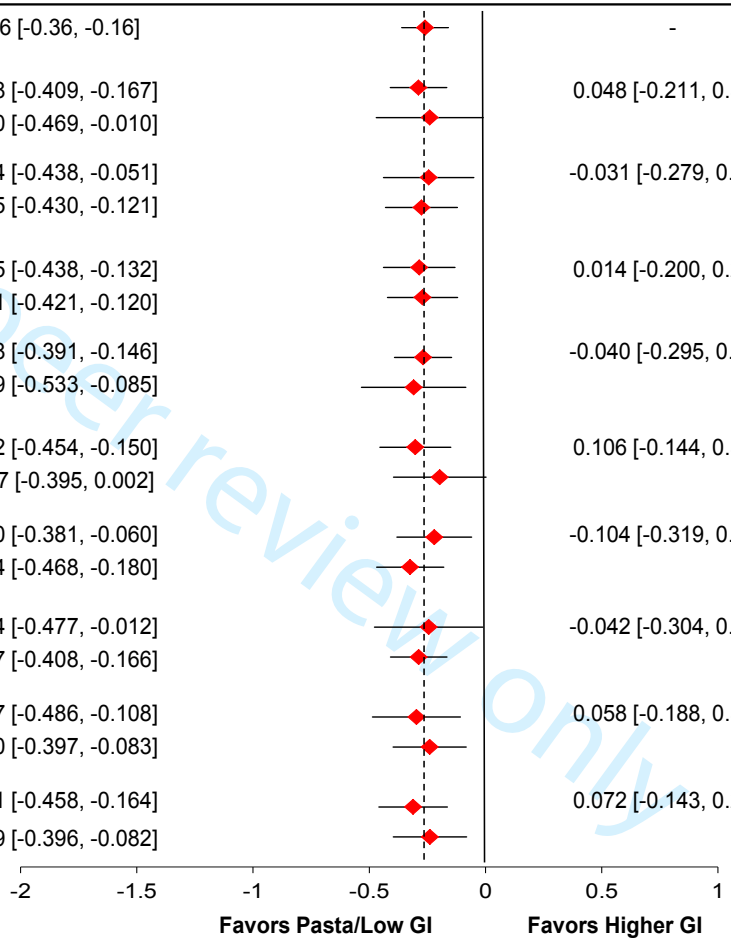
The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on BMI. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study

heterogeneity unexplained by the variability in response because of the specific between studies factor. P < 0.05 indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for dose (n=3 trials), therefore analyses were not performed.

*Pairwise between-subgroup mean differences (95% CIs) for Metabolic Phenotype were as follows: 0.059kg/m^2 (-0.156, 0.274) (1 vs. 2) to 0.584kg/m^2 (-1.009, 2.178) (1 vs. 3) to -0.525kg/m^2 (-2.122, 1.072) (2 vs. 3).
┼ data available on 17 studies
ǂ data available on 16 studies
‖ data available on 10 studies

BMI, body mass index; btw, between; CI, confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; trt, treatment.

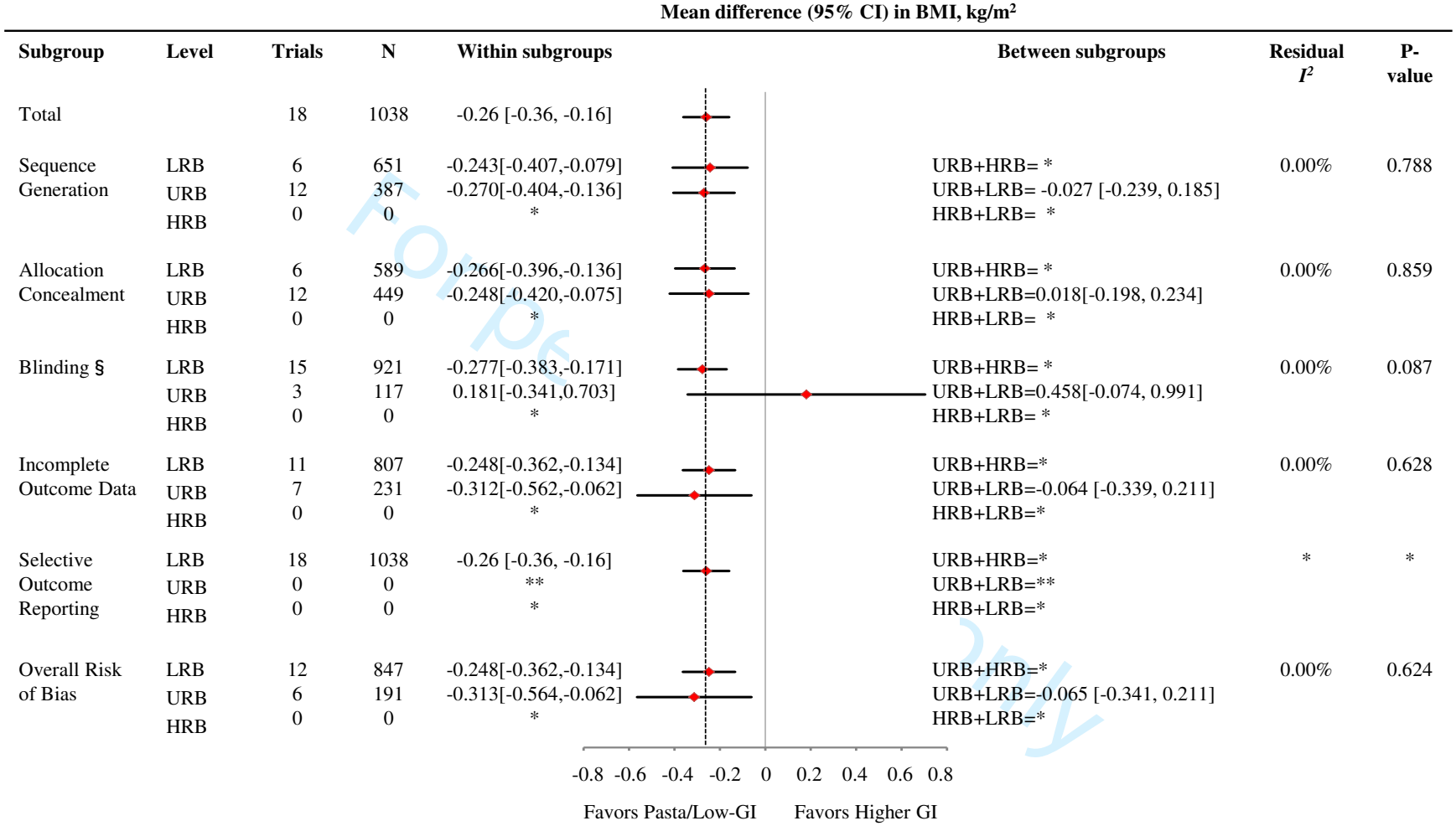
Subgroup	Level	No. comparisons	N	Mean difference [95% CI] in BMI [kg/m²] Within subgroups	Between subgroups	Residual I²	P-value
Total		18	1038	-0.26 [-0.36, -0.16]	-	-	-
Test CHO§	< 50%	9	659	-0.288 [-0.409, -0.167]	0.048 [-0.211, 0.308]	0.00%	0.696
	≥ 50%	7	276	-0.240 [-0.469, -0.010]			
CHO within trt¥	≤ 1.35%	5	436	-0.244 [-0.438, -0.051]	-0.031 [-0.279, 0.217]	0.00%	0.781
	> 1.35%	5	322	-0.275 [-0.430, -0.121]			
CHO btw trt§	≤ -1.75%	8	657	-0.285 [-0.438, -0.132]	0.014 [-0.200, 0.229]	0.00%	0.888
	> -1.75%	8	278	-0.271 [-0.421, -0.120]			
Test Protein§	< 20%	13	590	-0.268 [-0.391, -0.146]	-0.040 [-0.295, 0.215]	0.00%	0.741
	≥ 20%	3	345	-0.309 [-0.533, -0.085]			
Protein within trt¥	≤ 1.04%	5	339	-0.302 [-0.454, -0.150]	0.106 [-0.144, 0.356]	0.00%	0.358
	> 1.04%	5	419	-0.197 [-0.395, 0.002]			
Protein btw trt§	≤ 0.45%	8	373	-0.220 [-0.381, -0.060]	-0.104 [-0.319, 0.112]	0.00%	0.319
	> 0.45%	8	562	-0.324 [-0.468, -0.180]			
Test Fat§	< 30%	6	176	-0.244 [-0.477, -0.012]	-0.042 [-0.304, 0.220]	0.00%	0.734
	≥ 30%	10	759	-0.287 [-0.408, -0.166]			
Fat within trt¥	≤ -2.60%	5	303	-0.297 [-0.486, -0.108]	0.058 [-0.188, 0.303]	0.00%	0.604
	> -2.60%	5	455	-0.240 [-0.397, -0.083]			
Fat btw trt§	≤ 0.95%	8	253	-0.311 [-0.458, -0.164]	0.072 [-0.143, 0.287]	0.00%	0.483
	> 0.95%	8	682	-0.239 [-0.396, -0.082]			



Supplemental Figure S14: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body mass index, BMI (kg/m²) continued (n = 1038)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on BMI. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for test saturated fat (n=7 trials), saturated fat within treatment (n=6 trials) and saturated fat between treatments (n=7 trials), therefore analyses were not performed.
§ data available on 16 studies
¥ data available on 10 studies

BMI, body mass index; btw, between; CHO, carbohydrate; CI = confidence interval; GI, glycemic index (glucose scale); trt, treatment.



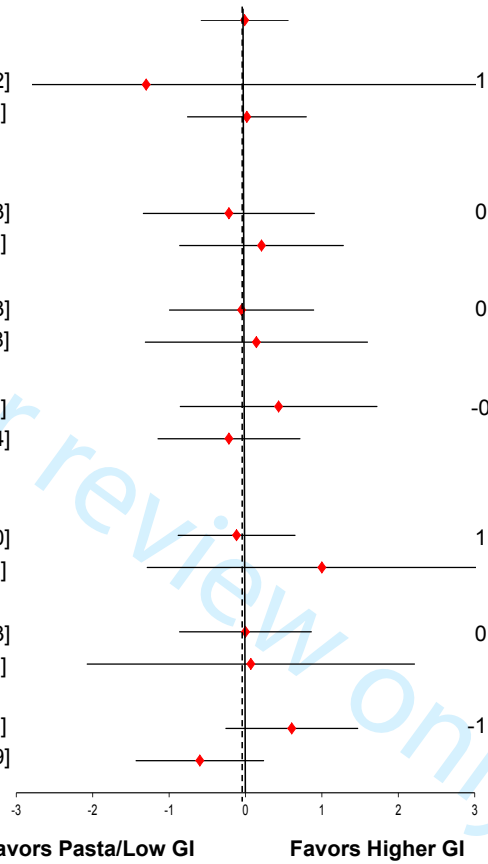
Supplemental Figure S15: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on body mass index, BMI (kg/m²) (n = 1038)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on BMI. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

*Within and between subgroup analyses could not be performed against HRB since no values for HRB subgroup.
** no values for URB subgroup.
§ Blinding of Participants, Personnel, and Outcome Assessors.

BMI, body mass index; CI, confidence interval; HRB, High Risk of Bias; GI, glycemic index; LRB, Low Risk of Bias; URB, Unclear Risk of Bias.

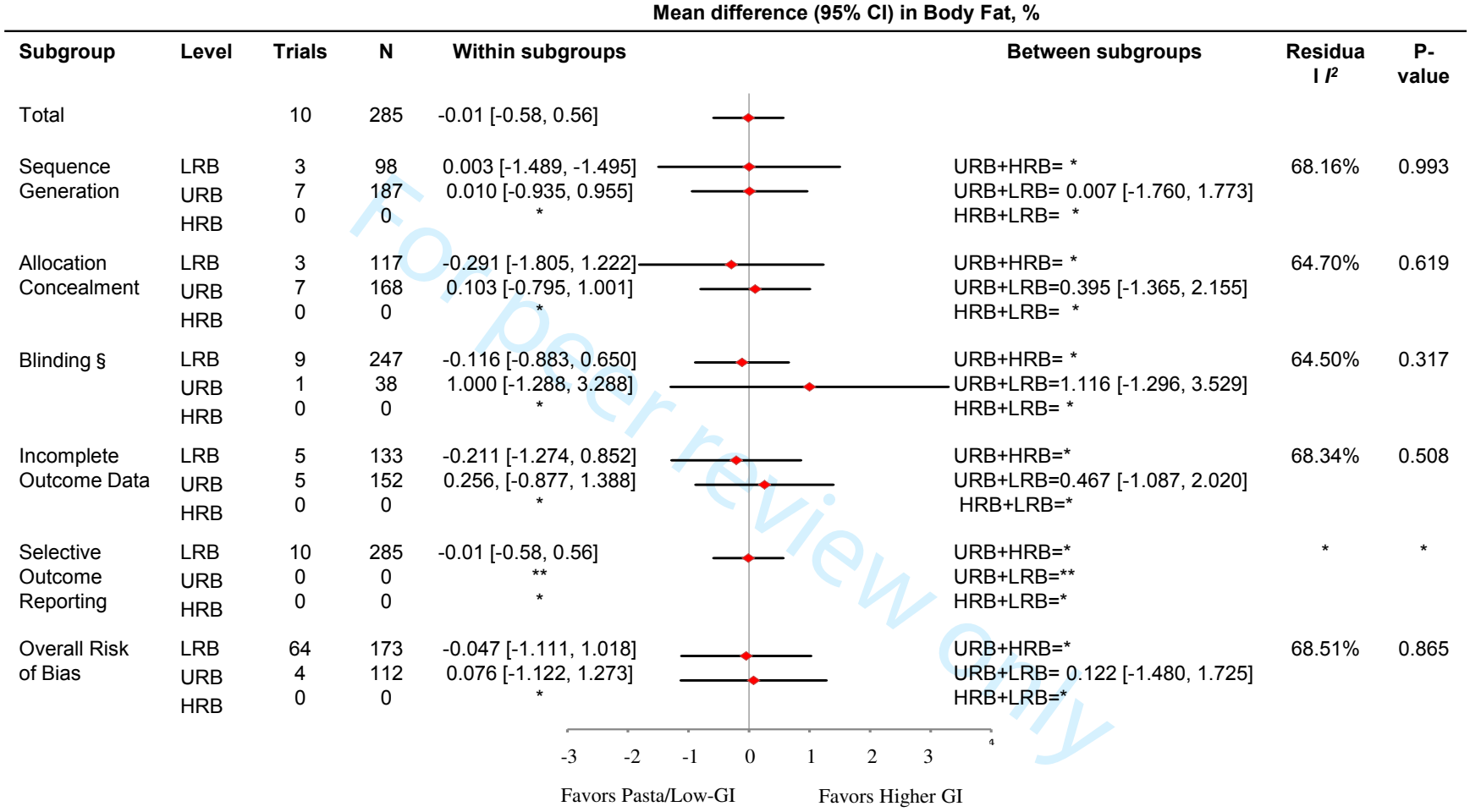
Subgroup	Level	No. comparisons	N	Mean difference [95% CI] in body fat [%] Within subgroups	Between subgroups	Residual I^2	P-value
Total		10	285	-0.01 [-0.58, 0.56]	-	-	-
Metabolic Phenotype	OW/OB	9	265	-1.300 [-7.782, 5.182]	1.319 [-5.210, 7.847]	68.17%	0.654
	Diabetes	1	20	0.019 [-0.760, 0.797]			
	CHD	0	0				
Baseline BMI	≤30kg/m^2	4	112	-0.217 [-1.338, 0.903]	0.427 [-1.125, 1.978]	67.72%	0.544
	>30kg/m^2	6	173	0.210 [-0.863, 1.282]			
Design	Parallel	7	229	-0.051 [-0.996, 0.893]	0.194 [-1.540, 1.929]	66.40%	0.803
	Crossover	3	56	0.143 [-1.312, 1.598]			
Energy Balance	Negative	4	130	0.434 [-0.856, 1.723]	-0.650 [-2.241, 0.940]	68.11%	0.373
	Neutral	6	155	-0.216 [-1.147, 0.714]			
	Positive						
Follow-up	<24-weeks	9	247	-0.116 [-0.883, 0.650]	1.116 [-1.300, 3.529]	64.50%	0.317
	≥24-weeks	1	38	1.000 [-1.288, 3.288]			
Test GI	≤55	9	266	-0.000 [-0.864, 0.863]	0.070 [-2.241, 2.382]	68.43%	0.946
	>55	1	19	0.070 [-2.074, 2.214]			
GI btw trt	≤14.1	5	152	0.606 [-0.258, 1.471]	-1.204 [-2.407, -0.008]	55.49%	0.050
	>14.1	5	153	-0.597 [-1.434, 0.239]			



Supplemental Figure S16: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body fat (%) (n = 285)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body fat. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for dose (n=3), test carbohydrate, protein and fat (n=9), carbohydrate, protein and fat within treatment (n=4), carbohydrate, protein and fat between treatment (n=9), test fibre (n=8), fibre within treatment (n=4), fibre between treatment (n=8), test saturated fat (n=3 trials), saturated fat within treatment (n=2 trials) and saturated fat between treatments (n=3 trials), therefore analyses were not performed.

BMI, body mass index; btw, between; CHO, carbohydrate; CI = confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; trt, treatment.

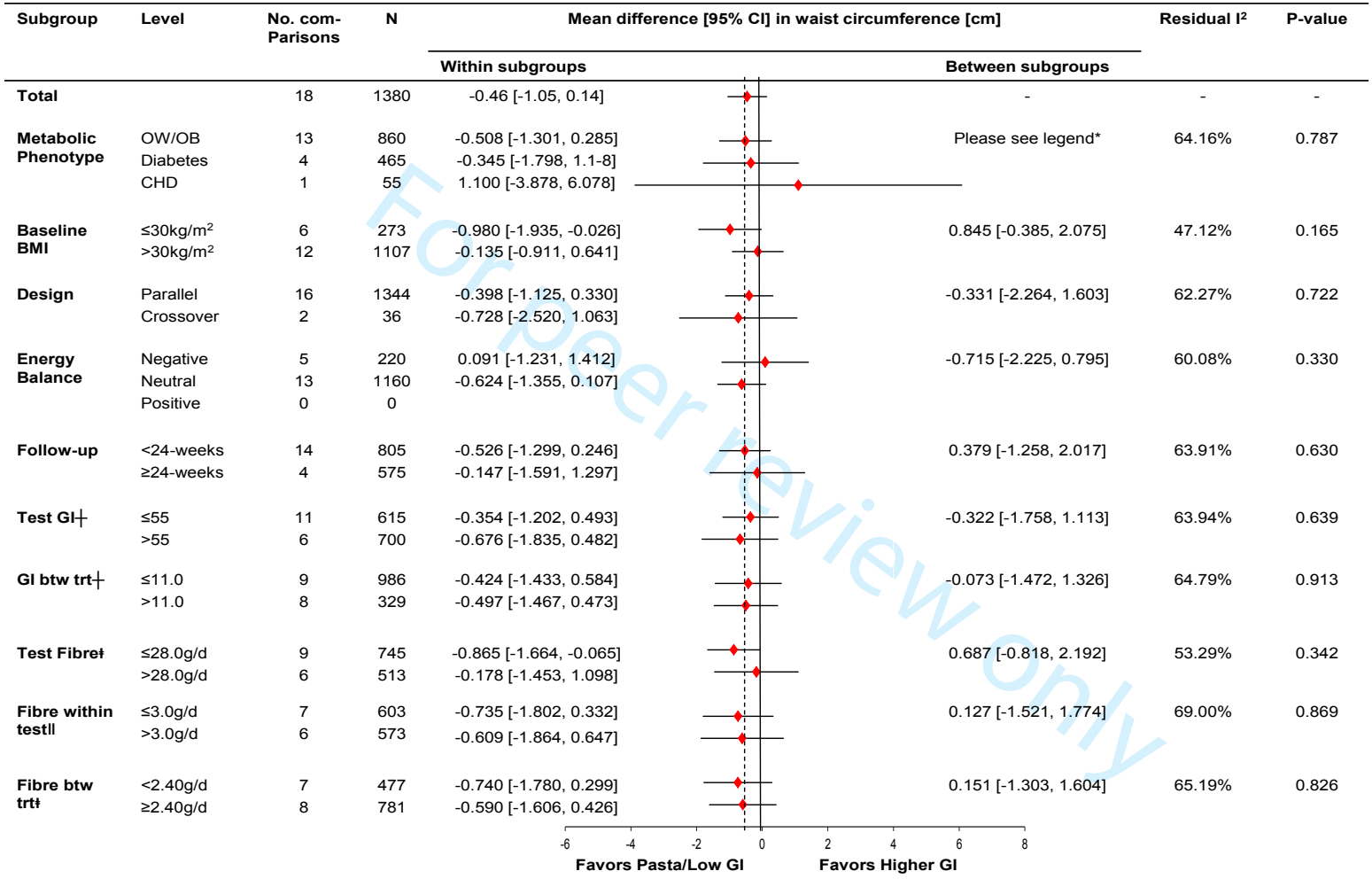


Supplemental Figure S17: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on body fat (%) (n = 285)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body fat. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

*Within and between subgroup analyses could not be performed against HRB since no values for HRB subgroup.
** no values for URB subgroup.
§ Blinding of Participants, Personnel, and Outcome Assessors.

CI, confidence interval; HRB, High Risk of Bias; GI, glycemic index; LRB, Low Risk of Bias; URB, Unclear Risk of Bias

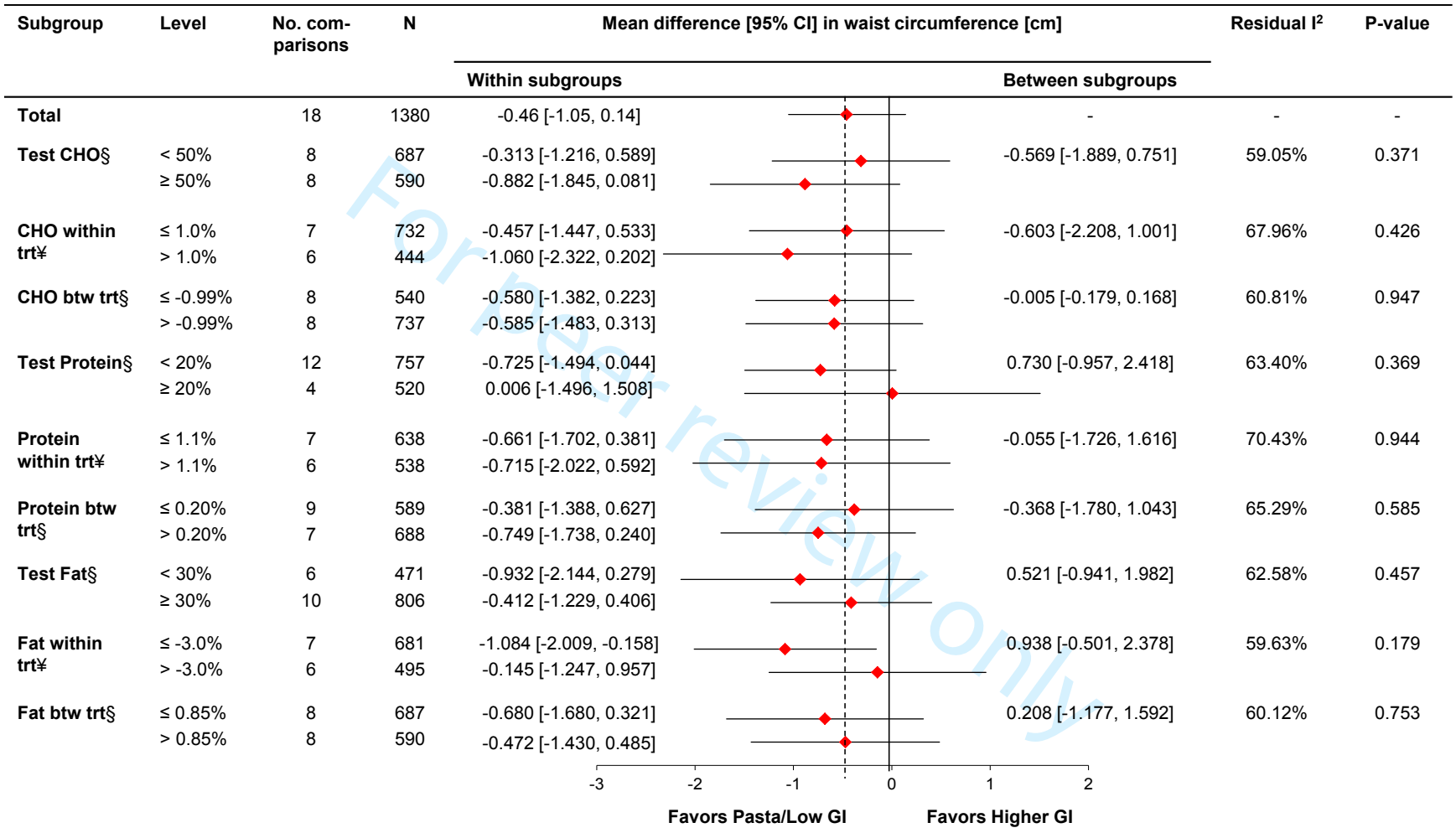


Supplemental Figure S18: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on waist circumference (cm) (n = 1380)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for dose (n=2 trials), therefore analyses were not performed.

*Pairwise between-subgroup mean differences (95% CIs) for Metabolic Phenotype were as follows: 0.163cm (-1.492, 1.818) (1 vs. 2) to 1.608cm (-3.432, 6.648) (1 vs. 3) to -1.445cm (-6.630, 3.740) (2 vs. 3).
┼ data available on 17 studies
ł data available on 15 studies

BMI, body mass index; btw, between; CI = confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; trt, treatment.

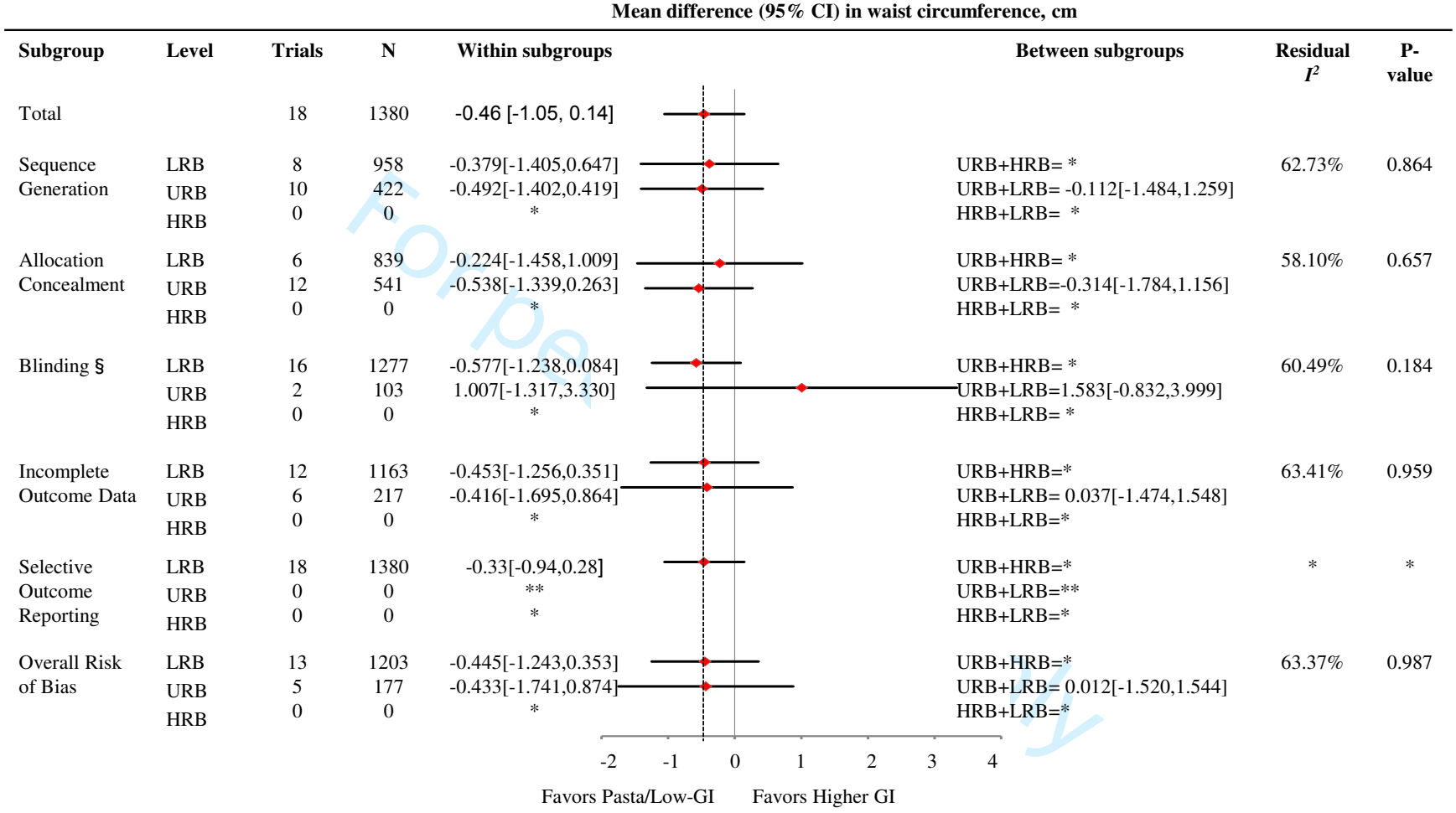


Supplemental Figure S19: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on waist circumference (cm) continued (n = 1380)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for test saturated fat (n=8 trials), saturated fat within treatment (n=7 trials) and saturated fat between treatments (n=8 trials), therefore analyses were not performed.

§ data available on 16 studies
¥ data available on 13 studies

BMI, body mass index; btw, between; CHO, carbohydrate; CI = confidence interval; GI, glycemic index (glucose scale); trt, treatment.



Supplemental Figure S20: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on waist circumference (cm) (n = 1380)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

*Within and between subgroup analyses could not be performed against HRB since no values for HRB subgroup.
** no values for URB subgroup.
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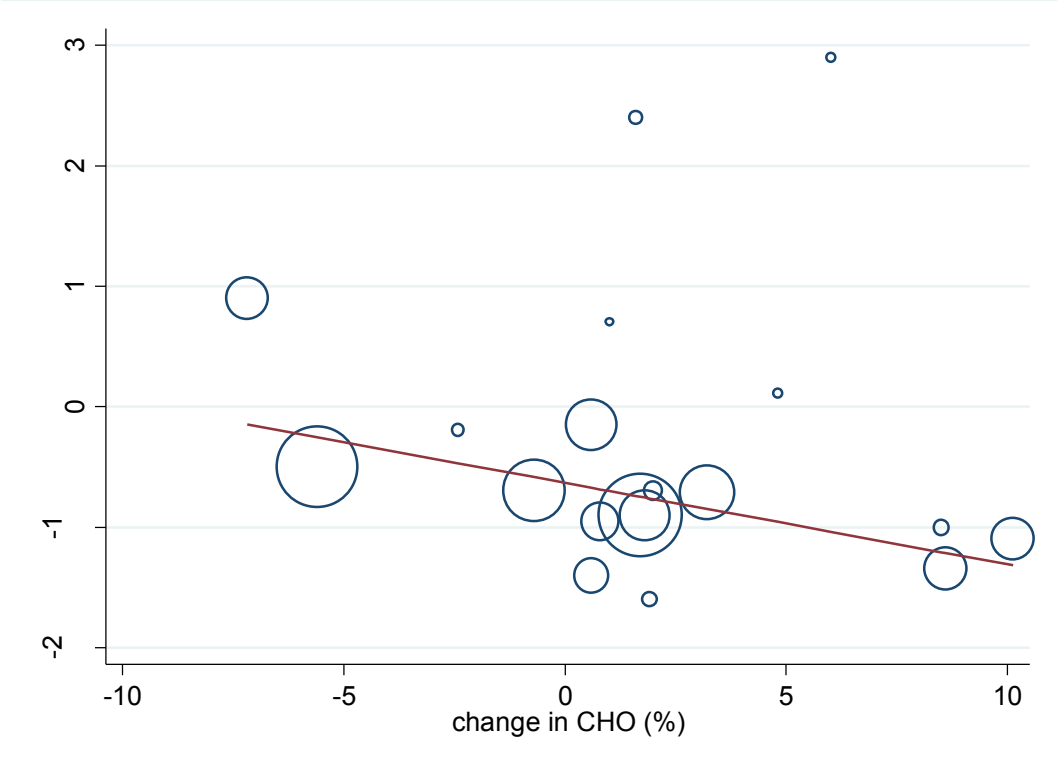
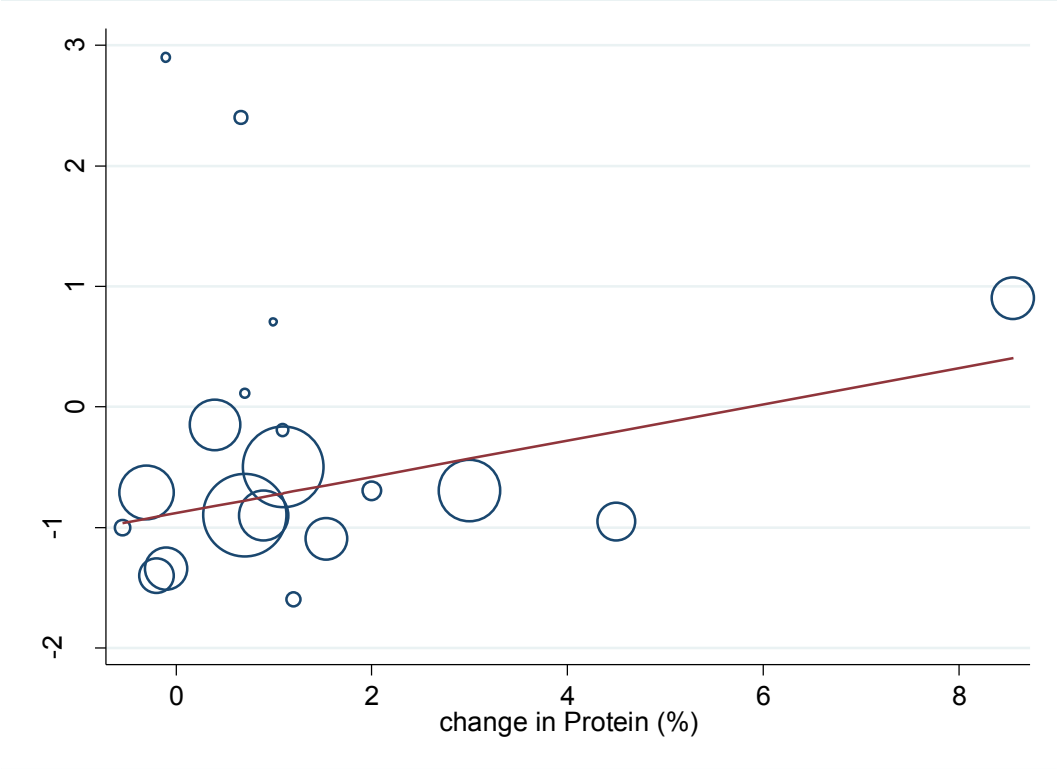
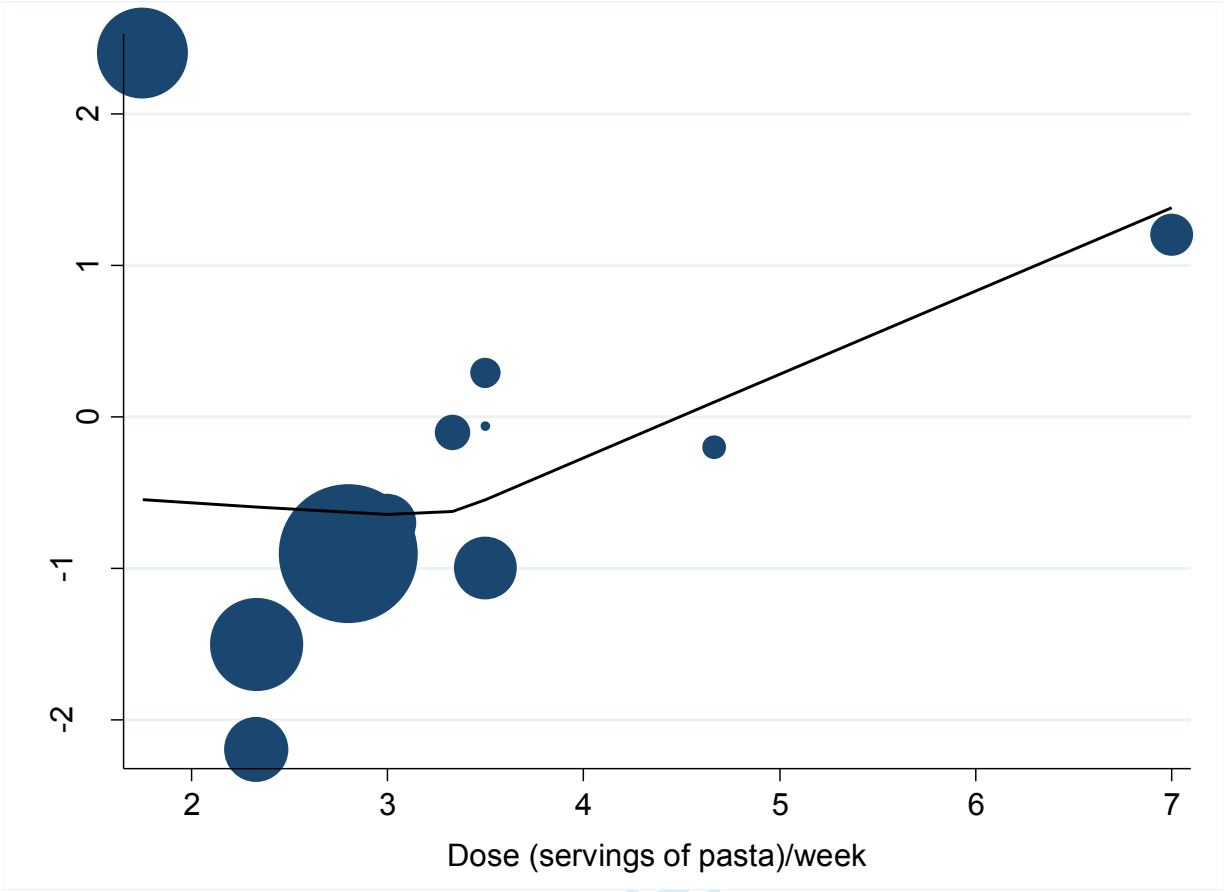
CI, confidence interval; HRB, High Risk of Bias; GI, glycemic index; LRB, Low Risk of Bias; URB, Unclear Risk of Bias



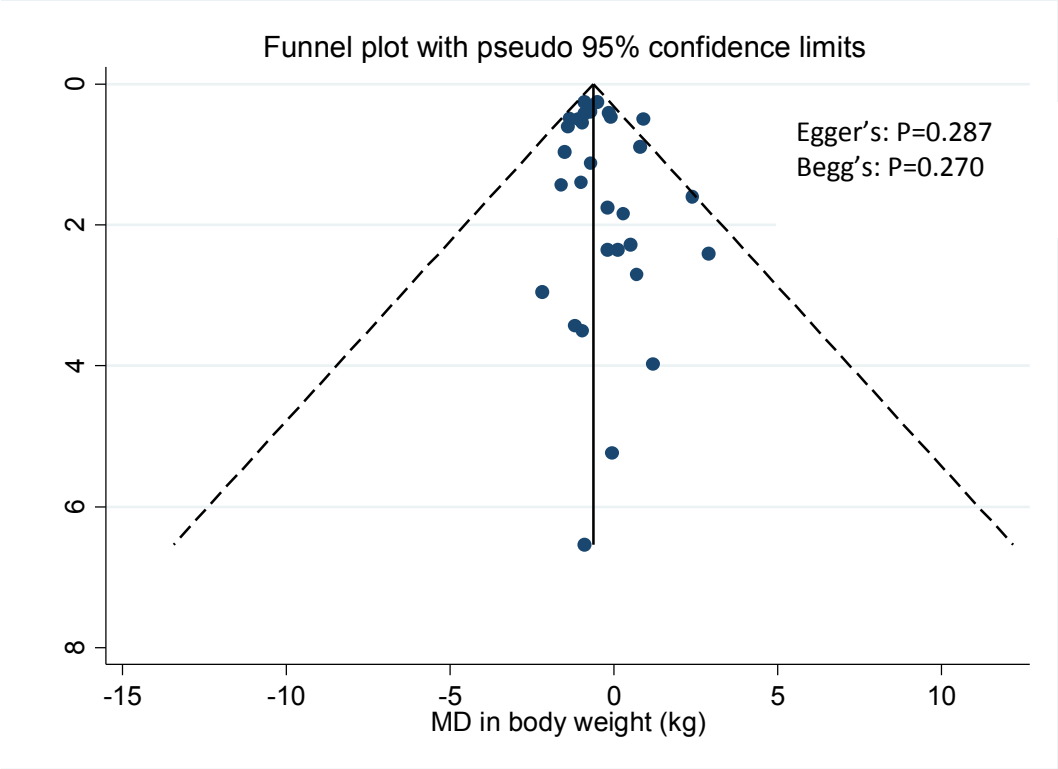
Supplemental Figure S21: Continuous meta-regression of change in carbohydrate intake in the low-GI dietary pattern intervention arms with change in body weight (n=19)
CHO, carbohydrate; MD, mean difference



Supplemental Figure S22: Continuous meta-regression of change in protein intake in the low-GI dietary pattern intervention arms with change in body weight (n=19)
MD, mean difference

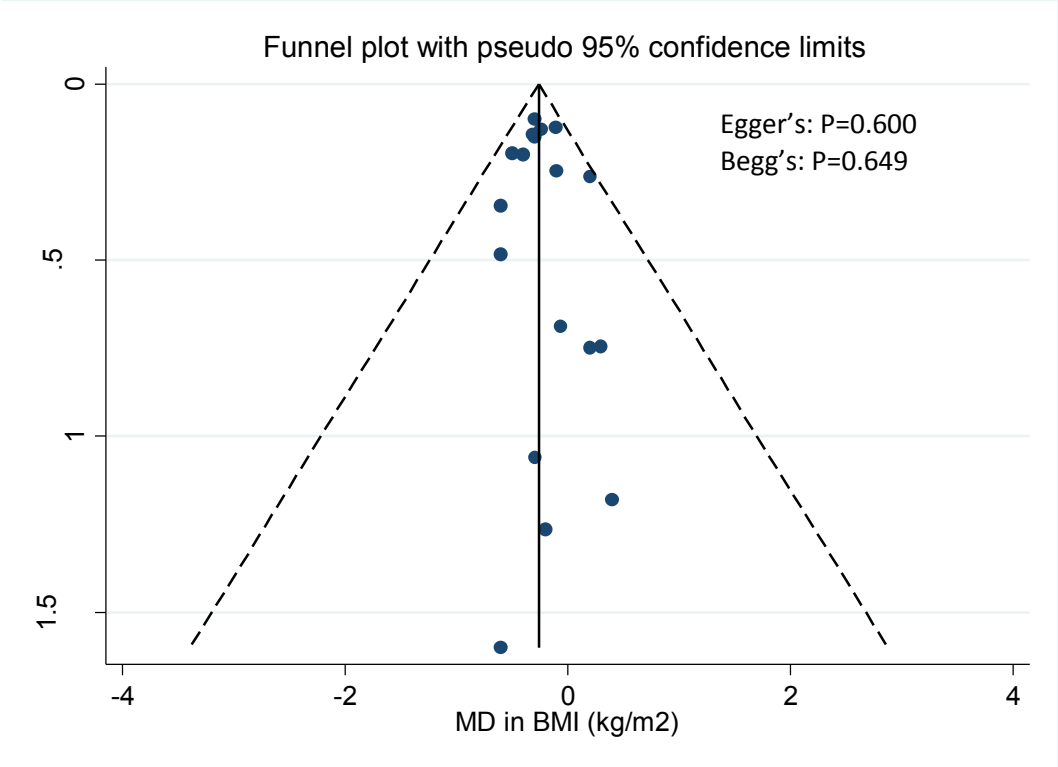


Supplemental Figure S23: Pasta intake dose-response analyses by spline curve modeling (MKSPLINE procedure)



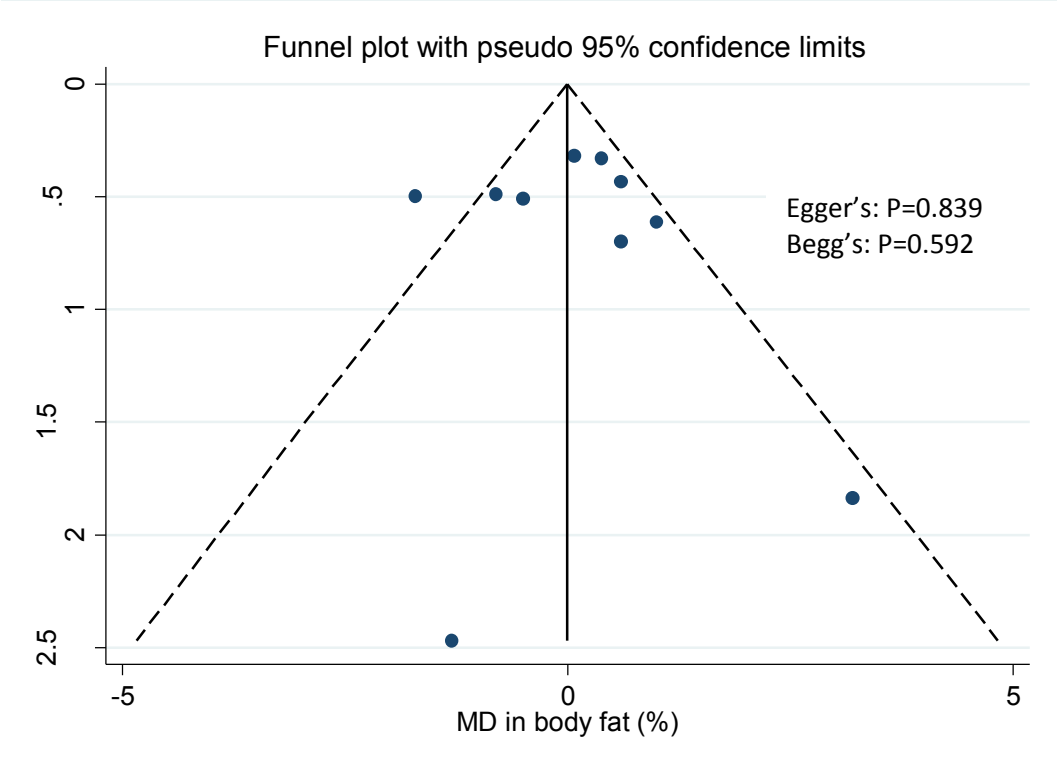
Supplemental Figure S24: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on body weight (kg)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.



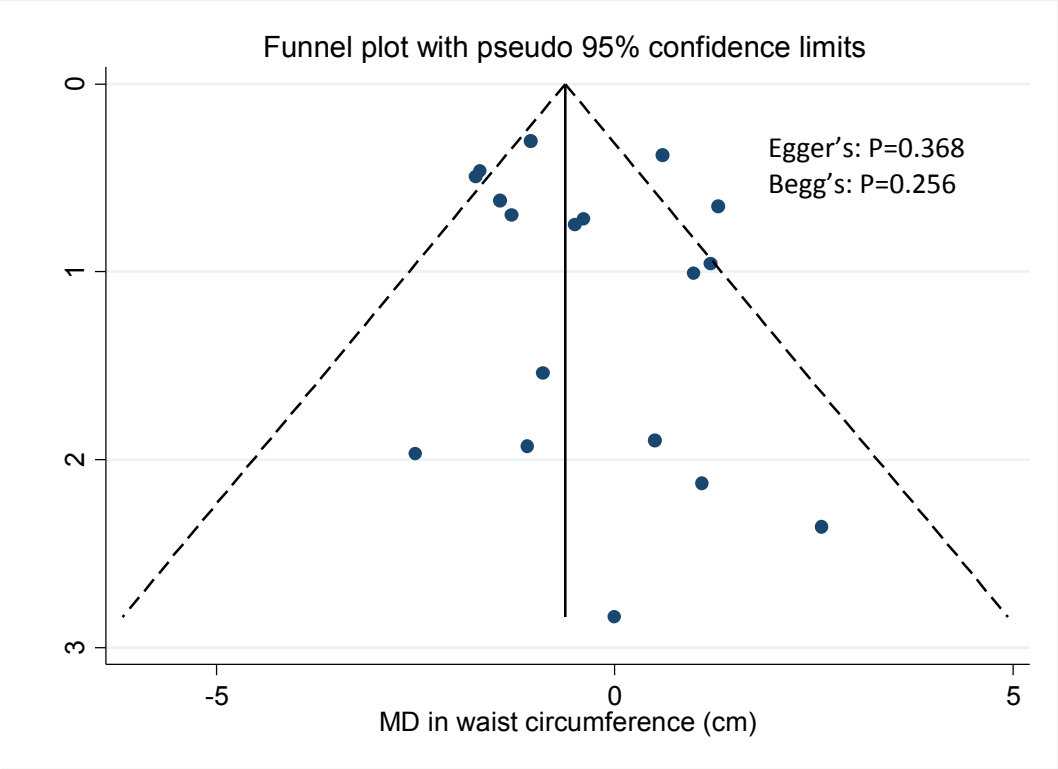
Supplemental Figure S25: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on BMI (kg/m^2)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.



Supplemental Figure S26: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on body fat (%)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.



Supplemental Figure S27: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on waist circumference (cm)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.

Supplemental Table S1: PRISMA Checklist

PRISMA 2009 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.	2-3
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.	6
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.	6-7, Supplemental Table S2
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.	6, Supplemental Table S1
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	Supplemental Table S1
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).	6-7, Figure 1
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.	7
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and	7, Supplemental

		simplifications made.	Table S2
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.	7
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	8
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.	8-10

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).	10
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	8-10
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.	11, 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.	11-12, Table 1, Supplemental Tables S2-S3
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).	12, 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.	12-13, Figures 2-3, Supplemental Figures S3-S7
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	12-13, Figures 2-3, Supplemental Figures S3-S7
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	12,16 Supplemental

			Table S1, S2, S12, S15, S17, S20
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	13-16, Supplemental Tables S4-S10, Supplemental Figures S8-S26
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).	17-20
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).	20-22
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	22-24
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.	27-28

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**.

BMJ Open

Effect of Pasta in the Context of Low Glycemic Index Dietary Patterns on Body Weight and Markers of Adiposity: A Systematic Review and Meta-analysis of Randomized Controlled Trials in Adults

Journal:	*BMJ Open*
Manuscript ID	bmjopen-2017-019438.R2
Article Type:	Research
Date Submitted by the Author:	26-Jan-2018
Complete List of Authors:	Chiavaroli, Laura; St. Michael's Hospital, Toronto 3D Knowledge Synthesis and Clinical Trials Unit; University of Toronto, Nutritional Sciences Kendall, Cyril; University of Toronto, Nutritional Sciences Braunstein, Catherine; University of Toronto, Nutritional Sciences; St. Michael's Hospital, Toronto 3D Knowledge Synthesis and Clinical Trials Unit Blanco Mejia, Sonia; Clinical Nutrition and Risk Factor Modification Centre, St. Michael's Hospital; Toronto 3D Knowledge Synthesis and Clinical Trials Unit, St. Michael's Hospital Leiter, Lawrence; St. Michael's Hospital, Endocrinology; University of Toronto, Nutritional Sciences Jenkins, David; St. Michael's Hospital, Clinical Nutrition and Risk Factor Modification Center; University of Toronto, Nutritional Sciences Sievenpiper, John; McMaster University, Department of Pathology and Molecular Medicine; St. Michael's Hospital, Clinical Nutrition and Risk Factor Modification Centre
Primary Subject Heading:	Nutrition and metabolism
Secondary Subject Heading:	Cardiovascular medicine
Keywords:	body weight, pasta, glycemic index, glycaemic index, systematic review and meta-analysis, weight loss

Effect of Pasta in the Context of Low Glycemic Index Dietary Patterns on Body Weight and Markers of Adiposity: A Systematic Review and Meta-analysis of Randomized Controlled Trials in Adults

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Word Count: 5,061

Tables: 1

Figures: 3

Online only supplementary material included

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ABSTRACT

Objective: Carbohydrate staples such as pasta have been implicated in the obesity epidemic. It is unclear whether pasta contributes to weight gain or like other low-glycemic index (GI) foods contributes to weight loss. We synthesized the evidence of the effect of pasta on measures of adiposity.

Design: Systematic review and meta-analysis using the GRADE approach.

Data sources: MEDLINE, Embase, CINAHL, and the Cochrane Library were searched through 07 February 2017.

Eligibility criteria for selecting studies: We included randomized controlled trials ≥3-weeks assessing the effect of pasta alone or in the context of low-GI dietary patterns on measures of global (body weight, BMI, body fat) and regional (waist circumference, waist-to-hip ratio, sagittal abdominal diameter) adiposity in adults.

Data extraction and synthesis: Two independent reviewers extracted data and assessed risk of bias. Data were pooled using the generic inverse-variance method and expressed as mean differences (MDs) with 95% confidence intervals (95% CIs). Heterogeneity was assessed (Cochran Q statistic) and quantified (I^2-statistic). GRADE assessed the certainty of the evidence.

Results: We identified no trials of the effect of pasta alone and 32 trial comparisons (n=2,448 participants) of the effect of pasta in the context of low-GI dietary patterns. Pasta in the context of low-GI dietary patterns significantly reduced body weight (MD=-0.63kg [95% CIs,-0.84,-0.42kg]) and BMI (MD=-0.26kg/m^2 [95% CIs,-0.36,-0.16kg/m^2]) compared with higher-GI dietary patterns. There was no effect on other measures of adiposity. The certainty of the evidence was graded as moderate for body weight, BMI, WHR, and SAD and low for WC and body fat.

Conclusions: Pasta in the context of low-GI dietary patterns does not adversely affect adiposity and even reduces body weight and BMI compared to higher GI dietary patterns. Future trials should assess the effect of pasta in the context of other healthy dietary patterns**.**

Protocol registration: ClinicalTrials.gov Identifier, NCT02961088

Strengths and limitations of this study

- The present systematic review and meta-analysis was based on a comprehensive search and includes a large number of randomized controlled trials which provide the best protection against bias.
- We used the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach to evaluate the strength and quality of the evidence.
- There was evidence of unexplained inconsistency in the intervention estimates across trials for waist circumference and body fat.
- The generalizability of our results is questionable with evidence of indirectness in the pooled estimates for all body and adiposity outcomes, as all of the trials assessed pasta in the context of low-GI dietary patterns (no trials assessed the effect of pasta alone or in the context of other dietary patterns) and most of the available trials did not quantify the amount of pasta consumed.

INTRODUCTION

As the role of saturated fat in chronic disease has been called into question, carbohydrates have come under attack in the media[1,2], popular books[3-9], statements of health advocacy groups[10] and commentaries in leading medical journals[11,12]. Much of the attention has focused on sugars but traditional carbohydrate staples like pasta, rice, and breads are increasingly being implicated in the epidemics of overweight and obesity[1,7]. Although systematic reviews and meta-analyses of randomized controlled trials of dietary patterns that are high in these foods but low in glycemic index (GI)[13,14], high in whole grains[15,16], and/or high in dietary fibre have shown advantages for weight related outcomes [17,18], there has been a general lack of recognition of the importance of carbohydrate quality.

Pasta is an important example of a food which is considered a refined carbohydrate but has a low-GI, a property that has been exploited extensively in studies of low-GI dietary patterns. It remains unclear whether pasta alone or in the context of a low-GI dietary pattern shares the advantages of other low-GI foods or on the contrary contributes to weight gain. We are not aware of any systematic reviews and meta-analyses which have synthesized the evidence of the effect of pasta on body weight outcomes. We undertook a systematic review and meta-analysis of randomized controlled trials (RCTs) using the GRADE approach to quantify the effect of pasta alone or in the context of low-GI dietary patterns on body weight and measures of adiposity relevant to the prevention and management of overweight and obesity.

METHODS

Design

Our protocol followed the guideline of the Cochrane Handbook for Systematic Reviews of Interventions[19], and findings are reported according to the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses[20] (**Supplemental Table S1**). The protocol is registered at clinicaltrials.gov (identifier, NCT02961088).

Data sources and searches

We searched MEDLINE (http://www.nlm.nih.gov/bsd/pmresources.html), Embase (https://www.embase.com), CINAHL (https://health.ebsco.com/products/the-cinahl-database), and the Cochrane Library (http://www.cochranelibrary.com/) from inception through 07 February 2017. The full search terms used in this study are presented in **Supplemental Tables S2-S3**. Briefly, we searched using variations of the terms pasta and glycemic index and glycemic load and body weight and BMI. The search was limited to human studies and had no language restrictions. Reference lists of selected studies and reviews were also searched to identify additional articles.

Study selection

We include RCTs that investigated the effect of pasta consumed alone or in the context of low-GI dietary patterns that emphasized pasta in comparison with higher GI diets that did not include pasta on body weight or other measures of global (BMI, body fat) or abdominal (waist circumference, waist-to-hip ratio, sagittal abdominal diameter, or visceral adipose tissue assessed by imaging modalities) adiposity in participants of all health backgrounds. Trials were included if the intervention arm assessed the effect of pasta consumed alone or assessed the effect of a low GI diet which emphasized pasta as part of the low GI dietary advice. Trials were excluded if they

had diet duration of <3 weeks, did not intend to use a calorie- and macronutrient-matched comparator arm that was higher in GI; included pregnant or breast-feeding women or children; or did not provide suitable end-point data. When multiple publications existed for the same study, the article with the most information was included (n=6). Published abstracts were not included.

Data extraction

Two reviewers (LC, CB) assessed the titles and abstracts of all identified studies and independently reviewed and extracted relevant data from each report, including study design, blinding, sample size, participant characteristics, follow-up duration, identification of pasta in the low-GI diet only, comparator diet, macronutrient profile, funding source, and outcome data. The primary outcome was body weight, and secondary outcomes included markers of global (BMI, body fat) and abdominal (waist circumference, waist-to-hip ratio, sagittal abdominal diameter, or visceral adipose tissue assessed by imaging modalities) adiposity. Mean±SD differences between test and control arms were extracted for each outcome.

In those trials where the data were included in figures and not provided numerically, we used the software program Plot Digitizer version 2.6.8 (http://plotdigitizer.sourceforge.net/) to extract the data. This program is a JAVA program that digitizes scanned figures of X and Y plots from GIF, JPEG, or PNG image file formats and allows one to calibrate the X and Y axes for the estimation data points. Additional information was requested from the authors of all included trials. Disagreement were resolved by consensus or where necessary by a third author (SBM).

Risk of bias assessment

Risk of bias for each individual study was assessed using the Cochrane Risk of Bias tool[19]. The level of bias was evaluated for sequence generation, allocation concealment, blinding, incomplete outcome data, and selective reporting and determined overall as either low (proper methods taken to reduce bias), high (improper methods creating bias), or unclear (insufficient information provided to determine the bias level).

Statistical analysis

Baseline, end-of-study, and between-treatment differences in body weight, BMI, waist circumference, body fat, waist-to-hip ratio, sagittal abdominal diameter or visceral adipose tissue assessed by imaging modalities were recorded as means±SDs. If not provided, between-treatment differences in change-from-baseline or end differences were calculated by subtracting means and variance measures such as SEs were imputed with the use of published formulas[19]. Missing SDs were imputed with the use of the pooled SD from other studies included in the analysis[19].

Data analyses were conducted using Review Manager (RevMan) version 5.3 (Copenhagen, Denmark: The Nordic Cochrane Centre, The Cochrane Collaboration, 2014) for primary analyses and Stata version 13 (College Station, TX: StataCorp LP) for subgroup analyses. A generic inverse-variance method with random-effects models was used to calculate pooled mean differences and 95% confidence intervals (CIs). Random-effects models were used even in the absence of statistically significant inter-study heterogeneity, as they yield more conservative summary effect estimates in the presence of residual heterogeneity. Change-from-baseline differences were preferred over end differences and paired analyses were applied to all crossover

trials with the use of a within-individual correlation coefficient between treatments of 0.5 as described by Elbourne et al.[21]

Inter-study heterogeneity was assessed by the Cochran Q statistic, where P<0.10 was considered statistically significant, and quantified by the I^2 statistic, where $I^2 \geq 50\%$ indicates substantial heterogeneity[19]. Sensitivity analysis were performed which included the removal of each single study from the meta-analyses one at a time and recalculation of the summary effect. An influential study was considered a study whose removal changed the magnitude of the pooled effect by >10%. Sensitivity analysis were also conducted using different correlation coefficient values for crossover trials (0.25 and 0.75) to test for the robustness of the effect size, conducting analyses using fixed effects models and restricting analyses to those trials for which pasta intake could be quantified.

If ≥10 trial comparisons were available, then sources of heterogeneity were explored by subgroup analyses. A priori categorical subgroups analyses were assessed by meta-regression analyses. These included patient type (normal body weight, overweight or obese [average baseline BMI >27kg/m^2]), diabetes, coronary heart disease), follow-up (<24-weeks, ≥24 weeks), baseline BMI (BMI ≤30, >30kg/m^2), design (parallel, crossover), energy balance (negative on both arms [weight loss diets], neutral on both arms [weight maintaining diets]) and dose of pasta (based on the median). A priori categorical subgroup analyses also included the following dietary factors: GI (absolute level [≤55, >55; glucose scale], within-treatment change, between-treatment change), fat intake (absolute level [<30%, ≥30% energy], within-treatment change, between-treatment change), carbohydrate intake (absolute level [<50%, ≥50% energy], within-treatment change, between-treatment change), protein intake (absolute level [<20%, ≥20% energy], within-treatment change, between-treatment change), dietary fibre intake (absolute level [<28g/day,

≥28g/day], within-treatment change, between-treatment change), and risk of bias. A priori continuous meta-regression analyses were conducted on the absolute levels and within- and between-treatment changes of these same dietary factors in the intervention arms of pasta in the context of low-GI dietary patterns. Linear and non-linear pasta intake dose-response analyses were assessed by using continuous meta-regression analyses and spline curve modeling (MKSPLINE procedure), respectively. Publication bias was assessed by visual inspection of funnel plots and the Egger[22] and Begg[23] tests, when ≥10 trial comparisons were available. If publication bias was suspected, then adjustment for funnel plot asymmetry was done by imputing missing study data using the Duval and Tweedie trim and fill method [24].

Grading the evidence

The grading of recommendations assessment, development, and evaluation (GRADE) approach was used to assess the certainty of the evidence [25]. Evidence was graded as high, moderate, low or very low quality. The included RCTs were graded as high quality evidence by default and downgraded based on pre-specified criteria. Criteria to downgrade evidence included risk of bias (weight of studies show risk of bias assessed by the Cochrane Risk of Bias tool), inconsistency (substantial unexplained heterogeneity, I^2>50%, P<0.10), indirectness (presence of factors that limited the generalizability of the results), imprecision (the 95% CI for effect estimates were wide or crossed pre-specified minimally important differences [MIDs] for harm), and publication bias (significant evidence of small-study effects).

Patient involvement

No patients were directly involved in the development of the research question, selection of the outcome measures, design and implementation of the study, or interpretation of the results.

RESULTS

Search results

Figure 1 shows the flow of the literature. We identified 4876 reports of which 29 met eligibility criteria. No reports were identified that assessed the effect of pasta alone, while 29 reports (including 32 trial comparisons involving 2448 participants) were identified that assessed the effect of pasta in the context of low-GI dietary patterns on any adiposity outcome in adults[26-54]. Of the 32 trial comparisons that assessed the effect of pasta in the context of a low-GI dietary pattern, there were 32 trial comparisons for body weight, 18 trial comparisons for BMI[27,28,31-33,35,36,39-41,43-46,48,49,52,53], 18 trial comparisons for waist circumference[27,28,31,33,34,36,38-40,42,44-47,52,53], 10 trial comparisons for body fat[27,28,31-33,36,38,41,43,53], 6 trial comparisons for waist-to-hip ratio[31,39,40,44,45,52], and 3 trial comparisons for sagittal abdominal diameter[34,39]. There was only one trial comparison identified[35] for visceral adipose tissue assessed by imaging modalities, thus a meta-analysis could not be undertaken for this outcome.

Trial characteristics

Table 1 and **Supplemental Table S4** show the characteristics of all included trials of the effect of pasta in the context of low-GI dietary patterns. The majority of trials had a parallel design (26/32) with a median follow-up of 12 weeks (IQR: 9–21 weeks) and a median number of participants per trial of 43 (IQR: 21–112). Most participants were middle aged (median age: 50

y; IQR: 40-58 y) men and women (median ratio of men to women: 0.4:1 in available trials). The median baseline BMI across studies was 30.4kg/m^2 (IQR: 28.2–32.0). Regarding metabolic phenotype, 21 (66%) trials included participants who were overweight or obese (had a baseline BMI≥27kg/m^2), 10 (31%) had diabetes and one trial (3%) with coronary heart disease (CHD). We did not retrieve any trials where participants had a normal BMI at baseline (≤25kg/m^2), although 6 trials did not include BMI >25 kg/m^2 as part of criteria, the average baseline BMI was ≥27 kg/m^2, therefore categorized as overweight.

Risk of bias

Supplemental Figures S1 and S2 show the individual Cochrane Risk of Bias tool assessments for each of the included trials of the effect of pasta in the context of low-GI dietary patterns. No serious risk of bias was detected.

Effect of Pasta in the Context of Low-GI dietary patterns and Body Weight

Figure 2 shows the effect of pasta in the context of low-GI dietary patterns on the primary outcome body weight. Pooled analyses showed pasta in the context of low-GI dietary patterns had the effect of reducing body weight by -0.63 kg (95% CI:-0.84, -0.42 kg; P<0.001) compared with higher GI control diets with no evidence of heterogeneity (I^2=0%, P-heterogeneity=0.51).

Effect of Pasta in the Context of Low-GI dietary patterns on Markers of Global Adiposity

Figure 3 and **Supplemental Figure S3-S4** show the effect of pasta in the context of low-GI dietary patterns on markers of global adiposity. Pooled analyses showed that pasta in the context of low-GI dietary patterns had the effect of reducing BMI by -0.26kg/m^2 (n=18 trials; MD=-

0.26kg/m^2; 95% CI:-0.36, -0.16 kg/m^2; P<0.001) compared with higher GI control diets with no evidence of heterogeneity (I^2=0%, P-heterogeneity=0.90). There was no effect on body fat (n=10 trials; MD=-0.01%, 95% CI:-0.58, 0.56%; P=0.98) with evidence of substantial heterogeneity (I^2=65%, P-heterogeneity<0.01).

Effect of Pasta in the Context of Low-GI dietary patterns on Markers of Abdominal Adiposity

Figure 3 and **Supplemental Figures S5-S7** show the pooled estimates for the markers of abdominal adiposity. Pooled analyses did not show a significant effect of pasta in the context of low-GI dietary patterns compared with higher-GI control diets on waist circumference (n=18 trials; MD=-0.46cm, 95% CI:-1.05, 0.14cm; P=0.13), waist-to-hip ratio (n=6 trials; MD=-0.00, 95% CI:-0.01, 0.00; P=0.27), or sagittal abdominal diameter (n=3 trials, MD=-0.09cm, 95% CI:-0.34, 0.16cm; P=0.48) There was only evidence of substantial heterogeneity in the analyses for waist circumference (I^2=62%, P-heterogeneity<0.01).

Sensitivity analyses

We conducted four sets of sensitivity analyses (**Supplemental Tables S5-6, Supplemental Figures S8-9**). The systematic removal of each trial did not modify the direction or significance of the effect estimates or the evidence of heterogeneity for any of the outcomes with the exception of waist circumference (**Supplemental Table S5**). In the sensitivity analysis for waist circumference, two studies were influential studies in that their removal altered the magnitude of the pooled effect in the remaining studies by >10%, where the removal of the studies of McMillan-Price et al. (high protein comparison)[42] and Jenkins et al.[44] rendered the results for

waist circumference statistically significant (MD= -0.62cm; 95% CI: -1.19, -0.05; P=0.03) and (MD= -0.61cm; 95% CI: -1.18, -0.04; P=0.04), respectively; forest plots not shown). Heterogeneity remained significant in both cases (I^2= 55%, P-heterogeneity<0.01 and I^2=50%, P-heterogeneity=0.01, respectively). Sensitivity analyses using correlation coefficients of 0.25 and 0.75 for paired analyses of crossover trials also did not modify the results (**Supplemental Table S6**). In the sensitivity analyses where fixed effects models were applied (**Supplemental Figure S8**), the direction, magnitude and significance of the pooled estimates were very similar to those produced by the random effects models with the exception of the sensitivity analysis for waist circumference which was significant (MD= -0.62; 95% CI: -0.93, -0.32; P<0.001). Finally, restricting analyses to the 11 trials for which pasta intake could be quantified (median pasta intake, 3.33 servings/week [range, 1.75 – 7 servings/week]) showed a similar reduction in body weight (MD= -0.70 kg; 95% CI:-1.10, -0.29 kg; P<0.001) when pasta was consumed in the context of low-GI dietary patterns compared with the higher GI control arms without evidence of heterogeneity (I^2=0%, P-heterogeneity=0.68) (**Supplemental Figure S9)**.

Subgroup analyses

We were only able to conduct a priori categorical and continuous subgroup analyses for body weight, BMI, body fat and waist circumference. Subgroup analyses for waist-to-hip ratio and sagittal abdominal diameter could not be assessed, owing to <10 trial comparisons in each case. **Supplemental Figures S10-S12** show the categorical a priori subgroup analyses for body weight. There was no evidence of significant effect modification in any of the subgroup analyses for body weight, including no effect modification of follow-up when comparing studies less than 24 weeks duration to those greater than or equal to 24 weeks (-0.63kg vs -0.57kg, respectively)

(**Supplemental Figure S10**). Neither was there evidence of significant effect modification in any of the subgroup analyses for BMI, body fat or waist circumference (**Supplemental Figures S13-20**).

Supplemental Table S7 and **Supplemental Figures S21-22** show the continuous subgroup analyses for body weight. There was evidence of significant effect modification by carbohydrate and protein intake, where an increase in carbohydrate intake in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with weight loss (β= -0.07, 95% CI: -0.12, -0.01, I^2=0.00%, P=0.02), and an increase in protein intake in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with weight gain (β=0.15, 95% CI: 0.03, 0.27, I^2=0.00%, P=0.02). None of the other continuous subgroup analyses were significant. There was no evidence of significant effect modification in any of the continuous subgroup analyses for BMI (**Supplemental Table S8**). For body fat, there was evidence of significant effect modification in the continuous meta-regression subgroup analysis of difference in GI between intervention and control groups, where greater difference in GI between the groups was associated with greater reduction in body fat in the intervention group (β=-0.09, 95% CI: -0.15, -0.03, I^2=19.39%, P=0.01) (**Supplemental Table S9**). None of the other continuous subgroup analyses were significant. For waist circumference, there was evidence of significant effect modification in the continuous meta-regression subgroup analysis of absolute carbohydrate level and absolute protein level, where greater carbohydrate level in the intervention group in which pasta was consumed in the context of low-GI dietary patterns was associated with greater loss in waist circumference (β=-0.11, 95% CI: -0.19, -0.04, I^2=27.06%, P<0.01) and a lower protein level in the intervention group in which pasta was

consumed in the context of low-GI dietary patterns was associated with an increase in waist circumference (β= 0.20, 95% CI: 0.01, 0.38, I^2=43.92%, P=0.04) (**Supplemental Table S10**). None of the other continuous subgroup analyses were significant.

Dose-response analyses

Supplemental Tables S7 and S11 and **Supplemental Figure S23** show the dose-response analysis for the 11 trials for which pasta intake could be quantified. No evidence of a linear dose response was seen for pasta intake by meta-regression analyses (**Supplemental Table S7**). There was also no evidence of a non-linear dose response by MKSPLINE (P=0.85) (**Supplemental Figure S23**) or piecewise linear meta-regression analyses (**Supplemental Table S11**).

Publication Bias

Supplemental Figures S24-S27 shows the funnel plots for body weight, BMI, body fat and waist circumference. There was no evidence of funnel-plot asymmetry. Formal testing with the Egger and Begg tests did not show evidence of small-study effects (P>0.05 for both). Publication bias was not assessed for waist-to-hip ratio and sagittal abdominal diameter, owing to <10 trial comparisons.

GRADE Assessment

Supplemental Table S12 shows a summary of the GRADE assessments for the effect of pasta in the context of low-GI dietary patterns on body weight and measures of adiposity**.** The evidence was graded as moderate for body weight, BMI, waist-to-hip ratio and sagittal abdominal

diameter owing to downgrades for indirectness and low for waist circumference and body fat, owing to downgrades for indirectness and inconsistency (I^2=59%, P-heterogeneity<0.001; I^2=66%, P-heterogeneity<0.01, respectively).

DISCUSSION

The present systematic review and meta-analysis quantified the effect of pasta alone and pasta in the context of low-GI dietary patterns on body weight and other markers of adiposity. We failed to identify any trial comparisons for the effect of pasta alone but did identity 32 trial comparisons for the effect of pasta in the context of low-GI dietary patterns in 2448 participants who were predominantly middle-aged and overweight or obese. The primary pooled analysis demonstrated that pasta in the context of low-GI dietary patterns did not contribute to weight gain, resulting in a significant weight loss of -0.63kg when compared to diets higher in GI over a median follow-up of 12 weeks. The lack of harm was reflected in the established clinical secondary outcome measures of global (BMI and body fat) and abdominal adiposity (waist circumference, waist-to-hip ratio and sagittal abdominal diameter). These findings were robust across subgroups. The findings did not differ by metabolic phenotype in those who were overweight or obese or had diabetes, which is noteworthy since these are populations who would benefit from weight management strategies. There was also no effect modification by the energy balance of the design such that the weight loss was seen even under conditions of neutral energy balance (in which participants were instructed to consume dietary advice ad libitum), suggesting that encouragement of the consumption of pasta in the context of a low-GI dietary patterns does not cause harm and may even lead to spontaneous weight loss. There was also no effect modification by follow-up either in continuous meta-regression or categorical, where the 24 trials with

<24weeks follow-up had a weight reduction similar to those 8 trials with ≥24weeks follow-up (-0.63kg vs -0.57kg, respectively). This is of relevance since many dietary studies are successful in demonstrating weight loss in the short term but not over the long term.

Findings in the context of existing studies

We are not aware of any RCTs directly assessing the effect of pasta intake on any health parameters including body weight. Our findings, however, agree with earlier systematic reviews and meta-analyses of RCTs of the effect of low-GI dietary patterns irrespective of pasta intake on body weight and adiposity. The systematic review and meta-analysis by Thomas et al. in 2007 found a significant -1.1kg weight loss and -1.3kg/m^2 reduction in BMI favouring low-GI or glycemic load (GL) diets compared to control diets in 6 RCTs of 5 weeks to 6 months in duration in overweight or obese individuals[13]. Another systematic review and meta-analysis by Schwingshackl et al. in 2013 found a -0.62kg weight loss favouring low-GI/GL diets compared to higher GI/GL diets in 14 RCTs with greater than 6 months duration in overweight individuals (BMI>25kg/m^2)[14].

Our findings also agree with trials in which pasta was emphasized in the context of other healthy dietary patterns. One trial done in children in Spain given Mediterranean dietary advice which included increasing the intake of pasta found that approximately 11.3% of the participants in the Mediterranean diet group who were classified as overweight and obese changed their weight status to normal weight compared to only approximately 2.6% of the participants in the control group [55].

Other lines of evidence from observational studies have demonstrated benefits of pasta consumption on body weight and adiposity. Pasta intake was recently assessed in the Moli-sani study and the Italian Nutrition & HEalth Survey (INHES), a cross-sectional study of over 20,000 Italians from all over Italy[56]. The study demonstrated that higher pasta consumption was associated with lower BMI, waist circumference and waist-to-hip ratio and with a lower prevalence of overweight and obesity[56]. Furthermore, greater pasta intake was associated with better adherence to the Mediterranean diet, a dietary pattern which has a demonstrated cardiovascular benefit[57]. Similar associations between greater pasta intake and lower body weight have also been observed in prospective U.S. cohorts[58]. A pooled analysis of the 3 Harvard cohorts also showed that higher GI diets (which would preclude pasta) were associated with weight gain[59].

Although the product form of pasta can vary widely, including in shape (e.g. macaroni, spaghetti, linguine), ingredients (e.g. type of wheat, egg content), and processing technique (e.g. drying temperature), studies have demonstrated that when comparing pastas varying in these parameters, despite slight variations in glycemic response among pastas, glycemic responses are still lower compared to a control, e.g. white bread[60,61]. One concern of the choice of pasta as a carbohydrate food is that is it a refined food low in fibre. Although there are whole grain pasta options available, studies have demonstrated that fiber added to pasta, does not significantly affect the glucose or insulin response, the secretion of gut hormones or satiety[62,63]. Furthermore, pasta has a similar glycemic response compared to many fiber-rich carbohydrates, including barley, legumes and steel cut oats, and still a lower glycemic response compared to other fiber-rich foods including whole wheat bread, breakfast cereals like bran flakes and potatoes with

skin[64]. The typically consumed white wheat pasta also has a higher micronutrient content compared to other white wheat products like bread since it contains the aleurone layer which is preserved as a result of the use of harder wheats (durum wheat); even when durum wheats are used in breads, pasta retains a lower glycemic response primarily because of the processing techniques used in pasta making which give pasta a compact structure and reduced starch hydrolysis[61].

The mechanism by which pasta in the context of low-GI dietary patterns lead to weight loss even under conditions of ad libitum dietary advice is unclear. Lower GI diets may result in greater body weight reduction compared to higher GI diets because lower GI foods may be more satiating [65] and have been shown to delay hunger and decrease subsequent energy intake[13]. Low-GI dietary patterns are also characterized by high fiber content[64,66] which may also contribute to improvements in satiety and hunger[17]. Furthermore, studies which have compared ad libitum low-GI dietary patterns to standard energy-restricted low-fat diets, demonstrated equivalency or better weight loss when following the low-GI diet, despite the fact that they could eat as much as they desired[13,67]. Thus, voluntary energy intake may be lower after low-GI meals, as has been previously demonstrated[68].

Strengths and limitations

The strengths of the present systematic review and meta-analysis include that it is comprehensive, includes RCTs which protects against bias and uses the GRADE approach to evaluate the quality of evidence. Additionally, a large number of trials were identified (32 trials) for the primary outcome of body weight, the median follow-up period was 12 weeks which

allows for the assessment of a moderate duration of intervention, none of the trials were rated as having a serious risk of bias, and there was no evidence of publication bias.

There are several limitations. First, we downgraded the certainty of the evidence for serious inconsistency in the treatment estimates across trials for some of the outcomes assessed. There was evidence of unexplained heterogeneity in waist circumference (I^2=62%) and in body fat (I^2=65%). Although the inconsistency in these outcomes may have related to measurement error[69] in the different techniques for measuring waist circumference and body fat, we were unable to conduct sensitivity or subgroup analyses to explore this source of heterogeneity. Second, we downgraded the certainty of the evidence for serious indirectness. Most of the available trials did not quantify the amount of pasta consumed in the context of the low-GI dietary patterns. Although sensitivity analyses in which analyses were restricted to the 11 trials that did quantify (providing a median 3.33 servings/week) pasta intake did not meaningfully alter our estimates (-0.70kg versus -0.63kg), it is difficult to quantify the effect of pasta in these diets. There is also the question of indirectness in the translation to other background diets. None of the available trials evaluated the effect of pasta alone or in the context of other dietary patterns. Whether the observed effect of pasta in the context of low-GI dietary patterns will hold in the context of other healthy dietary patterns, such as Mediterranean and Vegetarian dietary patterns, is unclear. Although there is no biological reason to doubt that the findings would hold across different dietary patterns, there was no direct evidence to support this conclusion. If the question had been asked from the perspective of benefit as opposed to that of harm, then the relatively short duration of the included trials is another reason to downgrade for serious indirectness. In the absence of long-term trials (>1 year diet duration), it is difficult to conclude with certainty

that the observed lack of harm implies an actual sustainable benefit. Finally, there was some evidence of imprecision for benefit. Whereas the 95% CI of the pooled estimates did not overlap with our pre-specified MID for harm (that is, they did not contain evidence for harm) and so were not downgraded for imprecision, the upper bound of the 95% CI did overlap with the lower bound of the same MID to assess the precision of the evidence for benefit for some outcomes.

Balancing these strengths and limitations, the GRADE approach assessed the overall quality and strength of the available evidence of the effect of pasta in the context of low-GI dietary patterns as moderate for the primary outcome of body weight and the secondary outcomes of BMI, waist-to-hip ratio and sagittal abdominal diameter owing to downgrades for indirectness. The evidence was assessed as low for the other secondary outcomes of body fat and waist circumference owing to downgrades for indirectness and inconsistency.

Implications

These results are important considering the negative messages directed at the public regarding carbohydrates, which is influencing their food choices, as is evident in recent reductions in carbohydrate intake[70-72], and in particular reductions in pasta consumption[70,73-76]. Contrary to these concerns, the available evidence shows that when pasta is consumed in the context of low-GI dietary patterns that there is not weight gain but rather marginally clinically significant weight loss (>0.5kg)[77].

Although we were able to approximate the amount of pasta consumed in one third of included trials, it is unclear what the effect of pasta would be in the context of other dietary patterns. Low-

GI dietary patterns, however, shares many similarities with a Mediterranean diet, which emphasizes many low-GI foods and has demonstrated a CVD benefit[57].

Current clinical practice guidelines already suggest the replacement of high GI foods with low-GI foods for improvement of glycemic control and cardiovascular risk factors[78,79]. The present evidence means that pasta may be highlighted as an important example of a low-GI food which can contribute to a low-GI dietary pattern, a pattern which in turn may potentially improve cardio-metabolic risk without an adverse effect on weight control.

CONCLUSIONS

In conclusion, the available evidence from RCTs does not allow us to conclude that pasta consumed in the context of low-GI dietary patterns has an adverse effect on body weight and adiposity outcomes of importance in the prevention and management of overweight and obesity. On the contrary, pasta in the context of low-GI dietary patterns reduces body weight and BMI compared with higher-GI dietary patterns. The results are generalizable in the context of a high carbohydrate dietary pattern composed of low-GI foods with or without the intention of weight loss in middle-aged individuals who are overweight or obese or have diabetes. Although the clinical significance of the observed weight loss is debatable, this finding increases our confidence that pasta in the context of low-GI dietary patterns does not result in weight gain. Further research may change our confidence in the estimates for our primary outcome body weight and several key secondary outcomes including BMI and two measures of abdominal adiposity, waist-to-hip ratio and sagittal abdominal diameter. More research is needed, to improve our estimates for the secondary outcomes, body fat and waist circumference and assess

whether our findings extend to related cardio-metabolic outcomes. There is also a need for more randomized trials of >1 year diet duration to clarify whether the lack of harm for pasta in the context of low-GI dietary patterns will translate into meaningful long-term benefits. Other randomized trials should focus on whether pasta will have similar effects in the context of other healthy dietary patterns such as a Mediterranean diet.

Acknowledgements

We wish to thank Teruko Kishibe, an information specialist in the Scotiabank Health Sciences Library, St Michael's Hospital, for her help in the development of search terms used. Aspects of this work were presented at the Società Italiana di Nutrizione Umana (SINU) Congress, in Parma, Italy, on April 12, 2017 and at the 35th International Symposium on Diabetes and Nutrition, of the Diabetes and Nutrition Study Group (DNSG) of the European Association for the Study of Diabetes (EASD) in Skagen, Denmark, on June 20 and 21, 2017.

Data Sharing

No additional data available.

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Ethics Approval

Not required.

Authorship:

All authors had full access to all of the data (including statistical reports and tables) in this study and take full responsibility for the integrity of the data and the accuracy of the data analysis.

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Transparency declaration: As guarantor, J.L. Sievenpiper affirms that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects

of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

Funding:

This work was funded by the Canadian Institutes of Health Research (funding reference number, 129920) through the Canada-wide Human Nutrition Trialists' Network (NTN). The Diet, Digestive tract, and Disease (3-D) Centre, funded through the Canada Foundation for Innovation (CFI) and the Ministry of Research and Innovation's Ontario Research Fund (ORF), provided the infrastructure for the conduct of this project. **L Chiavaroli** was funded by a Toronto 3D Postdoctoral Fellowship Award. **DJA Jenkins** was funded by the Government of Canada through the Canada Research Chair Endowment. **JL Sievenpiper** was funded by a PSI Graham Farquharson Knowledge Translation Fellowship, Canadian Diabetes Association (CDA) Clinician Scientist award, CIHR INMD/CNS New Investigator Partnership Prize, and Banting & Best Diabetes Centre Sun Life Financial New Investigator Award. None of the sponsors had a role in any aspect of the present study, including design and conduct of the study; collection, management, analysis, and interpretation of the data; and preparation, review, approval of the manuscript or decision to publish.

Conflicts of Interest:

All authors have completed the Unified Competing Interest form (available on request from the corresponding author) and declare: **CWC Kendall** has received research support from the Advanced Foods and Material Network, Agrifoods and Agriculture Canada, the Almond Board of California, the American Pistachio Growers, Barilla, the California Strawberry Commission, the Calorie Control Council, CIHR, the Canola Council of Canada, the Coca-Cola Company (investigator initiated, unrestricted grant), Hain Celestial, the International Tree Nut Council Nutrition Research and Education Foundation, Kellogg, Kraft, Loblaw Companies Ltd., Orafti, Pulse Canada, Saskatchewan Pulse Growers, Solae and Unilever. He has received travel funding, consultant fees and/or honoraria from Abbott Laboratories, the Almond Board of California, the American Peanut Council, the American Pistachio Growers, Barilla, Bayer, the Canola Council

of Canada, the Coca-Cola Company, Danone, General Mills, the International Tree Nut Council Nutrition Research and Education Foundation, Kellogg, Loblaw Companies Ltd., the Nutrition Foundation of Italy, Oldways Preservation Trust, Orafti, Paramount Farms, the Peanut Institute, PepsiCo, Pulse Canada, Sabra Dipping Co., Saskatchewan Pulse Growers, Solae, Sun-Maid, Tate and Lyle, and Unilever. He is on the Clinical Practice Guidelines Expert Committee for Nutrition Therapy of the European Association for the Study of Diabetes (EASD) and has served on the scientific advisory board for the Almond Board of California, the International Tree Nut Council, Oldways Preservation Trust, Paramount Farms and Pulse Canada. **DJAJ** has received research grants from Saskatchewan Pulse Growers, the Agricultural Bioproducts Innovation Program through the Pulse Research Network, the Advanced Foods and Material Network, Loblaw Companies Ltd., Unilever, Barilla, the Almond Board of California, Agriculture and Agri-food Canada, Pulse Canada, Kellogg's Company, Canada, Quaker Oats, Canada, Procter & Gamble Technical Centre Ltd., Bayer Consumer Care, Springfield, NJ, Pepsi/Quaker, International Nut & Dried Fruit (INC), Soy Foods Association of North America, the Coca-Cola Company (investigator initiated, unrestricted grant), Solae, Haine Celestial, the Sanitarium Company, Orafti, the International Tree Nut Council Nutrition Research and Education Foundation, the Peanut Institute, the Canola and Flax Councils of Canada, the Calorie Control Council (CCC), the CIHR, the Canada Foundation for Innovation and the Ontario Research Fund. He has received in-kind supplies for trial as a research support from the Almond board of California, Walnut Council of California, American Peanut Council, Barilla, Unilever, Unico, Primo, Loblaw Companies, Quaker (Pepsico), Pristine Gourmet, Bunge Limited, Kellogg Canada, WhiteWave Foods. He has been on the speaker's panel, served on the scientific advisory board and/or received travel support and/or honoraria from the Almond Board of California, Canadian Agriculture Policy Institute, Loblaw Companies Ltd, the Griffin Hospital (for the development of the NuVal scoring system, the Coca-Cola Company, EPICURE, Danone, Diet Quality Photo Navigation (DQPN), FareWell, Verywell, True Health Initiative, Saskatchewan Pulse Growers, Sanitarium Company, Orafti, the Almond Board of California, the American Peanut Council, the International Tree Nut Council Nutrition Research and Education Foundation, the Peanut Institute, Herbalife International, Pacific Health Laboratories, Nutritional Fundamental for Health, Barilla, Metagenics, Bayer Consumer Care, Unilever Canada and Netherlands, Solae, Kellogg, Quaker Oats, Procter & Gamble, the Coca-Cola Company, the Griffin Hospital, Abbott Laboratories, the Canola Council of Canada, Dean Foods, the California Strawberry Commission, Haine Celestial, PepsiCo, the Alpro Foundation, Pioneer Hi-Bred International, DuPont Nutrition and Health, Spherix Consulting and WhiteWave Foods, the Advanced Foods and Material Network, the Canola and Flax Councils of Canada, the Nutritional Fundamentals for Health, Agri-Culture and Agri-Food Canada, the Canadian Agri-Food Policy Institute, Pulse Canada, the Saskatchewan Pulse Growers, the Soy Foods Association of North America, the Nutrition Foundation of Italy (NFI), Nutra-Source Diagnostics, the McDougall Program, the Toronto Knowledge Translation Group (St. Michael's Hospital), the Canadian College of Naturopathic Medicine, The Hospital for Sick Children, the Canadian Nutrition Society (CNS), the American Society of Nutrition (ASN), Arizona State University, Paolo Sorbini Foundation and the Institute of Nutrition, Metabolism and Diabetes. He received an honorarium from the United States Department of Agriculture to present the 2013 W.O. Atwater Memorial Lecture. He received the 2013 Award for Excellence in Research from the International Nut and Dried Fruit Council. He received funding and travel support from the Canadian Society of Endocrinology and Metabolism to produce mini cases for the Canadian Diabetes Association

(CDA). He is a member of the International Carbohydrate Quality Consortium (ICQC). His wife, ALJ, is a director and partner of Glycemic Index Laboratories, Inc., and his sister received funding through a grant from the St. Michael's Hospital Foundation to develop a cookbook for one of his studies. **JL Sievenpiper** has received research support from the Canadian Institutes of health Research (CIHR), Canadian Diabetes Association (CDA), PSI Foundation, Banting and Best Diabetes Centre (BBDC), Canadian Nutrition Society (CNS), American Society for Nutrition (ASN), Calorie Control Council, INC International Nut and Dried Fruit Council Foundation, National Dried Fruit Trade Association, The Coca-Cola Company (investigator initiated, unrestricted), Dr. Pepper Snapple Group (investigator initiated, unrestricted), The Tate and Lyle Nutritional Research Fund at the University of Toronto, and The Alberta Pulse Growers Glycemic Control and Cardiovascular Disease in Type 2 Diabetes Fund at the University of Toronto. He has received in-kind research support from the Almond board of California, Walnut Council of California, American Peanut Council, Barilla, Unilever, Unico, Primo, Loblaw Companies, Quaker (Pepsico), Pristine Gourmet, Bunge Limited, Kellogg Canada, WhiteWave Foods. He has received reimbursement of travel expenses, speaker fees, and/or honoraria from the Canadian Diabetes Association (CDA), Canadian Nutrition Society (CNS), University of Alabama at Birmingham, Oldways Preservation Trust, Nutrition Foundation of Italy (NFI), Diabetes and Nutrition Study Group (DNSG) of the European Association for the Study of Diabetes (EASD), International Life Sciences Institute (ILSI) North America, Dr. Pepper Snapple Group, Corn Refiners Association, World Sugar Research Organization, Dairy Farmers of Canada, Società Italiana di Nutrizione Umana (SINU), III World Congress of Public Health Nutrition, C3 Collaborating for Health, Sprim Brasil, WhiteWave Foods, Rippe Lifestyle, mdBriefcase, Federation of European Nutrition Societies (FENS), New York Academy of Sciences, International Diabetes Federation, American Heart Association (AHA), American Society for Nutrition (ASN), FoodMinds LLC, Memac Ogilvy & Mather LLC, Pulse Canada, Pepsico, BCFN Foundation, The Ginger Network LLC, and Dietitians of Canada. He has ad hoc consulting arrangements with Winston & Strawn LLP, Perkins Coie LLP, and Tate & Lyle. He is a member of the European Fruit Juice Association Scientific Expert Panel. He is on Clinical Practice Guidelines Expert Committees of the Canadian Diabetes Association (CDA) European Association for the study of Diabetes (EASD), and Canadian Cardiovascular Society (CCS), as well as an expert writing panel of the American Society for Nutrition (ASN). He serves as an unpaid scientific advisor for the Food, Nutrition, and Safety Program (FNSP) and the Technical Committee on Carbohydrates of the International Life Science Institute (ILSI) North America. He is a member of the International Carbohydrate Quality Consortium (ICQC), Executive Board Member of the Diabetes and Nutrition Study Group (DNSG) of the EASD, and Director of the Toronto 3D Knowledge Synthesis and Clinical Trials foundation. His wife is an employee of Unilever Canada. No competing interests were declared by **CR Braunstein**, **S** Blanco Mejia**, and LA Leiter.**

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Figure Legend

Figure 1: Literature Search

Figure 2: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg). n = 2448. Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.
CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids; Pro, protein.

Figure 3: Plot of pooled effect estimates from randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on global and abdominal markers of adiposity. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.
BMI; body mass index; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet.

Table 1: Summary of trial characteristics

Trial Characteristics*	ALL	Neutral Energy Balance	Negative Energy Balance
Trial Number (n)	32	23	9
Trial Size (total, range)	2448 (8 - 250)	1989 (8 - 250)	459 (13 - 123)
Male: Female[a] (%)	40:60	47:53	27:73
Age (years)	50 (40 - 58)	52.0 (42.1 – 59.5)	49.5 (34.2 – 53.0)
Metabolic Phenotype (OW/OB:DM:CHD) (%)	66:31:3	57:39:4	89:11:0
Setting (IP:OP) (%)	3:97	4:96	0:100
Baseline Body Weight (kg)[b]	85.5 (80.0 - 91.9)	84.1 (79.5 – 87.5)	92.5 (86.1 – 93.9)
Baseline BMI (kg/m^2)[c]	30.4 (28.2 - 32.0)	29.5 (27.4 – 31.4)	31.7 (30.1 – 32.9)
Study Design (C:P) (%)	19:81	26:74	0:100
Dose Pasta (servings/week)[d]	3.3 (2.3 - 3.5)	3.4 (2.9 – 4.1)	2.3 (2.3-3.5)
GI in Pasta/LGI group	49.0 (44.0 - 55.1)	46.5 (49.9 – 55.5)	44.0 (42.3 - 49.4)
GI in Higher GI group	62.5 (61.6 - 63.2)	63.3 (60.1 – 64.4)	61.0 (59.2 – 66.6)
Calorie reduction in Pasta/LGI group (kcal)[e]	-179 (-90 - -448)	-165 (-74 - -313)	-447 (-134 - -594)
Calorie reduction in Higher GI group (kcal)[e]	-181 (-93 - -401)	-160 (-40 - -248)	-470 (-172 - -561)
Feeding Control (Met:Supp:DA) (%)	6:44:50	4:48:48	11:33:56
Follow-Up Duration (Weeks)	12 (9-21)	12 (6-24)	12 (10 - 21)
Funding Sources (A:I:AI:NR) (%)	47:9:25:19	44:13:26:17	56:0:22:22

* median (inter quartile range), unless otherwise indicated
[a]24/32 trials provided data on sex
[b] 30/32 trials reported baseline body weight
[c] 28/32 trials reported baseline BMI
[d] 11/32 trials provided data from which dose could be approximated
[e] 20/32 trials provided data from which to approximate changes in caloric intake
A, agency; AI, agency and industry; BMI, body mass index; C, crossover design; CHD, coronary heart disease; DA, dietary advice; DM, diabetes; GI, glycemic index; I, industry; IP, inpatient; LGI, low glycemic index; Met, metabolic; NR, not reported; OB, obese; OP, outpatient; OW, overweight; P, parallel design; Suppl, supplemented/provision of certain food

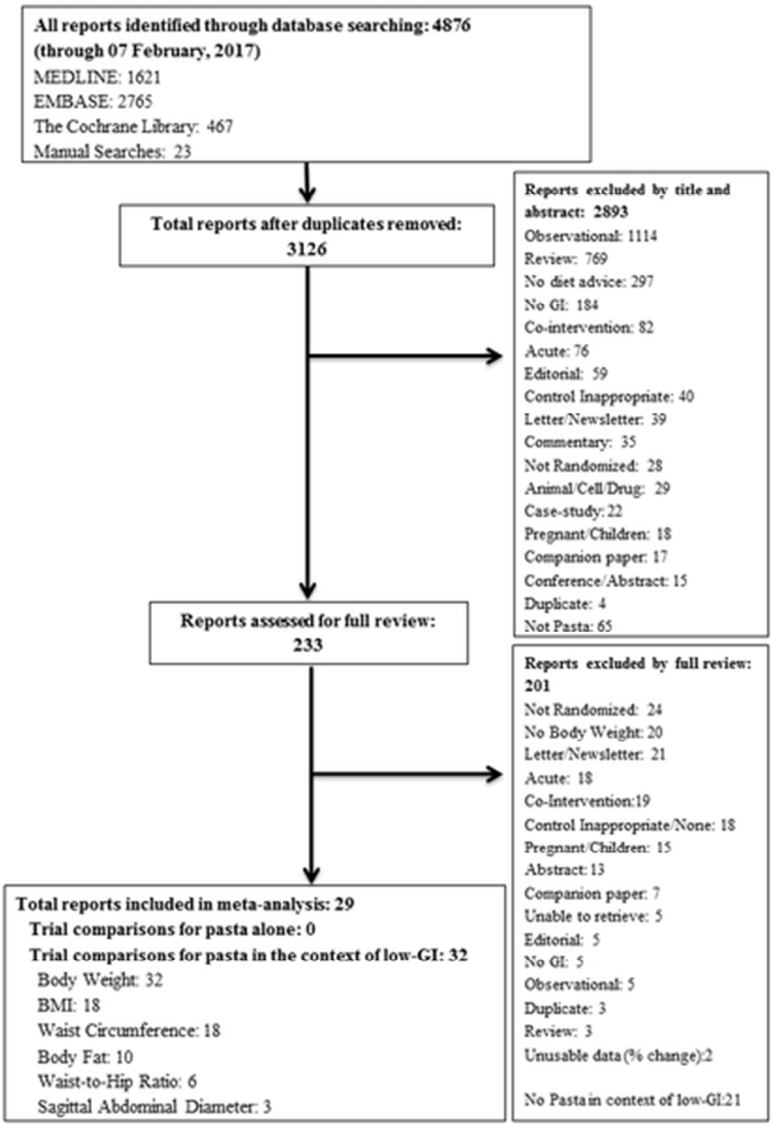

Figure 1: Literature Search

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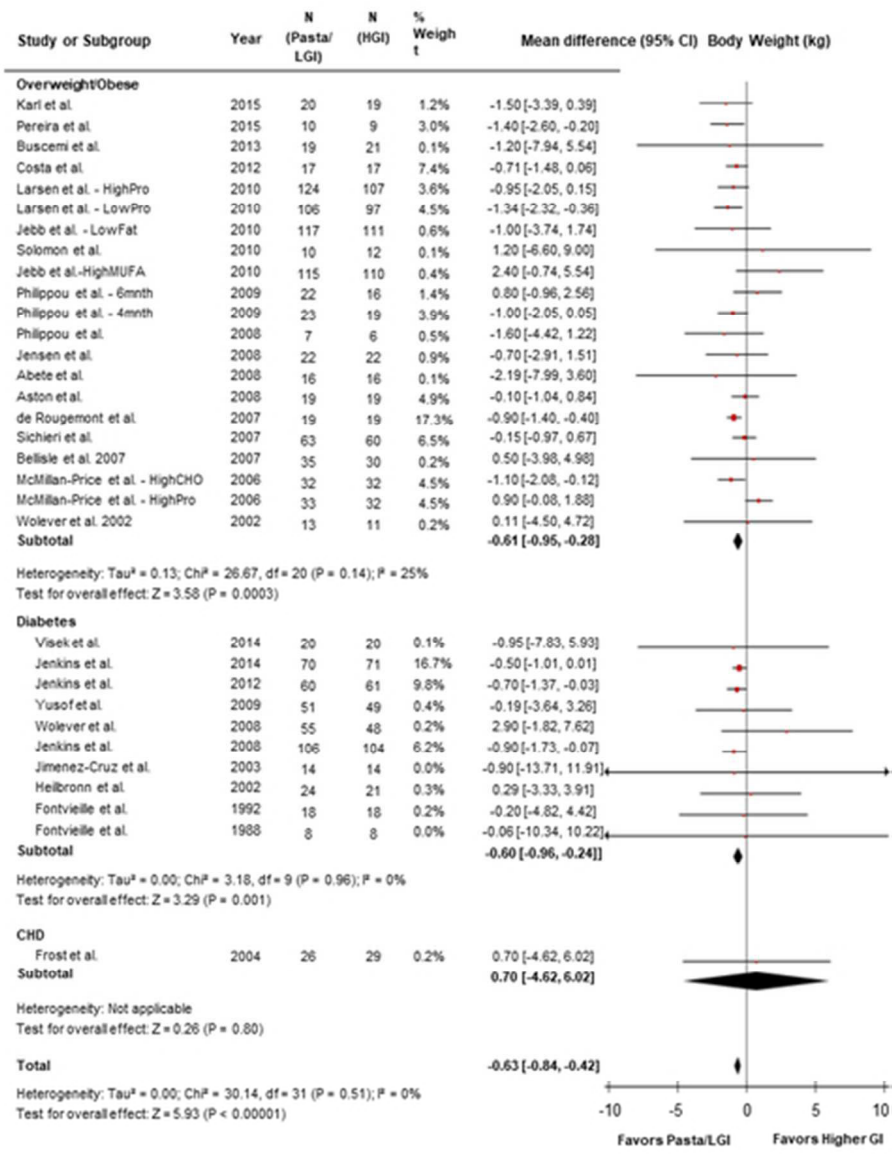

Figure 2: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg). n = 2448. Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I2 statistic where I2≥50% is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.

CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids; Pro, protein.

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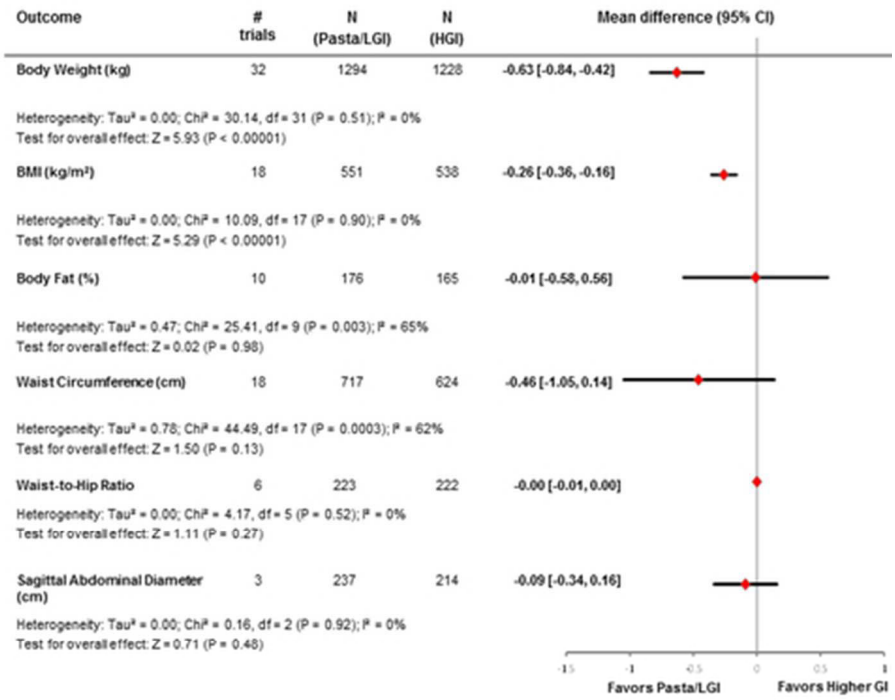


Figure 3: Plot of pooled effect estimates from randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on global and abdominal markers of adiposity. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I2 statistic where I2≥50% is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.
BMI; body mass index; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet.

40x54mm (300 x 300 DPI)

Online Supplemental Information

Table of Contents

Supplemental Tables

Supplemental Table S1: PRISMA Checklist

PRISMA 2009 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.	2-3
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	4
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	4
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 Table S2
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, Supplemental Table S1
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	Supplemental Table S1
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
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

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, Supplemental Table S2
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.	6-7
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	7-8
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.	8
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).	9
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	7-10
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.	10, 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.	10-11, Table 1, Supplemental Tables S2-S3
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).	11, 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.	11-13, Figures 2-3, Supplemental Figures S3-S7
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	11-13, Figures 2-3, Supplemental Figures S3-S7
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	11,15 Supplemental

			Table S1, S2, S12, S15, S17, S20
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	12-16, Supplemental Tables S4-S10, Supplemental Figures S8-S26
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).	16-22
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).	19-21
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	22-23
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.	26-31

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 studies assessing the effect of pasta in the context of low-GI dietary patterns on body weight in randomized controlled trials

Database	Search Period	Search Terms
Medline	1946 to February 07, 2017	1. pasta/ 2. spaghetti/ 3. macaroni/ 4. lasagna/ 5. fusilli/ 6. noodle/ 7. glycaemic index.tw. 8. glycemic index.tw. 9. glycaemic ind*.tw. 10. glycemic ind*.tw. 11. glycemic load*.tw. 12. glycaemic load*.tw. 13. glycemic index/ 14. body mass index/ 15. body mass index.tw. 16. BMI.tw. 17. overweight.tw. 18. weight*.tw. 19. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 20. 14 or 15 or 16 or 17 or 18 21. 19 and 20 22. limit 21 to animals 23. 21 not 22
Embase	1946 to February 07, 2017	1. pasta/ 2. spaghetti/ 3. macaroni/ 4. lasagna/ 5. fusilli/ 6. noodle/ 7. glycaemic index.tw. 8. glycemic index.tw. 9. glycaemic ind*.tw. 10. glycemic ind*.tw. 11. glycemic load*.tw. 12. glycaemic load*.tw. 13. glycemic index/ 14. body mass index/ 15. body mass index.tw. 16. BMI.tw. 17. overweight.tw. 18. weight*.tw.

		19. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 or 12 or 13 20. 14 or 15 or 16 or 17 or 18 21. 19 and 20 22. limit 21 to animals 23. 21 not 22
The Cochrane Library	1946 to February 07, 2017	1. pasta/ 2. spaghetti/ 3. macaroni/ 4. lasagna/ 5. fusilli/ 6. noodle/ 7. glycemic index/ 8. glycaemic ind*.tw. 9. glycemic ind*.tw. 10. glycemic load*.tw. 11. glycaemic load*.tw. 12. exp body weight/ 13. body weight*.tw. 14. BMI.tw. 15. body mass index/ 16. body mass index.tw. 17. weight*.tw. 18. 1 or 2 or 3 or 4 or 5 or 6 or 7 or 8 or 9 or 10 or 11 19. 12 or 13 or 14 or 15 or 16 or 17 20. 18 and 19 21. limit 20 to animals 22. 20 not 21

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**
Adult men and women excluding pregnant or breastfeeding women	Low glycemic index interventions where pasta is included as part of the intervention	Higher glycemic index diets where pasta is not included as part of the intervention	Body weight Body mass index (BMI) Body Fat (%) Waist circumference Waist-to-hip ratio

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

Supplement Table S4a: Trial characteristics

Overweight/Obese Trials

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Karl et al. 2015 ‡‡‡	39 (19M:20F)**	OB, OP					2.33	USA	P	Negative+ Neutral	17		CR to 2/3kcal; Metabolic	Agency
Low GI	20		56 (5)*	92.9 (13.6)*	32.3 (3.4)*	42:133						68:15:16		
Higher GI	19		56 (5)*	94 (9.7)*	33.4 (2.6)*	61:201						70:16:14		
Pereira et al. 2015	19 (4M:15F)**	OW, IP/OP					NR	Brazil	P	Neutral	6.4		Ad libitum	Unknown
Low GI	10		28(5)	80.0(12.6)	29.9 (2.1)	41.2(2.2) ¥ **						48.3:16.1:32.8		
Higher GI	9		26(3)	79.1(12.2)	29.1 (2.0)	74.1(2.9) ¥ **						54.6:12.7:34.4		
Buscemi et al. 2013	40 (19M:21F)**	OW/OB, high CVD risk, OP					NR	Italy	P	Negative	12		CR to 20kcal/kg/d; Ad libitum	Unknown
Low GI	19		51 (8)	93.8 (17.3)	34.3 (6.6)	48.1: 138						56:18:26		
Higher GI	21		49 (8)	93.2 (14.4)	34.5 (5.1)	59.3: 174						57:16:27		
Costa et al. 2012	17 (7M:10F)	OW, IP/OP	25.4 (5.8)	84.1(16.3)	26.3(3.2)		NR	Brazil	C	Neutral	4		Ab libitum,2 meals+3 fruit/d provided	NR
Low GI						47.5(3.8)						58.6:13.9:25.5		
Higher GI						61.6(2.8)						55.4:14.2:30.3		
Jebb et al. 2010 - High MUFA	225**	OH, some CV risk factors, OP	~52		~28.5+		1.75	UK	P	Neutral	24		Ad Libitum, key foods provided	Agency, foods by industry
Low GI	115			83.7 (69.6-93.1)¶¶		~55.2						~44.6:16.4:35.7****		
Higher GI	110			80.5 (70.0-92.1)¶¶		~63.3						~44.9:15.3:35.6****		
Jebb et al. 2010 - Low Fat	250**	OH, some CV risk factors, OP	~52		~28.5+		3.5	UK	P	Neutral	24		Ad Libitum, key foods provided	Agency, foods by industry
Low GI	117			79.4 (70.1- 91.8)¶¶		~56.3						~51.5:14.2:26.1****		
Higher GI	111			80.7 (71.4- 91.4)¶¶		~64.4						~51.1:15.7:27.5****		
Larsen et al. 2010-High Pro	231**	OW/OB, OP			NR		NR	Europe	P	Neutral	26		Ad libitum	Agency
Low GI	124		42.1 (6.5)	88.5 (15.6)		~56.5: 108.9						~43:22:32		
Higher GI	107		42 (5.7)	89.5 (17.1)		~61.4: 113.1						~45:23:31		
Larsen et al. 2010 - Low Pro	203 **	OW/OB, OP			NR		NR	Europe	P	Neutral	26		Ad libitum	Agency
Low GI	106		42.2 (5.7)	88.4 (15.7)		~56.2: 121						~51:18:30		
Higher GI	97		42 (5.9)	86.6 (13.8)		~61.6: 137.9						~51:17:31		

Supplement Table S4b: Trial characteristics continued

Overweight/Obese Trials continued

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Solomon et al. 2010	22 (8M:14F)**	OB, Pre-T2DM, OP					7	USA	P	Neutral	12		Metabolic plus excerise program	Agency
Low GI	10 (3M:7F)		67 (6)	97.4 (12.0)	34.9 (1.1)	39.8 (0.9)						54.7(0.3):28.3(0.3):17.0(0.3)		
Higher GI	12 (5M:7F)		64 (3)	94.7 (15.2)	34.1 (1.1)	80.0 (2.1)						55.6(0.7):27.8(0.7):16.6(0.3)		
Philippou et al. 2009- 6 mo	38**	≥1 CHD risk factors, OP	(35-65) ¶				NR	UK	P	Negative (for all but n=2)	24		500kcal CR; Ad libitum	Agency
Low GI	22			91.3(14.8)***	29.1 (3.6)***	50.6 (4.6): 114.4(31.5)								
Higher GI	16			97.5(16.4)***	30.5 (3.5)***	63.2(5.6): 175.0(45.6)								
Philippou et al. 2009- 4 mo	42**	OW, OP	(18-65)¶				NR	UK	P	Neutral	16		Ad libitum	Unknown
Low GI	23			87.2 (15.3)	32.5 (4.8)	49.7(5.7):89.7(27.5)						47.6(6.7):19.5(4.2):31.8(5.8)		
Higher GI	19			83.6 (13.4)	31.3 (4.8)	63.7(9.4):136.8(56.3)						48.9(7):19.3(4.9):30.9(9)		
Abete et al. 2008	32 (18M:14F)	OB, OP	36(7)				2.33	Spain	P	Negative	8		30% CR; Ad libitum, 3-day menus	Agency
Low GI	16			94.3(16.1)	32.8 (4.3)	(40-45)¶						50.2 (1.8);18.3(1.6);31.5(1.6)		
Higher GI	16			94.4(13.1)	32.2 (4.4)	(60-65)¶						47.8(6.8);19.6(5.6);32.6(4.3)		
Aston et al. 2008	19 (0M:19F)**	OW/OB, OP	51.9(7.6)	87.5 (15)	33.1 (4.9)		3.33	UK	C	Neutral	12		Ad libitum, key CHO foods provided	Agency
Low GI						55.5(3.8): 133.8(22.9)****						51.4(6.0):17.0(2.4):32.2(5.1)***		
Higher GI						63.9(3): 138.8(30.5)****						47.6(6.1):17.6(3.3):34.1(5.7)***		
Jensen et al. 2008	44 (0M:44F)**	OW, OP	(20-40)¶				3	Denmark	P	Neutral	10		Ad libitum, partial provision, menu plans	Agency, Industry
Low GI	22 (0M:22F)			77.9(6.9)	27.4 (1.5)	72¥						~57(5):17(0):23(5) ‡		
Higher GI	22 (0M:22F)			80.2(1.4)	27.6 (0.3)	95¥						~57(5):17(0):22(5) ‡		
Philippou et al. 2008	13 (5M:8F)**	≥1 CHD risk factors, OP					NR	UK	P	Negative	12		500kcal CR; Ad libitum	Agency
Low GI	7 (3M:4F)		54 (49-58)¶¶	81.5 (4.7)***	28.7 (2.1)***	51.3(51.0-52.0): 105.6 (76.9-110.1)¶¶						46.0(37.8-51.0): 17.1(15.7-17.4): 32.8(31.3-37.1)¶¶		
Higher GI	6 (2M:4F)		45 (39-50)¶¶	89.7 (12.8)***	31.5 (4.4)***	59.3(59.2-64.0): 114.7(98.5-134.9)¶¶						49.4(47.8-51.7):19.6(14.0-23.1):29.2(25.2-34.5)¶¶		

Supplement Table S4c: Trial characteristics continued

Overweight/Obese Trials continued

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Bellisle et al. 2007	65 (0M:65F)**	OW/OB, OP					NR	France	P	Neutral	12		Ad libitum	Industry
Low GI	35		46.1 (13.6)	80 (13.2)	30.2 (4.1)	na								
Higher GI	30		45.3 (12.0)	79 (13.1)	30.4 (4.4)	na								
de Rougemont et al. 2007	38 (22M:16F)**	OW, OP					2.8	France	P	Neutral	5		Ad libitum, some foods provided	Agency, Industry
Low GI	19		36.3 (8.7)	77.2 (9.6)	27.5 (1.3)	46.5 (1.3)						42.6 (3.9):19.8 (1.7):37.7 (4.4)		
Higher GI	19		40.4 (9.6)	77.3 (9.2)	27.2 (1.3)	66.3 (2.6)						44.1 (3.5):17.6 (1.7):38.4 (3.1)		
Sichieri et al. 2007	123 (OM:123F)**	OW, OP					NR	Brazil	P	Negative	72		100-300kcal CR; 6-d menu and exchange lists provided	Agency
Low GI	63		37.2 (5.4)*	67.7 (6.6)*	na	21(38): 74(84)						59.5 (6.3): 13.3: 27.2(4.6)		
Higher GI	60		37.5 (5.6)*	68.5 (7.5)*	na	51(28): 199(43)						61.6 (6.2): 12.3: 26.1(4.7)		
McMillian-Price et al. 2006-High Carb	64(16M:48F)	OW/OB, OP					NR	Australia	P	Negative	12		Ad libitum, key foods and meals provided	Agency-Industry
Low GI	32		30.5 (7.9)	87.1 (15.3)	30.6 (4.5)	45 (6):89 (28)						56 (6):19 (0):22 (6)		
Higher GI	32		31.8 (9.6)	86 (10.7)	30.9 (3.4)	70 (6):129 (45)						60 (6):18 (6):19 (6)		
McMillian-Price et al. 2006-High Protein	65(15M:50F)	OW/OB, OP					NR	Australia	P	Negative	12		Ad libitum, key foods and meals provided	Agency-Industry
Low GI	33		34.6 (8.6)	88.4 (17.2)	32.1 (5.2)	44 (6):59 (23)						40 (11):26 (6):28 (6)		
Higher GI	32		30.2 (8.5)	87.7 (16.4)	31.3 (4.5)	59 (6):75 (17)						42 (6):28 (6):27 (6)		
Wolever et al. 2002	24 (5M:19F)**	IGT, OP					NR	Canada	P	Neutral	16		Ad libitum, partial provision	Agency
Low GI	13(3M:10F)		55.2 (10.8)	79.7 (13.1)***	29.7 (4.3)	54.4 (2.5):91.8 (9.4)						54.8 (6.1):19.4 (1.8):24.7 (5.8)		
Higher GI	11(2M:9F)		58.8 (13.3)	76.4 (20.4)***	29.3 (7.3)	59.3 (2.0):96.8 (11.6)						52.8 (6.6):17.4 (2.3):27.9 (6.3)		

Supplement Table S4d: Trial characteristics continued

Diabetes Trials

Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Jenkins et al. 2014	141(77M:64F)	T2DM, OP					NR	Canada	P	Neutral	12		Ad libitum, bread supplement	Industry Association
Low GI	70 (38M:32F)		59 (10)	85 (20)	30 (5)	~51:53						~38.5:19.8:37.2		
Higher GI	71 (39M:32F)		59 (10)	84 (19)	31 (6)	~62:89						~49.2:19.8:27.4		
Visek et al. 2014	20 (12M:8F)	T2DM, OP	62.7 (5.8)	91.9 (14.1)	32 (4.2)		NR	Czech Republic	C	Neutral	12		Ad libitum	Agency
Low GI						49 (48-51)¶ ¶						~37.2:18.0:36.0		
Higher GI						68 (61-72)¶ ¶						~36.2:17.3:40.0		
Jenkins et al. 2012	121 (61M: 60F)	T2DM, OP					NR	Canada	P	Neutral	12		Ad libitum	Agency
Low GI	60		58 (10.1)	85.6 (20.1)	31.4 (7.0)	47: 80						45.4:22.8:30.5		
Higher GI	61		61 (7.8)	82.5 (17.2)	29.9 (5.5)	58: 100						48.3:21.4:28.5		
Yusof et al. 2009	100**	T2DM, OP	NR				NR	Malaysia	P	Neutral	12		Ad libitum, key foods provided to lowGI group	Agency
Low GI	51			69.12 (13.33)	27.05 (4.91)	57(6): 108(32)						52(4):18(3):30(4)		
Higher GI	49			66.83 (11.50)	26.79 (4.65)	64(5): 131(30)						54(4):17(3):28(5)		
Jenkins et al 2008	210 (125M:82F)	T2DM, OP					NR	Canada	P	Neutral	24		Ad libitum	Agency
Low GI	106 (65M:41F)		60 (10)	87.0 (20.0)	30.6 (6.0)	49.4: 91.5						44.0:21.2:33.3		
Higher GI	104 (63M:41F)		61 (9)	87.8 (19.4)	31.2 (5.8)	59.3: 117.9						47.5:20.7:30.5		
Wolever et al. 2008	103	T2DM, OW/OB, OP					NR	Canada	P	Neutral	52		Ad libitum, key foods provided	Agency
Low GI	55		60.6 (7.5)*	81.1 (18.7)*	31.6 (4.5)*	55.1 (3.0): 133 (14.8)						51.9 (6.7):20.6(3.0):26.5 (5.9)		
Higher GI	48		60.4 (7.9)*	84.4(18.0)*	30.1 (4.3)*	63.2 (2.8): 135 (20.8)						46.5 (6.2):20.4 (2.8):30.8 (4.8)		

Supplement Table S4e: Trial characteristics continued

Diabetes Trials continued														
Study	Subjects	Sample Description	Mean Age (y) (SD)	Mean Baseline Body Weight (kg) (SD)	Mean Baseline BMI (kg/m^2) (SD)	Mean GI:GL of diet (SD) ¶ ¶ ¶	Pasta Dose (serv/wk)	Setting	Design	Energy Balance§	Duration (wks)	Diet Composition % (SD)§§	Dietary Prescription	Funding Source
Jimenez-Cruz et al. 2003	14 (6M:8F)**	T2DM, OP	59 (34)				NR	Mexico	C	Neutral	6		Ad libitum	Industry
Low GI				91.6 (24.3)	32.4 (6.0)	44(3.4): 86(19.8)						60:21:23		
Higher GI				92.6 (25.4)	32.3 (6.0)	56(4.9): 139(27.3)						64:18:20		
Heilbronn et al. 2002	45 (23M:22F)**	T2DM, OW, OP			NR		3.5	Australia	P	Negative	8		CR to1500kcal/d; Ad libitum, partial provision	Unknown
Low GI	24 (11M:13F)		56.0(9.4)	91.7(16.2)		43						58.9 (2.9):22.2 (1.5):17.9 (3.9)		
Higher GI	21 (12M:9F)		57.5(9.6)	93.2 (13.3)		75						60.8 (2.3):21.7 (0.9):17.1 (2.3)		
Fontvieille et al. 1992	18 (12M:6F)	T1DM/T2DM, OP	47.2(11.6)	NR	24.8(2.6)		4.7	France	C	Neutral	5		Ad libitum	Agency, Industry
Low GI						38.1(5.3)						45.8(7.2):18.0(2.5):36.2(6.8)		
Higher GI						64.2(3.1)						44.9(7.3):18.8(1.6):36.3(6.0)		
Fontvielle et al. 1988	8 (4M:4F)	T1DM, OP	43.5 (9.9)	NR	24.1 (6.8)		3.5	France	C	Neutral	3		Ad libitum, partial provision	Agency, Industry
Low GI						46.5(2.5)						46.1 (4.5):17.4 (1.4):35.0 (2.8)		
Higher GI						60.1 (5.1)						45.4 (4.5):16.9 (1.7):36.0 (2.8)		
CHD Trial														
Frost et al. 2004	55 (48M:7F)**	CHD, OP					NR	UK	P	Neutral §§§	12		Ad Libitum	Unknown
Low GI	26 (23M:3F)		63.6 (9.4)	81.2 (12.2)	26.9 (3.3)	50(4):115(39)						49 (5):18 (5):31 (5)		
Higher GI	29 (25M: 4F)		61.8 (9)	81.7 (16.7)	28.7 (4.6)	57(4):106(34)						47 (10):18 (5):32 (10)		

Abbreviations: BMI=Body Mass Index; C=Crossover design; Carb=Carbohydrate; CED= carbohydrate exchange diet; CHD= Coronary Heart Disease; CR= calorie restriction; CV=cardiovascular; GI= glycemic index; GL=glycemic load; IGT= impaired glucose tolerance; IP=inpatient; IR= insulin resistant; High Carb= high carbohydrate; HI= hyperinsulinemic; M = male; mo=months; MUFA=monounsaturated fatty acids; na=not available; T1DM=type 1 diabetes mellitus; T2DM = type 2 diabetes mellitus; OB= obese; OH=otherwise healthy; OP = outpatient; OW= overweight; P=Parallel design; SD= standard deviation; F = female; UK= United Kingdom; USA= United States of America; wks=weeks; y= years

§Negative energy balance diets were designed for weight loss, Neutral energy balance diets for weight maintenance, where all diets are isocaloric between test and control groups; §§Energy from carbohydrate: protein: fat for the planned diet or if not planned; §§§ Participants were advised to lose weight if they had a BMI>28kg/m2.

¶ Range of values; ¶¶ median and interquartile range (IQR); ¶ ¶ ¶ Actual GI/GL or if not available, planned;

* Calculated before dropout; **Completer Analysis, as used in data analysis; *** completer analysis, data obtained from authors; **** based on less participants than in analysis;

+ approximate based on all study arms; ¥ approximate based on test meals; ‡ approximate based on n=45 from Sloth et al. 2004, the original publication of this study;

‡‡‡ analysis includes weight loss and weight maintenance phases

Supplemental Table S5: Sensitivity analysis of the systematic removal of each trial*

	MD [95% CI], P-value I^2, P-value					
	Body Weight (kg) N=32	**BMI (kg/m^2) N=18**	**Body Fat (%) N=10**	**Waist Circumference (cm) N=18**	**Waist-to-hip Ratio N=6**	**Sagittal Abdominal Diameter (cm) N=3**
All studies	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.51	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.91	-0.01 [-0.58, 0.56], P=0.98 65%, P=0.003	-0.46 [-1.05, 0.14], P=0.13 62%, P<0.01	-0.00 [-0.01, 0.00], P=0.27 0.00%, P=0.52	-0.09 [-0.34, 0.16], P=0.48 0.00%, P=0.92
Removal of:						
OW/OB						
Pereira et al. 2015	-0.61 [-0.82, -0.39], P<0.01 0.00%, P=0.54	-0.24 [-0.34, -0.14], P<0.01 0.00%, P=0.93	0.19 [-0.25, 0.63], P=0.39 36%, P=0.13	-0.37 [-1.04, 0.31], P=0.28 62%, P<0.01	-0.00 [-0.01, 0.01], P=0.67 0.00%, P=0.58	n/a
Karl et al. 2015	-0.62 [-0.83, -0.41], P<0.01 0.00%, P=0.50	-0.25 [-0.35, -0.16], P<0.01 0.00%, P=0.91	0.10 [-0.51, 0.71], P=0.74 64%, P<0.01	n/a	n/a	n/a
Buscemi et al. 2013	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.86	-0.08 [-0.63, 0.48], P=0.79 64%, P<0.01	-0.46 [-1.06, 0.15], P=0.14 64%, P<0.01	n/a	n/a
Costa et al. 2012	-0.62 [-0.84, -0.41], P<0.01 0.00%, P=0.46	-0.26 [-0.37, -0.16], P<0.01 0.00%, P=0.86	-0.06 [-0.73, 0.61], P=0.86 66%, P<0.01	-0.33 [-0.94, 0.28], P=0.29 59%, P<0.01	n/a	n/a
Jebb et al. 2010 - LowFat	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
Larsen et al. 2010 - LowPro	-0.60 [-0.81, -0.38], P<0.01 0.00%, P=0.57	n/a	n/a	-0.45 [-1.09, 0.18], P=0.16 64%, P<0.01	n/a	-0.05 [-0.39, 0.29], P=0.79 0.00%, P=0.86
Jebb et al. 2010-HighMUFA	-0.64 [-0.85, -0.43], P<0.01 0.00%, P=0.65	n/a	n/a	n/a	n/a	n/a
Larsen et al. 2010 - HighPro	-0.62 [-0.83, -0.41], P<0.01 0.00%, P=0.48	n/a	n/a	-0.37 [-1.00, 0.26], P=0.26 62%, P<0.01	n/a	-0.13 [-0.45, 0.19], P=0.42 0.00%, P=0.92
Solomon et al. 2010	-0.63 [-0.84, -0.42], P<0.01	-0.26 [-0.36, -0.16], P<0.01	n/a	n/a	n/a	n/a

	0.00%, P=0.47	0.00%, P=0.88				
Philippou et al. 2009 - 6mnth	-0.65 [-0.86, -0.44], P<0.01 0.00%, P=0.59	-0.28 [-0.37, -0.18], P<0.01 0.00%, P=0.97	-0.12 [-0.71, 0.48], P=0.70 65%, P<0.01	-0.55 [-1.15, 0.04], P=0.07 61%, P<0.01	n/a	n/a
Philippou et al. 2009 - 4mnth	-0.61 [-0.83, -0.40], P<0.01 0.00%, P=0.48	-0.25 [-0.35, -0.15], P<0.01 0.00%, P=0.89	-0.09 [-0.72, 0.54], P=0.78 66%, P<0.01	-0.44 [-1.08, 0.19], P=0.17 64%, P<0.01	n/a	n/a
Abete et al. 2008	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	n/a	n/a	n/a	n/a	n/a
Philippou et al. 2008	-0.62 [-0.83, -0.42], P<0.01 0.00%, P=0.48	-0.26 [-0.35, -0.16], P<0.01 0.00%, P=0.89	-0.06 [-0.68, 0.55], P=0.84 68%, P<0.01	-0.41 [-1.02, 0.19], P=0.18 63%, P<0.01	n/a	n/a
Aston et al. 2008	-0.66 [-0.87, -0.44], P<0.01 0.00%, P=0.52	n/a	-0.00 [-0.70, 0.69], P=0.99 68%, P<0.01	-0.54 [-1.14, 0.07], P=0.08 62%, P<0.01	n/a	n/a
Jensen et al. 2008	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.27 [-0.36, -0.17], P<0.01 0.00%, P=0.88	n/a	-0.44 [-1.05, 0.18], P=0.16 64%, P<0.01	-0.00 [-0.01, 0.00], P=0.24 0.00%, P=0.41	-0.09 [-0.35, 0.18], P=0.51 0.00%, P=0.69
de Rougemont et al. 2007	-0.57 [-0.80, -0.34], P<0.01 0.00%, P=0.53	-0.25 [-0.36, -0.14], P<0.01 0.00%, P=0.87	0.06 [-0.57, 0.70], P=0.84 67%, P<0.01	n/a	n/a	n/a
Sichieri et al. 2007	-0.66 [-0.88, -0.45], P<0.01 0.00%, P=0.53	n/a	n/a	n/a	n/a	n/a
Bellisle et al. 2007	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.88	n/a	-0.47 [-1.08, 0.13], P=0.13 64%, P<0.01	-0.00 [-0.01, 0.00], P=0.14 0.00%, P=0.70	n/a
McMillan-Price et al. 2006 - HighCHO	-0.61 [-0.82, -0.39], P<0.01 0.00%, P=0.51	n/a	n/a	-0.38 [-1.01, 0.25], P=0.23 63%, P<0.01	n/a	n/a
McMillan-Price et al. 2006 - HighPro	-0.70 [-0.91, -0.49], P<0.01 0.00%, P=0.91	n/a	n/a	**-0.62 [-1.19, -0.05], P=0.03 55%, P<0.01**	n/a	n/a
Wolever et al. 2002	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
Diabetes:						
Visek et al. 2014	-0.63 [-0.84, -0.42], P<0.01	-0.26 [-0.36, -0.16], P<0.01	0.01 [-0.57, 0.60], P=0.96	n/a	n/a	n/a

	0.00%, P=0.46	0.00%, P=0.86	68%, P<0.01			
Jenkins et al. 2014	-0.66 [-0.88, -0.43], P<0.01 0.00%, P=0.47	-0.29 [-0.39, -0.18], P<0.01 0.00%, P=0.94	n/a	**-0.61 [-1.18, -0.04], P=0.04 50%, P=0.01**	-0.00 [-0.01, 0.00], P=0.21 0.00%, P=0.43	n/a
Jenkins et al. 2012	-0.62 [-0.84, -0.40], P<0.01 0.00%, P=0.46	-0.25 [-0.35, -0.15], P<0.01 0.00%, P=0.87	n/a	-0.44 [-1.05, 0.17], P=0.16 64%, P<0.01	-0.00 [-0.01, 0.01], P=0.64 0.00%, P=0.53	n/a
Yusof et al. 2009	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.87	n/a	-0.33 [-0.95, 0.28], P=0.29 58%, P<0.01	n/a	n/a
Jenkins et al. 2008	-0.61 [-0.83, -0.40], P<0.01 0.00%, P=0.48	-0.25 [-0.36, -0.15], P<0.01 0.00%, P=0.87	n/a	n/a	n/a	n/a
Wolever et al. 2008	-0.64 [-0.84, -0.43], P<0.01 0.00%, P=0.57	n/a	n/a	-0.50 [-1.10, 0.09], P=0.10 62%, P<0.01	n/a	n/a
Jimenez-Cruz et al. 2003	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	-0.26 [-0.35, -0.16], P<0.01 0.00%, P=0.86	n/a	n/a	n/a	n/a
Heilbronn et al. 2002	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	n/a	n/a	n/a	n/a	n/a
Fontvieille et al. 1992	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
Fontvielle et al. 1988	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.46	n/a	n/a	n/a	n/a	n/a
CHD						
Frost et al. 2004	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.47	-0.26 [-0.36, -0.17], P<0.01 0.00%, P=0.89	n/a	-0.48 [-1.09, 0.12], P=0.12 63%, P<0.01	-0.00 [-0.01, 0.00], P=0.25 1%, P=0.40	n/a

*Sensitivity analysis included the removal of each single study from the meta-analyses one at a time and the summary effect was recalculated. An influential outlier was considered a study whose removal changed the magnitude of the pooled effect by >10%.

BMI, body mass index; CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; MD, mean difference; mnth, month; MUFA, monounsaturated fatty acids; n/a, not applicable; OB, obese; OW, overweight; Pro, protein

Supplemental Table S6: Sensitivity analyses of the use of correlation coefficients of 0.25 and 0.75 for crossover trials

	MD (95% CI), P-value I^2, P-value		
	Correlation Coefficient used in the Primary Analysis	Correlation Coefficient used in Sensitivity Analyses	
Outcome (no. crossover trials/total)	**0.5**	**0.25**	**0.75**
Body Weight, kg (6*/32)	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.51	-0.63 [-0.84, -0.42], P<0.01 0.00%, P=0.51	-0.63 [-0.84, -0.43], P<0.01 0.00%, P=0.51
BMI, kg/m^2 (3/18)	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.91	-0.26 [-0.36, -0.16], P<0.01 0.00%, P=0.90	-0.26 [-0.35, -0.17], P<0.01 0.00%, P=0.90
Body Fat, % (3*/10)	-0.01 [-0.58, 0.56], P=0.98 65%, P<0.01	-0.00 [-0.58, 0.58], P=0.99 64%, P<0.01	-0.02 [-0.57, 0.54], P=0.96 66%, P<0.01
Waist Circumference, cm (2*/18)	-0.46 [-1.05, 0.14], P=0.13 62%, P<0.01	-0.44 [-1.04, 0.15], P=0.14 60%, P<0.01	-0.46 [-1.07, 0.14], P=0.13 65%, P<0.01
Waist-to-hip Ratio (0/6)	-0.00 [-0.01, 0.00], P=0.27 0.00%, P=0.52	n/a	n/a
Sagittal Abdominal Diameter, cm (0/6)	-0.09 [-0.34, 0.16], P=0.48 0.00%, P=0.92	n/a	n/a

* one of these crossover trials, however, did not require the use of a correlation coefficient as complete data was available

BMI, body mass index; CI, confidence interval; MD, mean difference; no., number

Supplement Table S7. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on body weight (kg)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	24.1 – 37.1 kg/m^2	32	2448	-0.04 (-0.13, 0.06)	0.00%	0.454
Follow-up	3 – 72 wks	32	2448	0.01 (-0.01, 0.02)	0.00%	0.314
Dose Pasta	1.75 – 7.0 serv/wk	11	740	0.29 (-0.91, 1.50)	0.00%	0.595
GI[3]	21 - 72	31	2383	-0.01 (-0.04, 0.02)	0.45%	0.482
Difference in GI[2]	4.9 - 40.2	31	2383	-0.00 (-0.04, 0.03)	2.75%	0.811
Fiber[3]	8.0 - 39.4g/d	27	1851	0.02 (-0.01, 0.06)	0.00%	0.221
Change in Fiber[4]	-7.5 - +13.8g/d	17	1571	-0.02 (-0.07, 0.04)	23.13%	0.595
Difference in Fiber[2]	-8.5 - +15.3g/d	27	1851	-0.01 (-0.06, 0.04)	0.00%	0.701
Saturated Fat[3]	5.1 - 12.5%	14	1396	-0.05 (-0.30, 0.20)	34.32%	0.648
Change in Saturated Fat[4]	-8.2 - -1.2%	12	1309	-0.01 (-0.29, 0.28)	46.27%	0.965
Difference in Saturated Fat[2]	-2.0 - +2.3%	14	1396	-0.34 (-1.08, 0.39)	32.80%	0.329
CHO[3]	37.2 - 68.0%	30	2345	-0.01 (-0.05, 0.03)	0.00%	0.541
Change in CHO[4]	-7.2 - +10.1%	19	2046	-0.07 (-0.12, -0.01)	0.00%	**0.016**
Difference in CHO[2]	-11.1 - +5.4%	30	2345	0.02 (-0.05, 0.10)	0.00%	0.547
Protein[3]	13.3 - 26.1%	30	2345	0.04 (-0.04, 0.12)	0.00%	0.332

Change in Protein[4]	-0.5 - +8.6%	19	2046	0.15 (0.03, 0.27)	0.00%	**0.017**
Difference in Protein[2]	-2.5 - +3.4%	30	2345	-0.18 (-0.38, 0.02)	0.00%	0.069
Fat[3]	16.0 - 37.7%	30	2345	0.01 (-0.05, 0.06)	0.00%	0.852
Change in Fat[4]	-12.2 - +5.4%	19	2046	0.05 (-0.02, 0.12)	17.73%	0.122
Difference in Fat[2]	-4.4 - +10.6%	30	2345	0.02 (-0.05, 0.09)	0.00%	0.610

1 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 body weight on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in body weight. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.

2 Difference in diet variable between the intervention and control arms

3 Intake at the end of study in the intervention arm

4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index

Supplemental Table S8. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on BMI (kg/m^2)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	26.3 – 37.1 kg/m^2	18	1038	-0.01 (-0.06, 0.03)	0.00%	0.559
Follow-up	4 – 24 wks	18	1038	0.00 (-0.01, 0.02)	0.00%	0.559
Dose Pasta*	2.33 – 7.0 serv/wk	4	143			
GI[3]	39.8 - 72	17	973	0.01 (-0.01, 0.03)	0.00%	0.153
Difference in GI[2]	7.0 - 40.2	17	973	-0.01 (-0.03, 0.01)	0.00%	0.275
Fiber[3]	8.0 - 39.4g/d	16	935	0.01 (-0.01, 0.02)	0.00%	0.366
Change in Fiber[4]	-1.8 - +12.4g/d	10	758	0.00 (-0.03, 0.03)	0.00%	0.846
Difference in Fiber[2]	-4.9 - +13.0g/d	16	935	-0.00 (-0.02, 0.02)	0.00%	0.821
Saturated Fat*[3]	7.6 - 12.5%	7				
Change in Saturated Fat*[4]	-2.4 - -1.2%	6				
Difference in Saturated Fat*[2]	-1.0 - +2.3%	7				
CHO[3]	37.2 - 68.0%	16	935	-0.00 (-0.02, 0.01)	0.00%	0.594
Change in CHO[4]	-5.6 - +3.2%	10	758	-0.02 (-0.06, 0.02)	0.00%	0.325
Difference in CHO[2]	-11.1 - +2.0%	16	935	-0.01 (-0.03, 0.02)	0.00%	0.531
Protein[3]	13.9 – 22.8%	16	935	0.00 (-0.04, 0.04)	0.00%	0.905

Change in Protein[4]	-0.2 - +3.0%	10	758	0.01 (-0.11, 0.14)	0.00%	0.786
Difference in Protein[2]	-2.5 - +3.4%	16	935	-0.03 (-0.12, 0.06)	0.00%	0.441
Fat[3]	16.0 - 37.7%	16	935	0.00 (-0.02, 0.02)	0.00%	0.717
Change in Fat[4]	-4.8 - +5.4%	10	758	0.02 (-0.02, 0.06)	0.00%	0.240
Difference in Fat[2]	-4.4 - +10.6%	16	935	0.01 (-0.01, 0.04)	0.00%	0.299

*For Dose, there were <10 trials so subgroup analyses were not performed.

1 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 BMI on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in BMI. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.

2 Difference in diet variable between the intervention and control arms

3 Intake at the end of study in the intervention arm

4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index; wk, week

Supplemental Table S9. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on body fat (%)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	26.3 – 36.0 kg/m^2	10	285	-0.02 (-0.26, 0.23)	67.69%	0.890
Follow-up	4 – 24 wks	10	285	0.06 (-0.06, 0.17)	66.00%	0.303
Dose Pasta*	2.33 - 3.33 serv/wk	3	96			
GI[3]	39.8 - 72	10	285	0.12 (-0.00, 0.25)	52.21%	0.053
Difference in GI[2]	7.0 - 40.2	10	285	-0.09 (-0.15, -0.03)	19.39%	**0.008**
Fiber*[3]	8.0 - 39.4g/d	8	228			
Change in Fiber*[4]	-1.8 - +12.4g/d	4	87			
Difference in Fiber*[2]	-4.9 - +13.0g/d	8	228			
Saturated Fat*[3]	7.6 - 12.5%	3	93			
Change in Saturated Fat*[4]	-2.4 - -1.2%	2	51			
Difference in Saturated Fat*[2]	-1.0 - +2.3%	3	93			
CHO*[3]	37.2 - 68.0%	9	247			
Change in CHO*[4]	-5.6 - +3.2%	4	87			
Difference in CHO*[2]	-11.1 - +2.0%	9	247			
Protein*[3]	13.9 – 22.8%	9	247			

Change in Protein*[4]	-0.2 - +3.0%	4	87			
Difference in Protein*[2]	-2.5 - +3.4%	9	247			
Fat*[3]	16.0 - 37.7%	9	247			
Change in Fat*[4]	-4.8 - +5.4%	4	87			
Difference in Fat*[2]	-4.4 - +10.6%	9	247			

*There were <10 trials so subgroup analyses were not performed.

1 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 body fat on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in body fat. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.
2 Difference in diet variable between the intervention and control arms
3 Intake at the end of study in the intervention arm
4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index; wk, week

Supplemental Table S10. Continuous meta-regression analysis for the effect of pasta in the context of low-GI dietary patterns on waist circumference (cm)[1]

Subgroup	Range	No. trials	N	β (95% CI)	Residual I^2	P value
Baseline BMI	26.3 – 36.0 kg/m^2	18	1380	0.10 (-0.13, 0.32)	59.02%	0.372
Follow-up	4 – 52 wks	18	1380	0.05 (-0.03, 0.13)	60.50%	0.225
Dose Pasta*	3.0 – 3.33 serv/wk	2	63			
GI[3]	41.2 – 72.0	17	1315	-0.01 (-0.12, 0.10)	66.00%	0.841
Difference in GI[2]	4.9 – 32.9	17	1315	-0.02 (-0.10, 0.06)	64.51%	0.596
Fiber[3]	8.0 - 39.4g/d	15	1258	0.05 (-0.05, 0.14)	59.60%	0.342
Change in Fiber[4]	-1.8 - +13.8g/d	13	1176	0.01 (-0.17, 0.20)	70.59%	0.861
Difference in Fiber[2]	-4.9 - +15.3g/d	15	1258	-0.00 (-0.14, 0.13)	64.86%	0.939
Saturated Fat*[3]	6.1 – 10.1%	8	604			
Change in Saturated Fat*[4]	-7.6 - -1.2%	7	562			
Difference in Saturated Fat*[2]	-2.0 - +2.3%	8	604			
CHO[3]	38.5 - 58.6%	16	1277	-0.11 (-0.19, -0.04)	27.06%	**0.007**
Change in CHO[4]	-7.2 - +10.1%	13	1176	-0.09 (-0.23, 0.04)	57.19%	0.148
Difference in CHO[2]	-11.1 - +5.4%	16	1277	-0.01 (-0.18, 0.17)	60.81%	0.947
Protein[3]	13.9 – 26.1%	16	1277	0.20 (0.01, 0.38)	43.92%	**0.038**

Change in Protein[4]	-0.3 - +8.6%	13	1176	0.19 (-0.09, 0.46)	63.38%	0.165
Difference in Protein[2]	-2.5 - +3.4%	16	1277	-0.14 (-0.66, 0.38)	62.79%	0.570
Fat[3]	22.0 - 37.2%	16	1277	0.10 (-0.05, 0.26)	53.48%	0.185
Change in Fat[4]	-12.2 - +5.4%	13	1176	0.08 (-0.07, 0.24)	61.15%	0.274
Difference in Fat[2]	-4.4 - +10.6%	16	1277	0.09 (-0.06, 0.25)	47.17%	0.220

*There were <10 trials so subgroup analyses were not performed.

1 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 waist circumference on the pasta/low-GI intervention as the subgroup variable increases, and a negative β -coefficient implies a decrease in waist circumference. Residual I^2 reports inter-study heterogeneity not explained by the subgroup and was estimated using the Cochran's Q statistic.

2 Difference in diet variable between the intervention and control arms

3 Intake at the end of study in the intervention arm

4 Change in intake from end of study from baseline in intervention arm

BMI, body mass index; CHO, carbohydrate; GI, glycemic index; wk, week

Supplementary Table S11. Post-hoc piecewise linear continuous dose-response meta-regression analyses for the effect of pasta intake in the context of low-GI dietary patterns on body weight (kg)

Dose threshold of servings of pasta consumed per week	Dose ranges of servings of pasta consumed per week	β (95% CIs) *	Residual I^2 †	*p*-value
3.0	≤3.0	-0.70 (–3.27, 1.86)	0.00%	0.890
	>3.0	0.91 (–0.89, 2.70)		
3.33	≤3.33	0.05 (–1.80, 1.89)	0.00%	0.518
	>3.33	0.44 (–1.75, 2.63)		
3.5	≤3.5	0.09 (–1.65, 1.82)	0.00%	0.888
	>3.5	0.46 (–1.89, 2.81)		

* β is the slope derived from the piecewise linear meta-regression analyses and represents the treatment effect on body weight for doses above and below each dose-threshold representing servings of pasta consumption per week; † The residual I^2 value indicates heterogeneity unexplained by each dose-threshold.

Supplementary Table S12: GRADE assessment of study quality

Quality assessment*							№ of patients		Effect	Quality Importance
№ of studies	**Design**	**Risk of bias**	**Inconsistency**	**Indirectness**	**Imprecision**	**Publication Bias**	**Pasta/LGI diet**	**Higher GI diet**	**Absolute (95% CI)**	
Body Weight (follow up: median 12 weeks)										
32	randomised trials	not serious	not serious	serious [a]	not serious	none	1294	1228	MD **-0.63 kg** (-0.84 to -0.42)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness
BMI (follow up: median 12 weeks)										
18	randomised trials	not serious	not serious	serious [a]	not serious	none	551	538	MD **-0.26 kg/m^2** (-0.36 to -0.16)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness
Waist Circumference (follow up: median 12 weeks)										

Quality assessment*							№ of patients		Effect	Quality Importance
№ of studies	**Design**	**Risk of bias**	**Inconsistency**	**Indirectness**	**Imprecision**	**Publication Bias**	**Pasta/LGI diet**	**Higher GI diet**	**Absolute (95% CI)**	
18	randomised trials	not serious	serious [b]	serious [a]	not serious	none	717	624	MD **-0.46 cm** (-1.05 to 0.14)	⊕⊕◯◯ LOW [a,b] Due to downgrade for inconsistency and indirectness
Body Fat (follow up: median 12 weeks)										
10	randomised trials	not serious	serious [c]	serious [a]	not serious	none	176	165	MD **-0.01 %** (-0.58 to 0.56)	⊕⊕◯◯ LOW [a,c] Due to downgrade for inconsistency and indirectness
Waist-to-hip Ratio (follow up: median 12 weeks)										

Quality assessment*							№ of patients		Effect	Quality Importance
№ of studies	Design	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Pasta/LGI diet	Higher GI diet	Absolute (95% CI)	
6	randomised trials	not serious	not serious	serious [a]	not serious	none [d]	223	222	MD **-0.00** (-0.01 to 0.00)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness
Sagittal Abdominal Diameter (follow up: median 26 weeks)										
3	randomised trials	not serious	not serious	serious [a]	not serious	none [d]	237	214	MD **-0.09 cm** (-0.34 to 0.16)	⊕⊕⊕◯ MODERATE[a] Due to downgrade for indirectness

CI, Confidence interval; GI, glycemic index; LGI, low glycemic index; MD, Mean difference

*All outcomes started with high quality evidence since all studies were randomized controlled trials. Risk of Bias –We rated down for risk of bias if the majority of studies were considered to be at high risk of bias. Inconsistency – We assessed inconsistency using I^2 estimates where an I^2 of 50% or higher indicates substantial heterogeneity. I^2 is the percentage of variability in the treatment estimates that is attributable to heterogeneity between studies. We rated down for inconsistency if there was substantial heterogeneity that was unexplained by any a priori sensitivity or subgroup analyses. Indirectness – We rated down for indirectness if there were factors present relating to the population, interventions, and outcomes that limited the generalizability of the results. Imprecision – We rated down for imprecision if the 95% confidence interval (95% CI) crossed the minimally important difference (MID) for harm. MIDs used for each outcome are: 0.5kg for body weight (based on Johnston et al. JAMA 2014;312(9):923-33); 0.2kg/m^2 for BMI; 2.0cm for waist circumference; 2.0% for body fat; 0.02 for waist-to-hip ratio; 2.0cm for sagittal abdominal diameter.

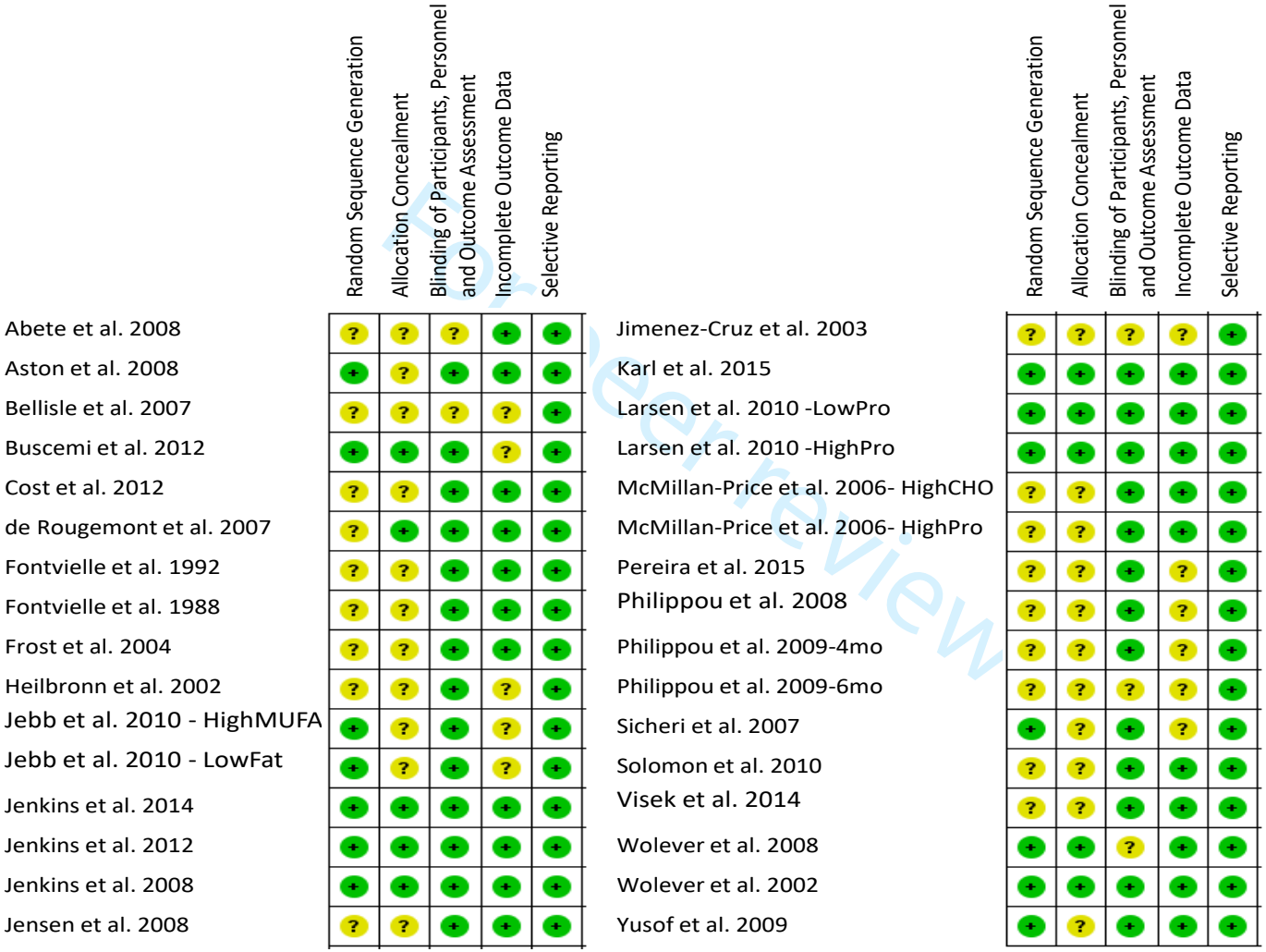
a. Downgrade for serious indirectness, since most of the available trials did not quantify the amount of pasta consumed in the context of the low-GI dietary patterns

b. Downgrade for serious inconsistency, as there was evidence of substantial inter-study (I^2=62%, P-heterogeneity<0.001), which could not be explained

c. Downgrade for serious inconsistency, as there was evidence of substantial inter-study (I^2= 65%, P-heterogeneity=0.003), which could not be explained

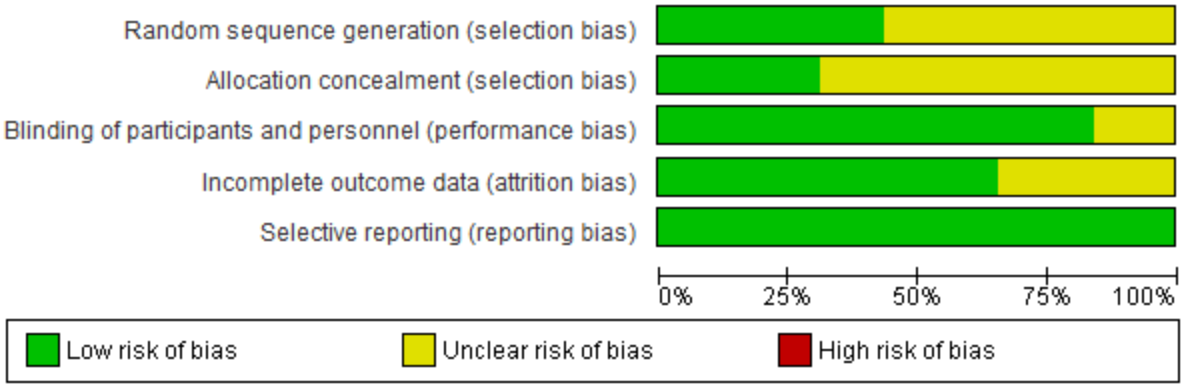
d. No downgrade for publication bias, as publication bias could not be assessed due to lack of power for assessing funnel plot asymmetry and small study effects (<10 trials included in the meta-analysis)

Supplemental Figures

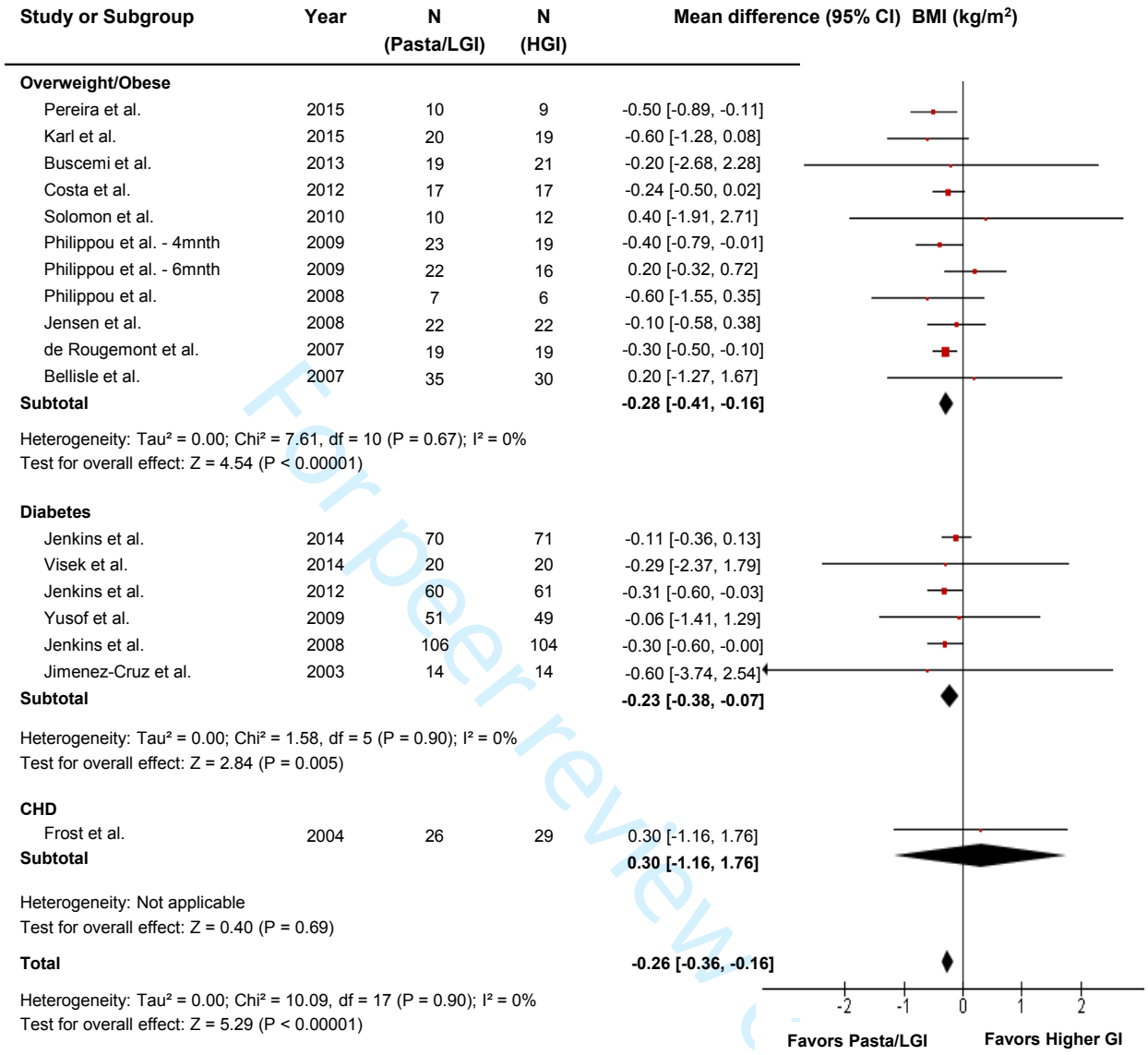


Supplemental Figure S1: Cochrane risk of bias summary for all included trials

Summary of risk of bias ratings for each individual study included in the meta-analysis.



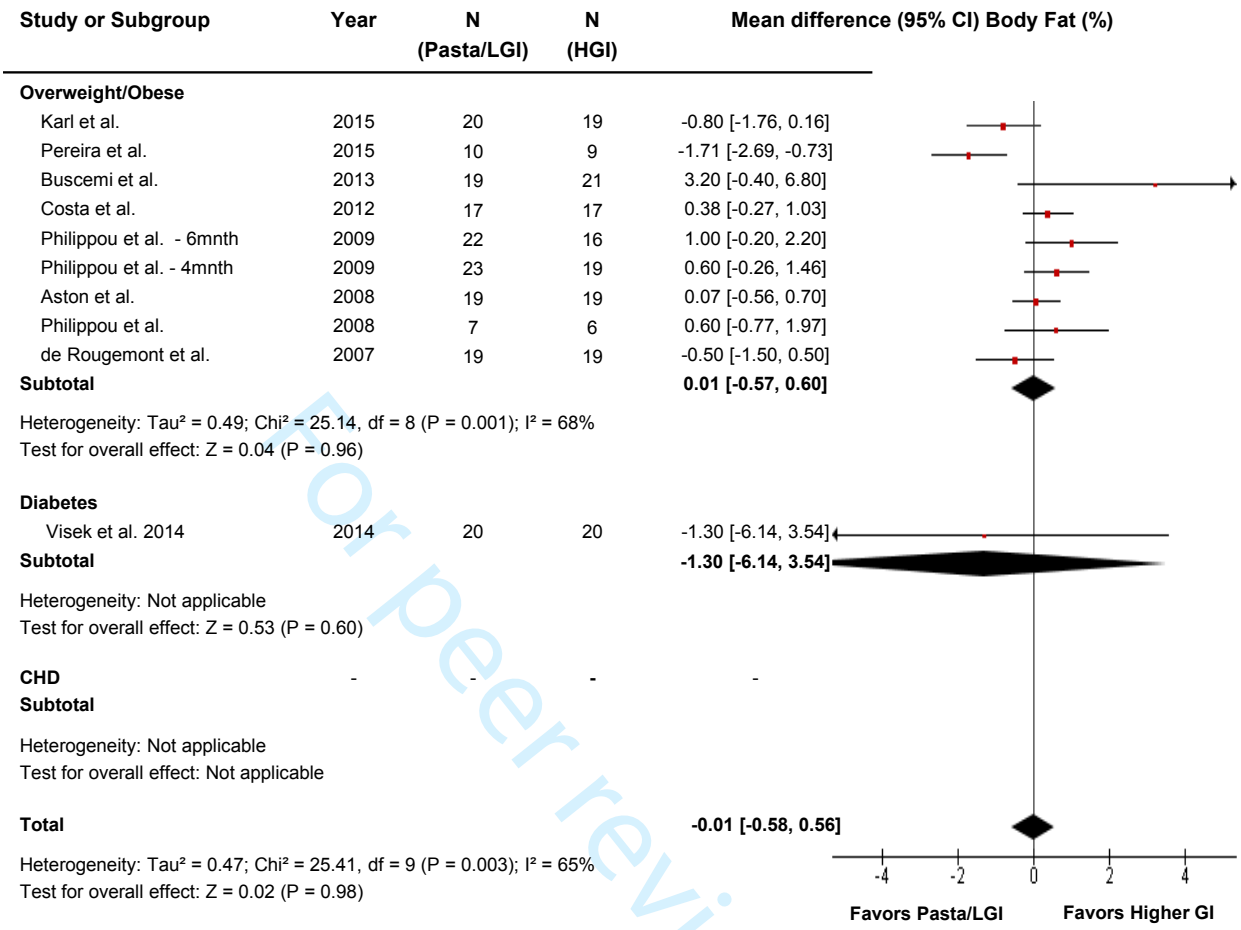
Supplemental Figure S2: Risk of bias proportion graph for all included trials



Supplemental Figure S3: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on BMI (kg/m^2) (n= 1038)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.

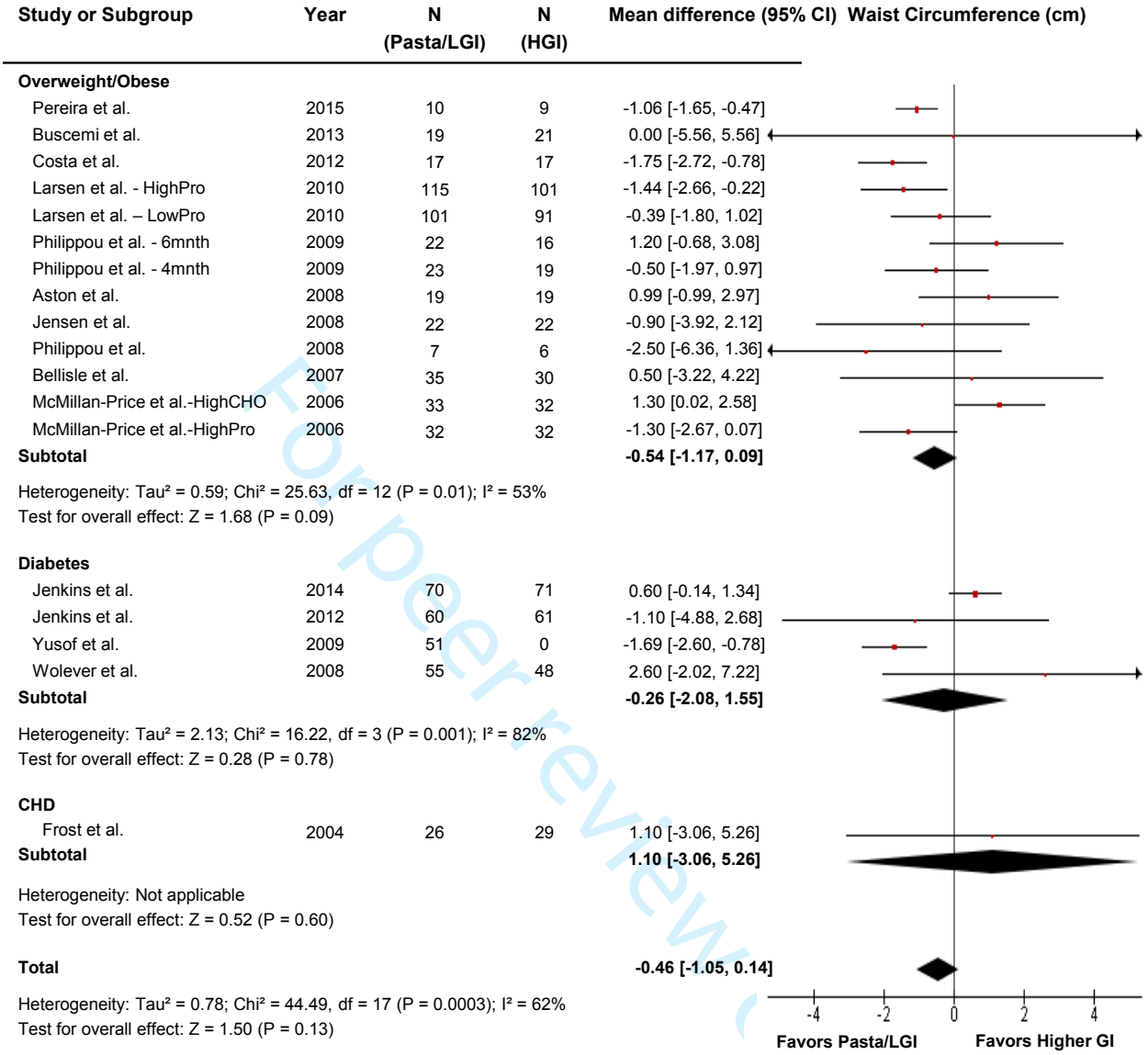
BMI, body mass index; CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet



Supplemental Figure S4: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body fat (%) (n= 285)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

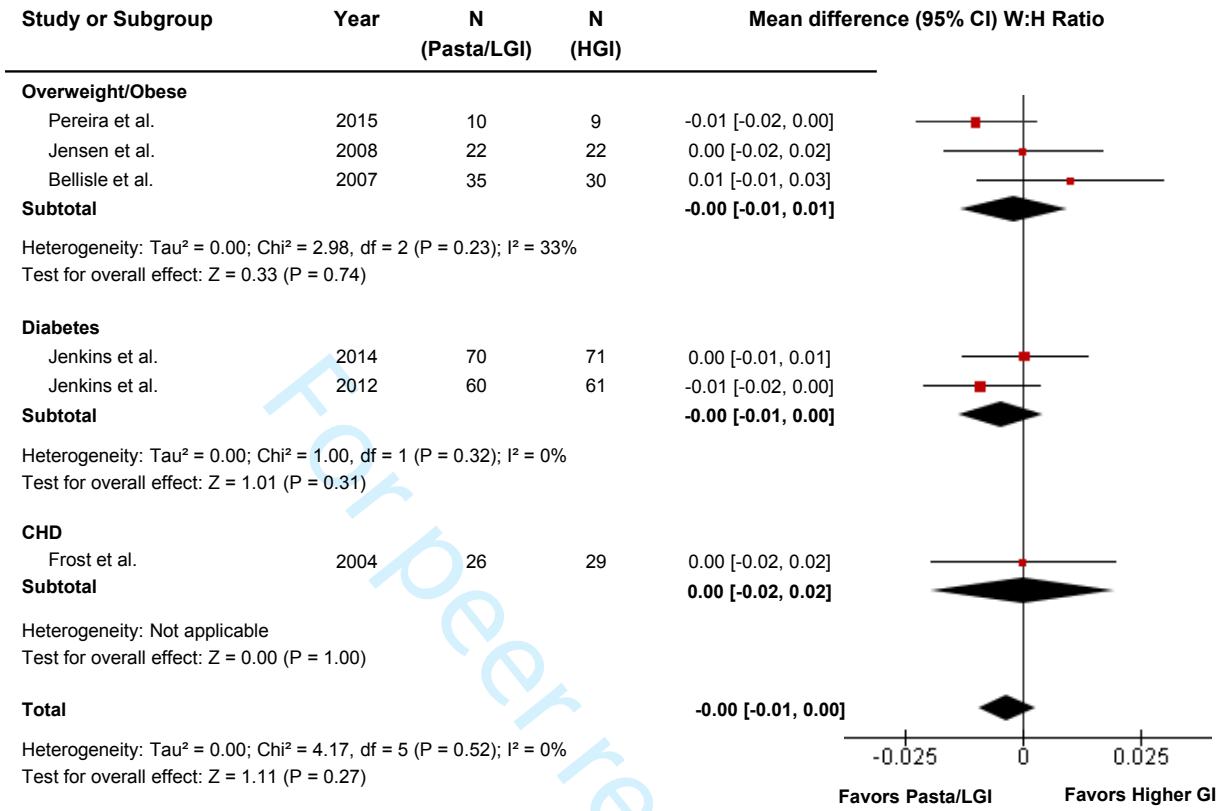
CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet



Supplemental Figure S5: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on waist circumference (cm) (n = 1380)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

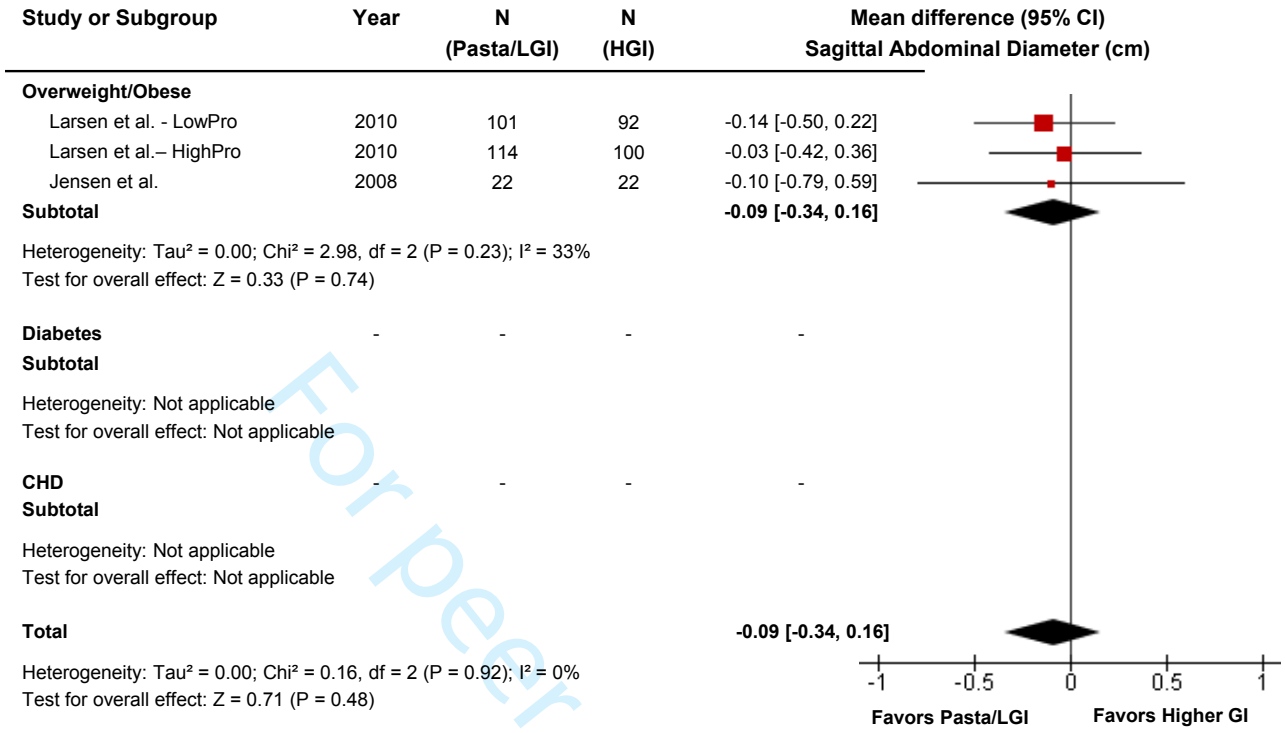
CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; Pro, protein



Supplemental Figure S6: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on waist-to-hip ratio (n = 445)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

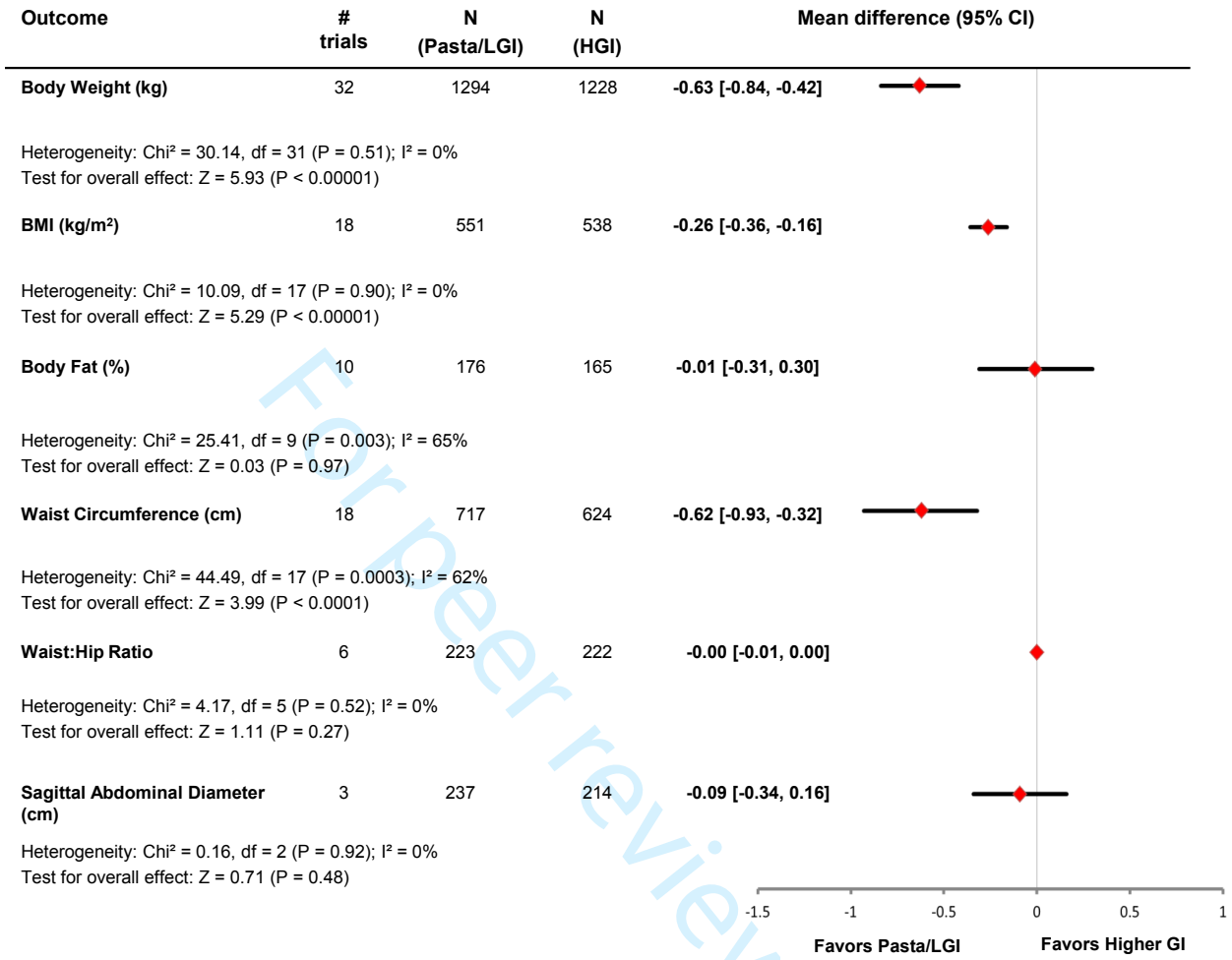
CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet



Supplemental Figure S7: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on sagittal abdominal diameter (cm) (n = 478)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

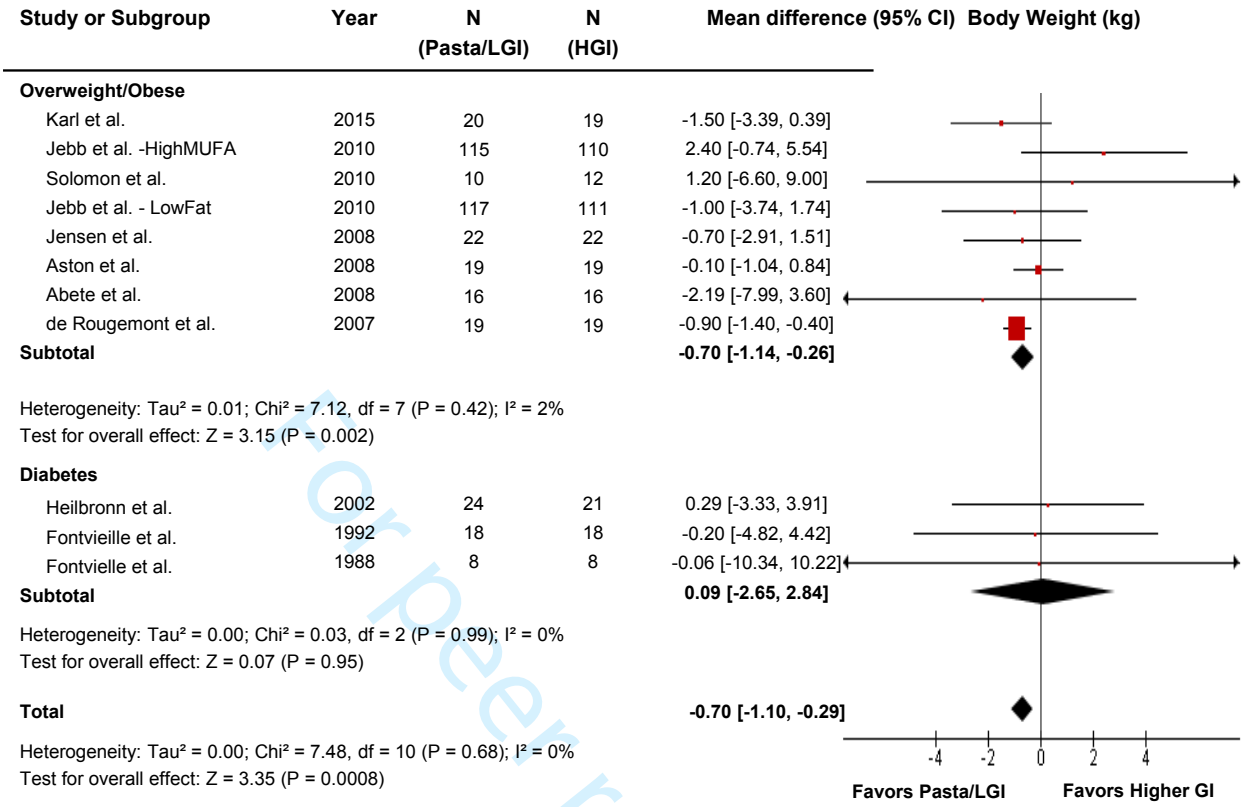
CHD, coronary heart disease; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; Pro, protein



Supplemental Figure S8: Forest plot of randomized controlled trials investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg) using fixed effects models (n = 2448)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance fixed effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of P<0.10 and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and ≥75% considerable heterogeneity.

CHD, coronary heart disease; CHO, carbohydrate; CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids; Pro, protein.

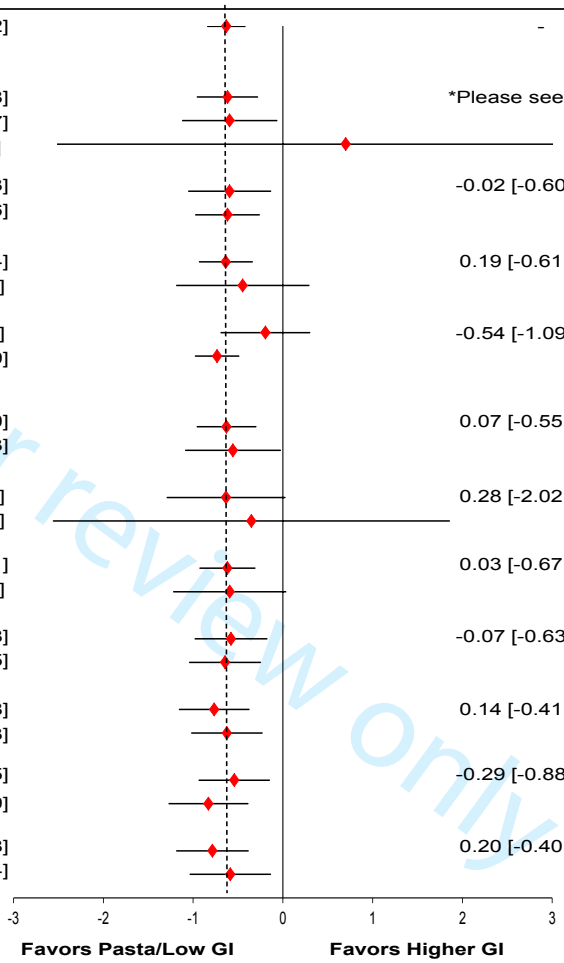


Supplemental Figure S9: Forest plot of randomized controlled trials which contain data for approximating pasta intake which are investigating the effects of pasta in the context of low-GI dietary patterns on body weight (kg) (n = 740)

Data are expressed as mean differences represented by a square and 95% CIs by the line through the square. 95% CIs exceeding the plot's bounds are represented by an arrowhead. Pooled effect estimates are represented by diamonds and were estimated with the use of generic inverse variance random effects models. Between-study heterogeneity was detected with the use of the Cochran's Q statistic at a significance level of $P<0.10$ and quantified with the use of the I^2 statistic where $I^2 \geq 50\%$ is considered to be evidence of substantial heterogeneity and $\geq 75\%$ considerable heterogeneity.

CI, confidence interval; HGI, higher glycemic index diet; GI, glycemic index; LGI, low glycemic index diet; MUFA, monounsaturated fatty acids

Subgroup	Level	No. comparisons	N	Mean difference [95% CI] in body weight [kg] Within subgroups	Between subgroups	Residual I^2	P-value
Total		32	2448	-0.63 [-0.84, -0.42]	-	-	-
Metabolic Phenotype	OW/OB	21	1613	-0.62 [-0.95, -0.28]	*Please see legend	2.86%	0.889
	Diabetes	10	780	-0.59 [-1.12, -0.07]			
	CHD	1	55	0.70 [-4.89, 6.29]			
Baseline BMI	≤30kg/m²	14	972	-0.59 [-1.05, -0.13]	-0.02 [-0.60, 0.56]	0.24%	0.934
	>30kg/m²	18	1476	-0.62 [-0.97, -0.26]			
Design	Parallel	26	2352	-0.64 [-0.93, -0.34]	0.19 [-0.61, 0.98]	0.00%	0.632
	Crossover	6	96	-0.45 [-1.18, 0.29]			
Energy Balance	Negative	9	459	-0.19 [-0.69, 0.30]	-0.54 [-1.09, 0.01]	0.00%	**0.056**
	Neutral	23	1989	-0.73 [-0.98, -0.49]			
	Positive	0	0				
Follow-up	<24-weeks	24	1065	-0.63 [-0.96, -0.30]	0.07 [-0.55, 0.70]	0.35%	0.814
	≥24-weeks	8	1383	-0.56 [-1.09, -0.03]			
Dose Pasta	<3.3 serv/wk	5	378	-0.63 [-1.29, 0.02]	0.28 [-2.02, 2.58]	0.00%	0.788
	≥3.3serv/wk	6	362	-0.35 [-2.56, 1.86]			
Test GI┼	≤55	23	1208	-0.62 [-0.93, -0.31]	0.03 [-0.67, 0.72]	2.99%	0.941
	>55	8	1175	-0.59 [-1.22, 0.03]			
GI btw trt┼	≤12.6	16	1787	-0.58 [-0.98, -0.18]	-0.07 [-0.63, 0.50]	2.60%	0.810
	>12.6	15	596	-0.64 [-1.04, -0.25]			
Test Fibre‡	≤28.0g/d	14	861	-0.77 [-1.15, -0.38]	0.14 [-0.41, 0.69]	0.00%	0.604
	>28.0g/d	13	990	-0.62 [-1.02, -0.23]			
Fibre within test‖	≤4.63g/d	9	867	-0.54 [-0.93, -0.15]	-0.29 [-0.88, 0.30]	18.80%	0.316
	>4.63g/d	8	704	-0.83 [-1.27, -0.39]			
Fibre btw trt‡	≤2.40g/d	15	1005	-0.79 [-1.19, -0.38]	0.20 [-0.40, 0.80]	0.00%	0.500
	>2.40g/d	12	846	-0.59 [-1.03, -0.14]			

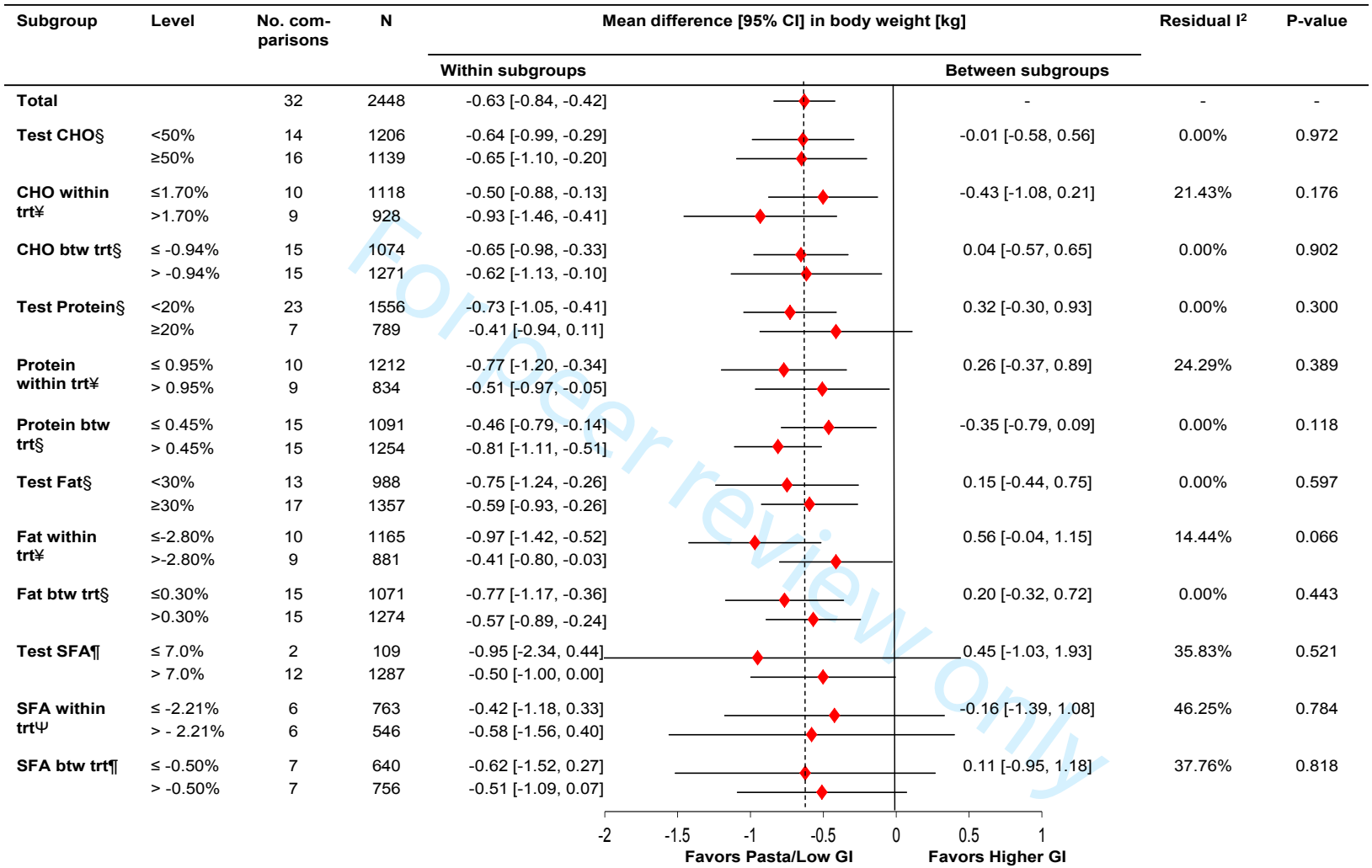


Supplemental Figure S10: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body weight (kg) (n = 2448)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body weight. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes.

BMI, body mass index; btw, between; CHD, coronary heart disease; CI, confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; serv, serving; trt, treatment; wk, week.

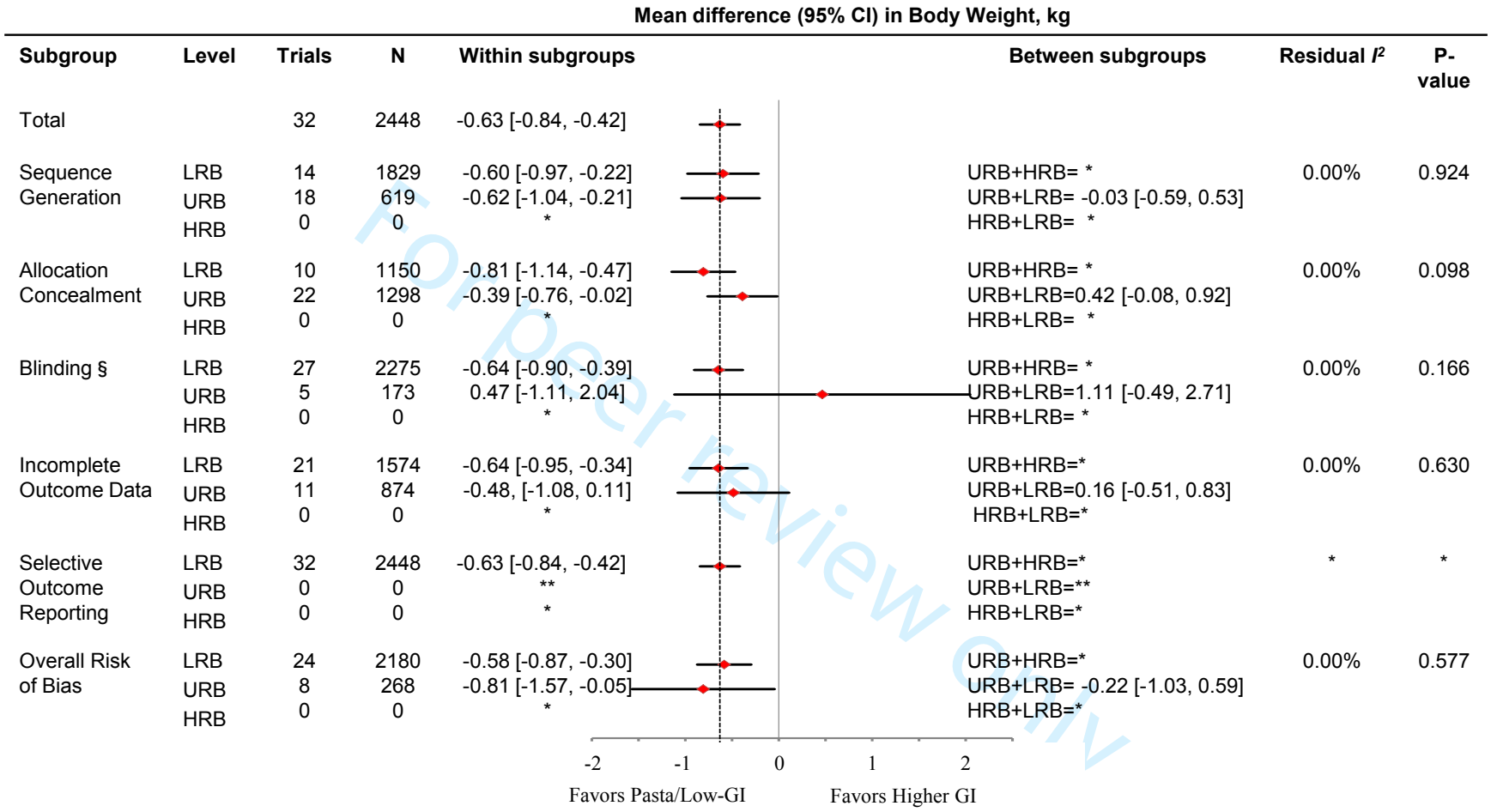
*Pairwise between-subgroup mean differences (95% CIs) for Metabolic Phenotype were as follows: 0.02kg (-0.08, 0.65) (1 vs. 2) to 1.32kg (-4.28, 6.91) (1 vs. 3) to -1.29kg (-6.91, 4.32) (2 vs. 3).
┼ data available on 31 studies
‖ data available on 17 studies
ǂ data available on 27 studies



Supplemental Figure S11: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body weight (kg) continued (n = 2448)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body weight. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes.

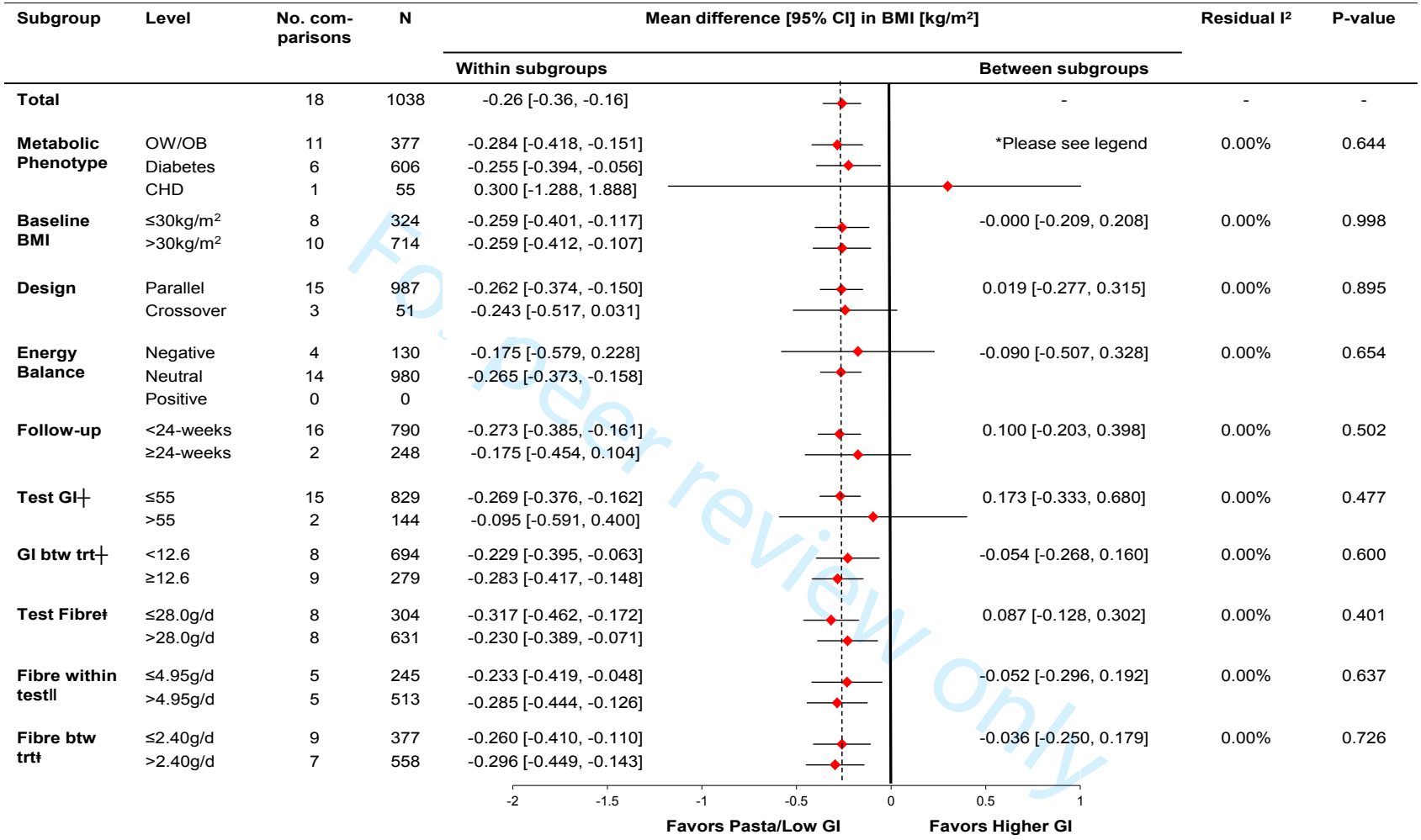
BMI, body mass index; btw, between; CI, confidence interval; GI, glycemic index (glucose scale); trt, treatment.
§ data available on 30 studies
¥ data available on 19 studies
¶ data available on 14 studies
Ψ data available on 12 studies



Supplemental Figure S12: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on body weight (kg) (n = 2448)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body weight. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

LRB=Low Risk of Bias, URB= Unclear Risk of Bias, HRB=High Risk of Bias. *Within and between subgroup analysis could not be performed against HRB since no values for HRB subgroup. ** no values for URB subgroup. § Blinding of Participants, Personnel, and Outcome Assessors.



Supplemental Figure S13: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body mass index, BMI (kg/m^2) (n = 1038)

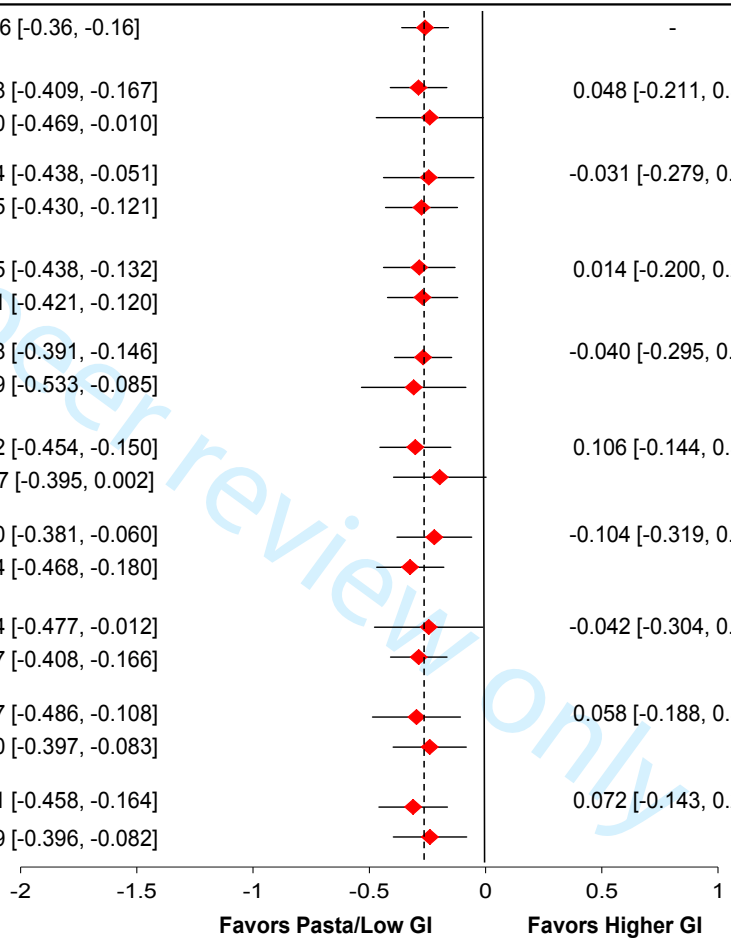
The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on BMI. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study

heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for dose (n=3 trials), therefore analyses were not performed.

*Pairwise between-subgroup mean differences (95% CIs) for Metabolic Phenotype were as follows: 0.059kg/m^2 (-0.156, 0.274) (1 vs. 2) to 0.584kg/m^2 (-1.009, 2.178) (1 vs. 3) to -0.525kg/m^2 (-2.122, 1.072) (2 vs. 3).
┼ data available on 17 studies
ǂ data available on 16 studies
‖ data available on 10 studies

BMI, body mass index; btw, between; CI, confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; trt, treatment.

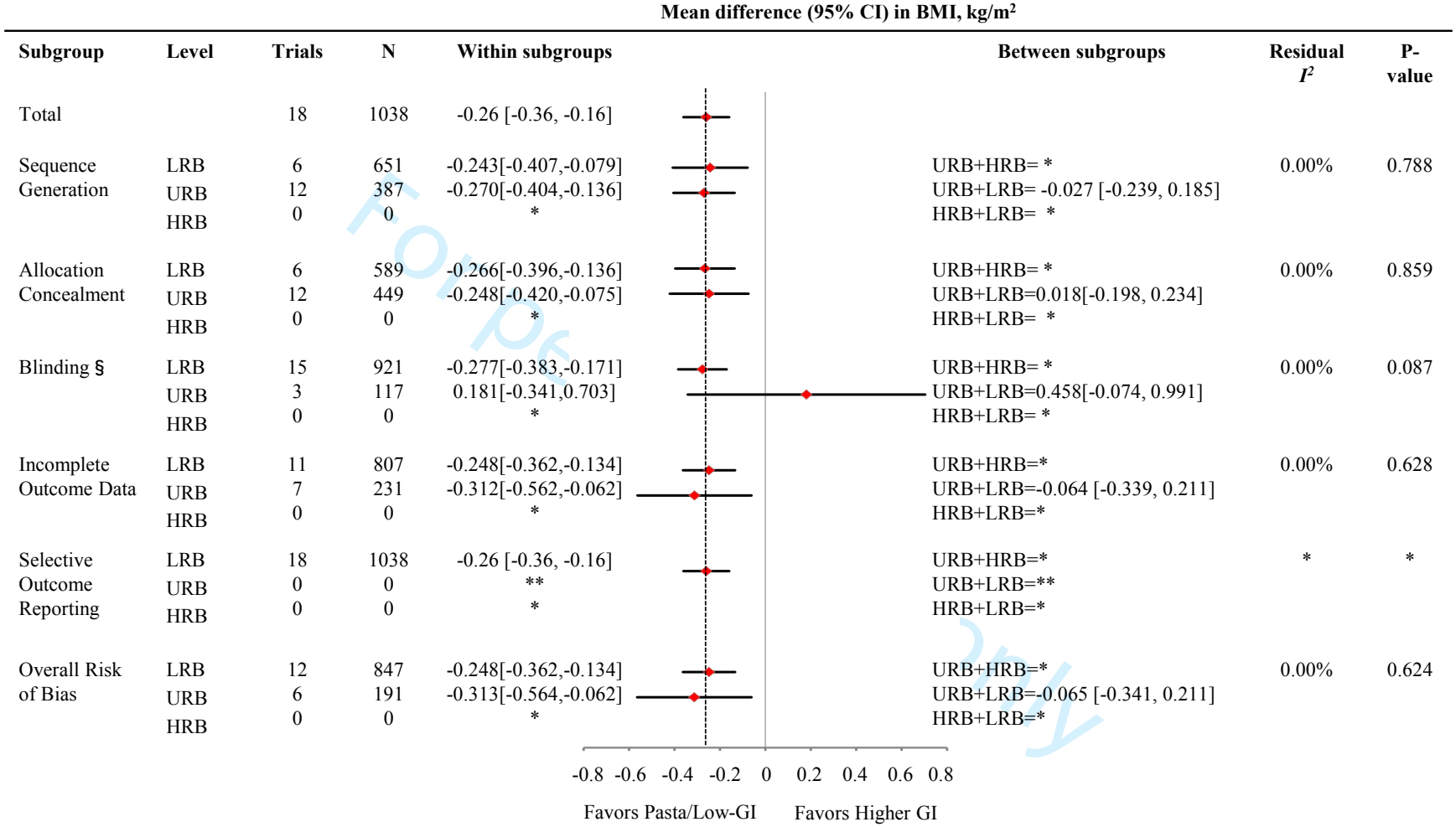
Subgroup	Level	No. comparisons	N	Mean difference [95% CI] in BMI [kg/m²]		Residual I²	P-value
				Within subgroups	Between subgroups		
Total		18	1038	-0.26 [-0.36, -0.16]	-	-	-
Test CHO§	< 50%	9	659	-0.288 [-0.409, -0.167]	0.048 [-0.211, 0.308]	0.00%	0.696
	≥ 50%	7	276	-0.240 [-0.469, -0.010]			
CHO within trt¥	≤ 1.35%	5	436	-0.244 [-0.438, -0.051]	-0.031 [-0.279, 0.217]	0.00%	0.781
	> 1.35%	5	322	-0.275 [-0.430, -0.121]			
CHO btw trt§	≤ -1.75%	8	657	-0.285 [-0.438, -0.132]	0.014 [-0.200, 0.229]	0.00%	0.888
	> -1.75%	8	278	-0.271 [-0.421, -0.120]			
Test Protein§	< 20%	13	590	-0.268 [-0.391, -0.146]	-0.040 [-0.295, 0.215]	0.00%	0.741
	≥ 20%	3	345	-0.309 [-0.533, -0.085]			
Protein within trt¥	≤ 1.04%	5	339	-0.302 [-0.454, -0.150]	0.106 [-0.144, 0.356]	0.00%	0.358
	> 1.04%	5	419	-0.197 [-0.395, 0.002]			
Protein btw trt§	≤ 0.45%	8	373	-0.220 [-0.381, -0.060]	-0.104 [-0.319, 0.112]	0.00%	0.319
	> 0.45%	8	562	-0.324 [-0.468, -0.180]			
Test Fat§	< 30%	6	176	-0.244 [-0.477, -0.012]	-0.042 [-0.304, 0.220]	0.00%	0.734
	≥ 30%	10	759	-0.287 [-0.408, -0.166]			
Fat within trt¥	≤ -2.60%	5	303	-0.297 [-0.486, -0.108]	0.058 [-0.188, 0.303]	0.00%	0.604
	> -2.60%	5	455	-0.240 [-0.397, -0.083]			
Fat btw trt§	≤ 0.95%	8	253	-0.311 [-0.458, -0.164]	0.072 [-0.143, 0.287]	0.00%	0.483
	> 0.95%	8	682	-0.239 [-0.396, -0.082]			



Supplemental Figure S14: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body mass index, BMI (kg/m^2) continued (n = 1038)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on BMI. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for test saturated fat (n=7 trials), saturated fat within treatment (n=6 trials) and saturated fat between treatments (n=7 trials), therefore analyses were not performed.
§ data available on 16 studies
¥ data available on 10 studies

BMI, body mass index; btw, between; CHO, carbohydrate; CI = confidence interval; GI, glycemic index (glucose scale); trt, treatment.



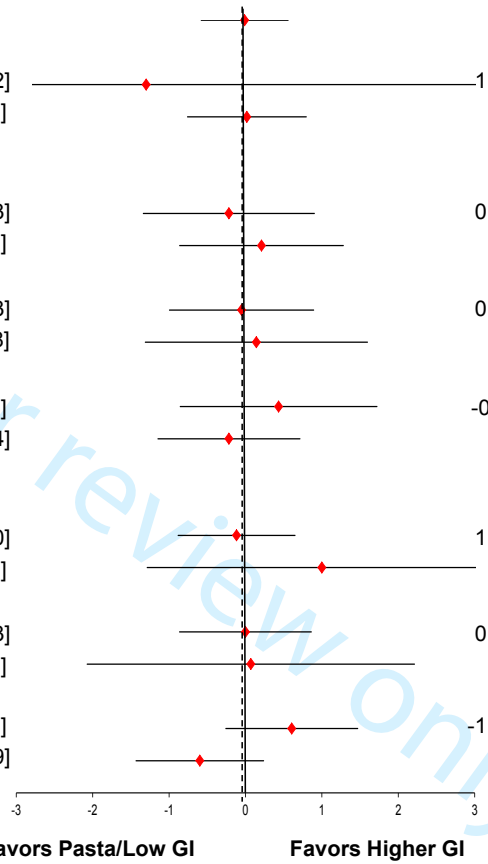
Supplemental Figure S15: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on body mass index, BMI (kg/m²) (n = 1038)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on BMI. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

*Within and between subgroup analyses could not be performed against HRB since no values for HRB subgroup.
** no values for URB subgroup.
§ Blinding of Participants, Personnel, and Outcome Assessors.

BMI, body mass index; CI, confidence interval; HRB, High Risk of Bias; GI, glycemic index; LRB, Low Risk of Bias; URB, Unclear Risk of Bias.

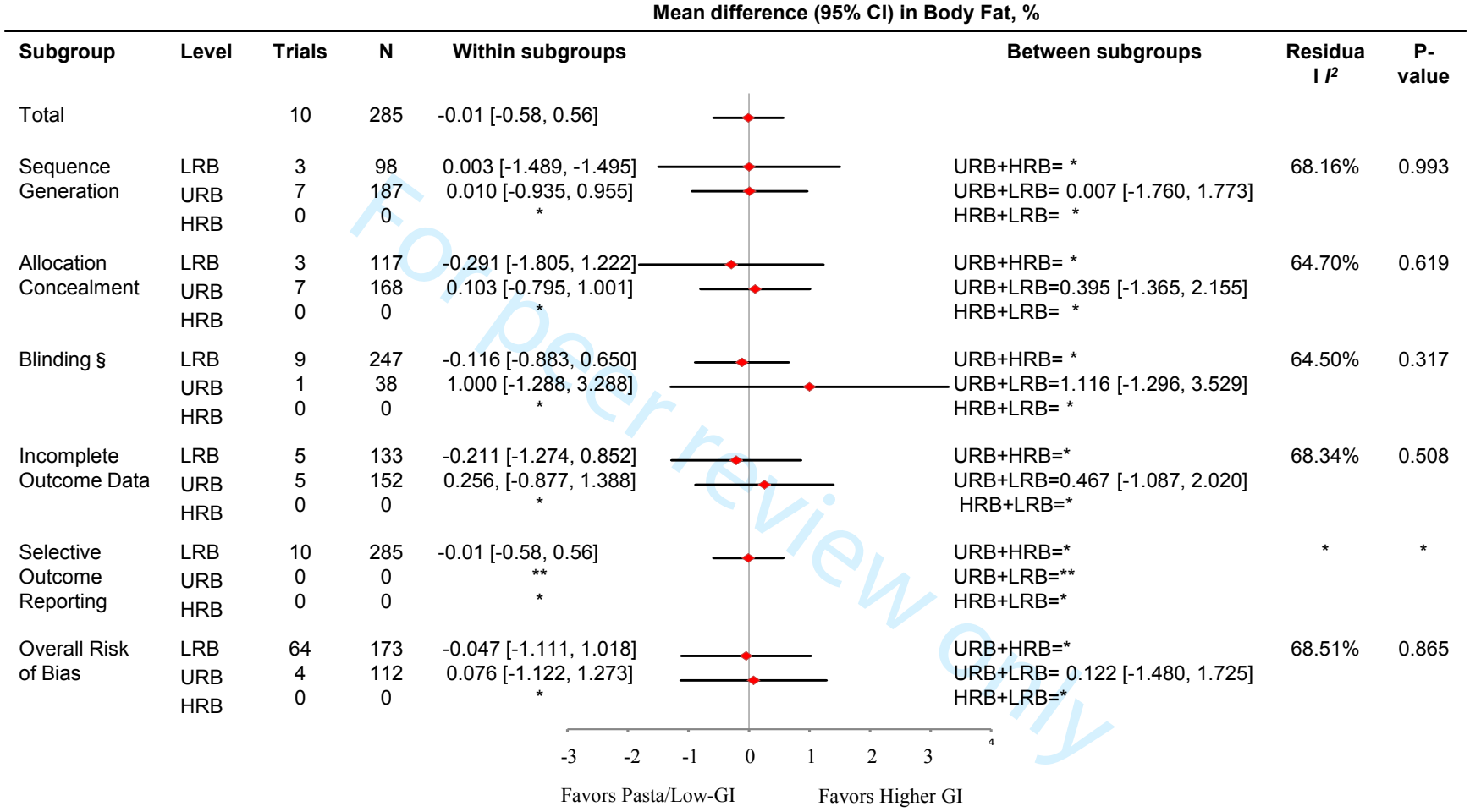
Subgroup	Level	No. comparisons	N	Mean difference [95% CI] in body fat [%] Within subgroups	Between subgroups	Residual I^2	P-value
Total		10	285	-0.01 [-0.58, 0.56]	-	-	-
Metabolic Phenotype	OW/OB	9	265	-1.300 [-7.782, 5.182]	1.319 [-5.210, 7.847]	68.17%	0.654
	Diabetes	1	20	0.019 [-0.760, 0.797]			
	CHD	0	0				
Baseline BMI	≤30kg/m^2	4	112	-0.217 [-1.338, 0.903]	0.427 [-1.125, 1.978]	67.72%	0.544
	>30kg/m^2	6	173	0.210 [-0.863, 1.282]			
Design	Parallel	7	229	-0.051 [-0.996, 0.893]	0.194 [-1.540, 1.929]	66.40%	0.803
	Crossover	3	56	0.143 [-1.312, 1.598]			
Energy Balance	Negative	4	130	0.434 [-0.856, 1.723]	-0.650 [-2.241, 0.940]	68.11%	0.373
	Neutral	6	155	-0.216 [-1.147, 0.714]			
	Positive						
Follow-up	<24-weeks	9	247	-0.116 [-0.883, 0.650]	1.116 [-1.300, 3.529]	64.50%	0.317
	≥24-weeks	1	38	1.000 [-1.288, 3.288]			
Test GI	≤55	9	266	-0.000 [-0.864, 0.863]	0.070 [-2.241, 2.382]	68.43%	0.946
	>55	1	19	0.070 [-2.074, 2.214]			
GI btw trt	≤14.1	5	152	0.606 [-0.258, 1.471]	-1.204 [-2.407, -0.008]	55.49%	0.050
	>14.1	5	153	-0.597 [-1.434, 0.239]			



Supplemental Figure S16: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on body fat (%) (n = 285)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body fat. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for dose (n=3), test carbohydrate, protein and fat (n=9), carbohydrate, protein and fat within treatment (n=4), carbohydrate, protein and fat between treatment (n=9), test fibre (n=8), fibre within treatment (n=4), fibre between treatment (n=8), test saturated fat (n=3 trials), saturated fat within treatment (n=2 trials) and saturated fat between treatments (n=3 trials), therefore analyses were not performed.

BMI, body mass index; btw, between; CHO, carbohydrate; CI = confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; trt, treatment.

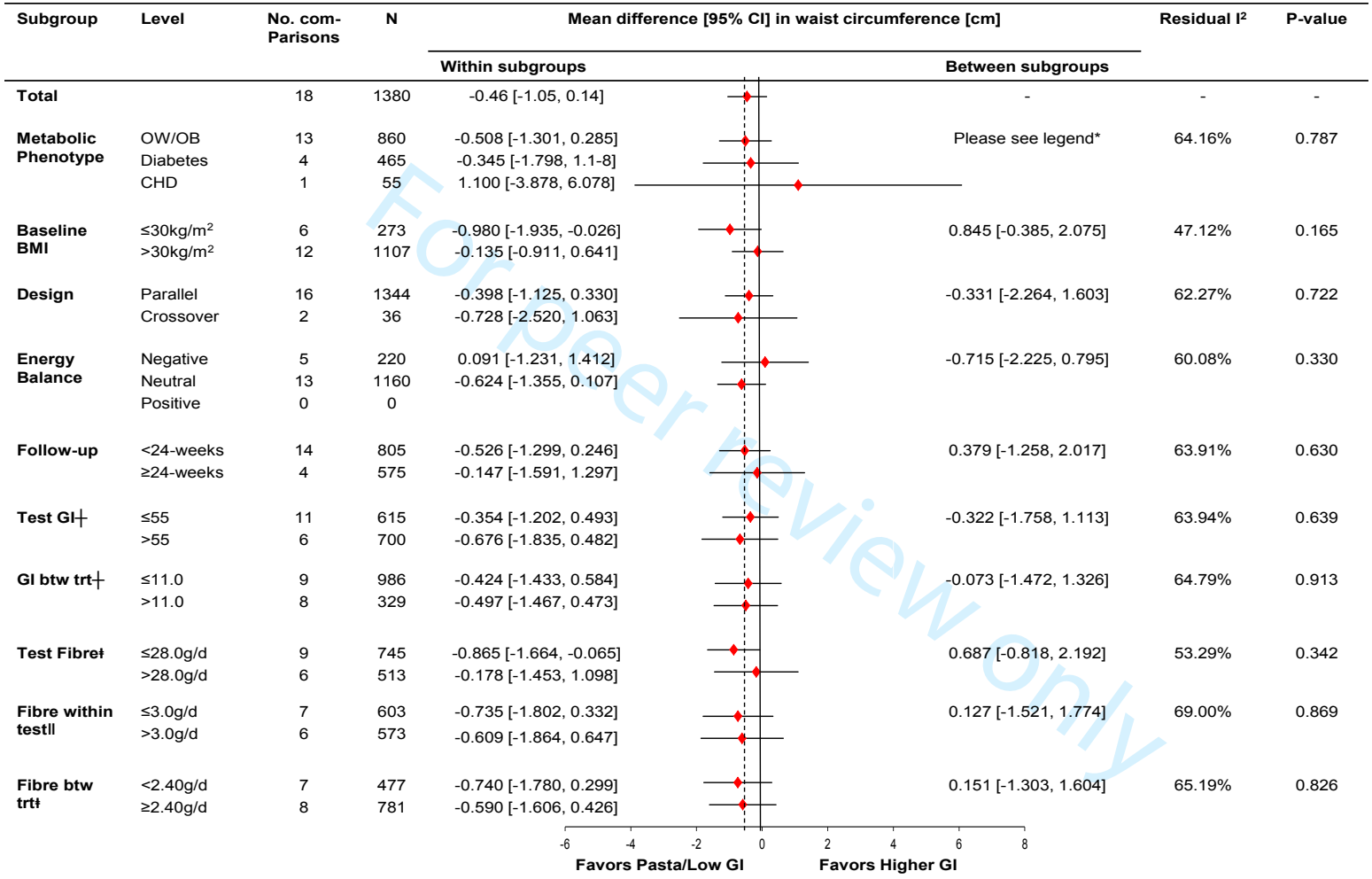


Supplemental Figure S17: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on body fat (%) (n = 285)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on body fat. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

*Within and between subgroup analyses could not be performed against HRB since no values for HRB subgroup.
** no values for URB subgroup.
§ Blinding of Participants, Personnel, and Outcome Assessors.

CI, confidence interval; HRB, High Risk of Bias; GI, glycemic index; LRB, Low Risk of Bias; URB, Unclear Risk of Bias

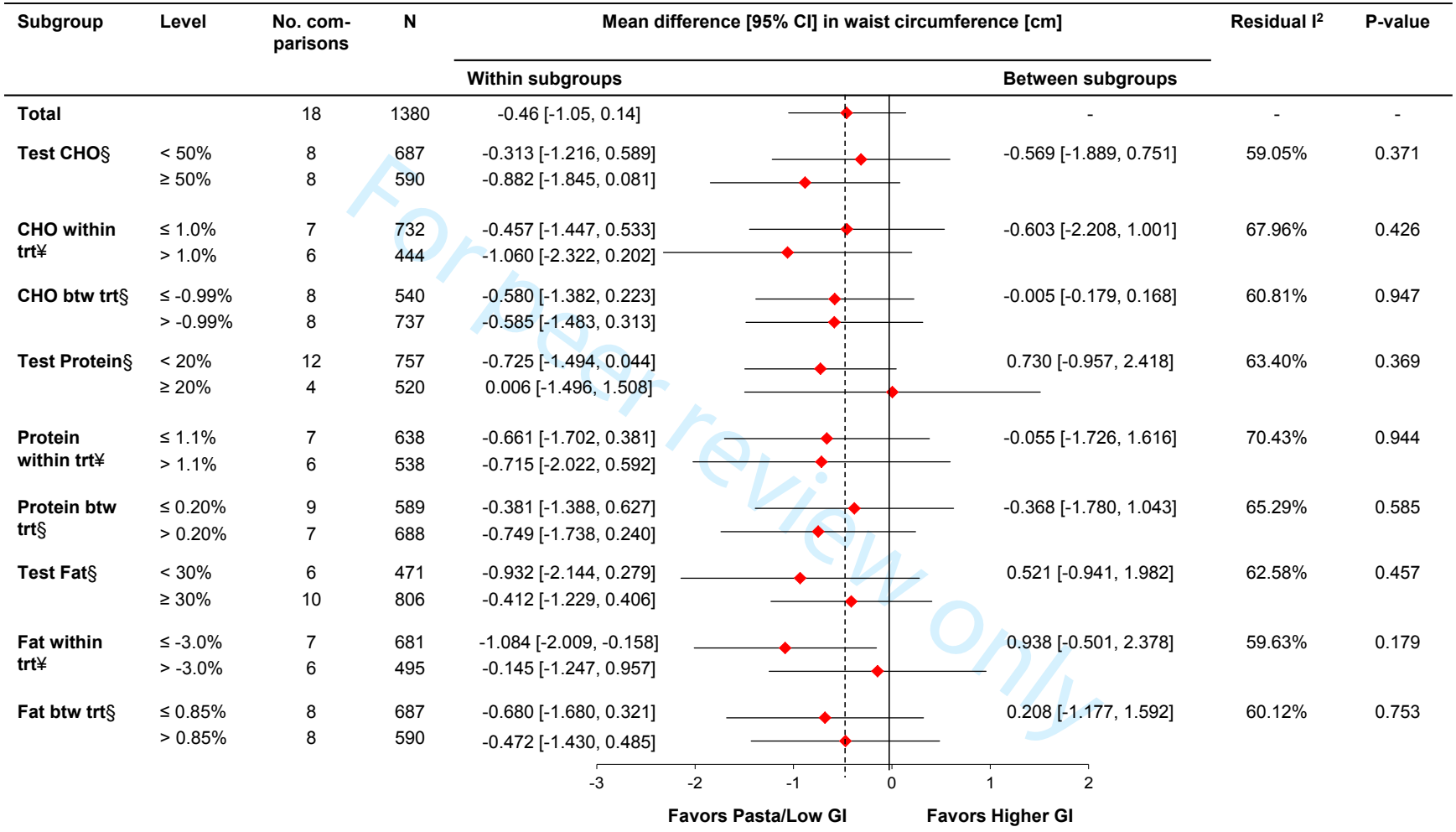


Supplemental Figure S18: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on waist circumference (cm) (n = 1380)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for dose (n=2 trials), therefore analyses were not performed.

*Pairwise between-subgroup mean differences (95% CIs) for Metabolic Phenotype were as follows: 0.163cm (-1.492, 1.818) (1 vs. 2) to 1.608cm (-3.432, 6.648) (1 vs. 3) to -1.445cm (-6.630, 3.740) (2 vs. 3).
┼ data available on 17 studies
ł data available on 15 studies

BMI, body mass index; btw, between; CI = confidence interval; GI, glycemic index (glucose scale); OB, obese; OW, overweight; trt, treatment.

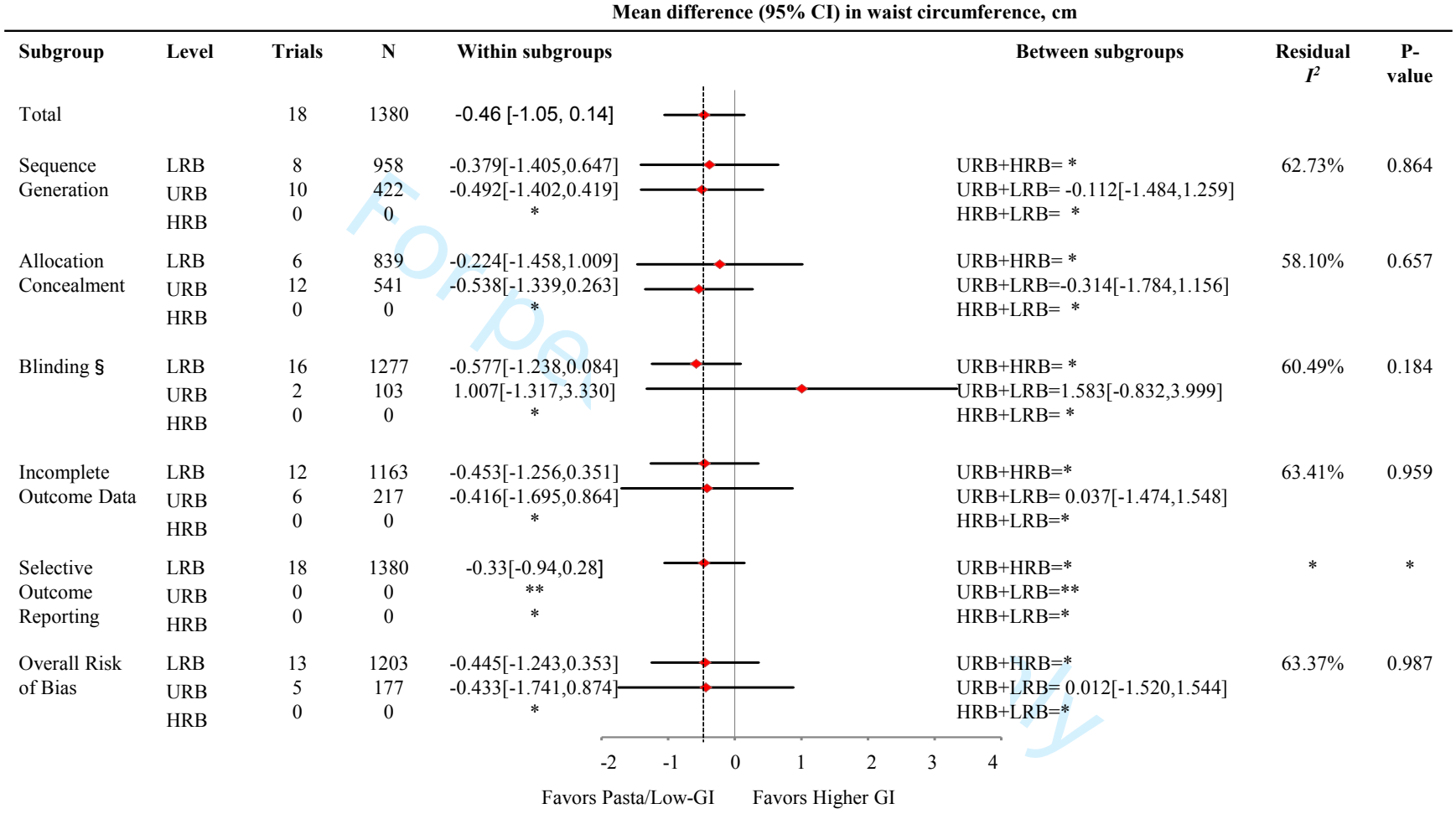


Supplemental Figure S19: Subgroup analysis for mean differences (95% CIs) of the effects of pasta in the context of low-GI dietary patterns on waist circumference (cm) continued (n = 1380)

The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup. Within and between-treatment changes were categorically assessed based on the median intakes. There were less than 10 trials available for categorical subgroup assessment for test saturated fat (n=8 trials), saturated fat within treatment (n=7 trials) and saturated fat between treatments (n=8 trials), therefore analyses were not performed.

§ data available on 16 studies
¥ data available on 13 studies

BMI, body mass index; btw, between; CHO, carbohydrate; CI = confidence interval; GI, glycemic index (glucose scale); trt, treatment.

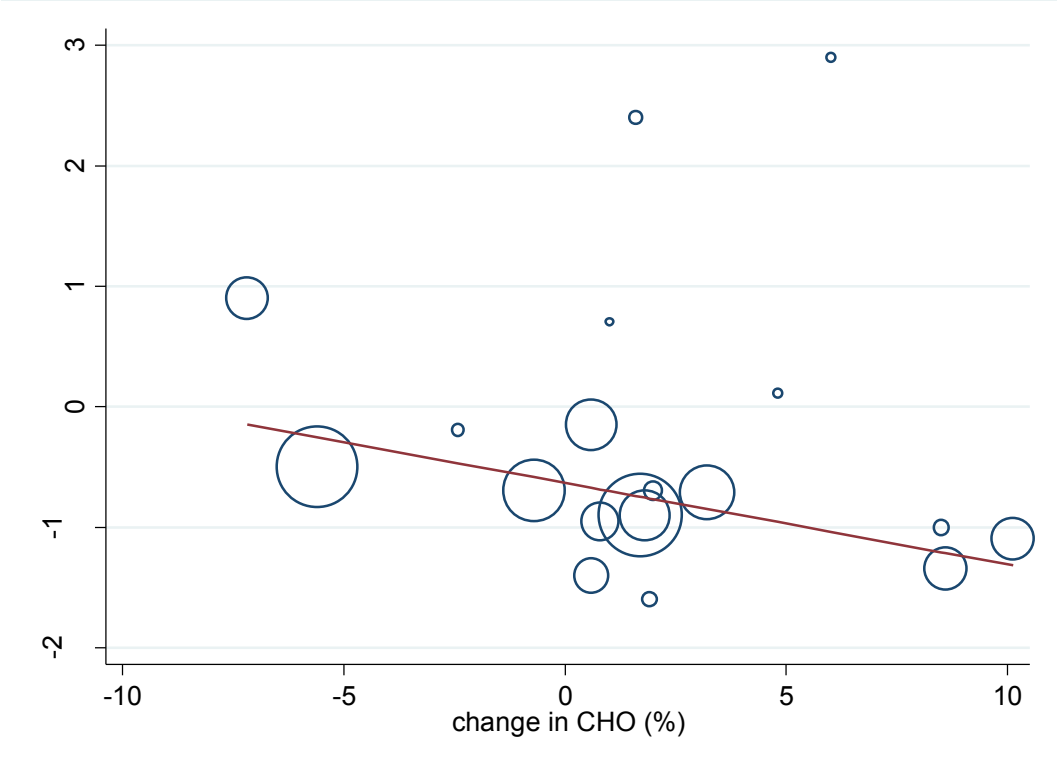


Supplemental Figure S20: A priori subgroup analyses on risk of bias for mean differences (95% CIs) of the effect of pasta in the context of low-GI dietary patterns on waist circumference (cm) (n = 1380)

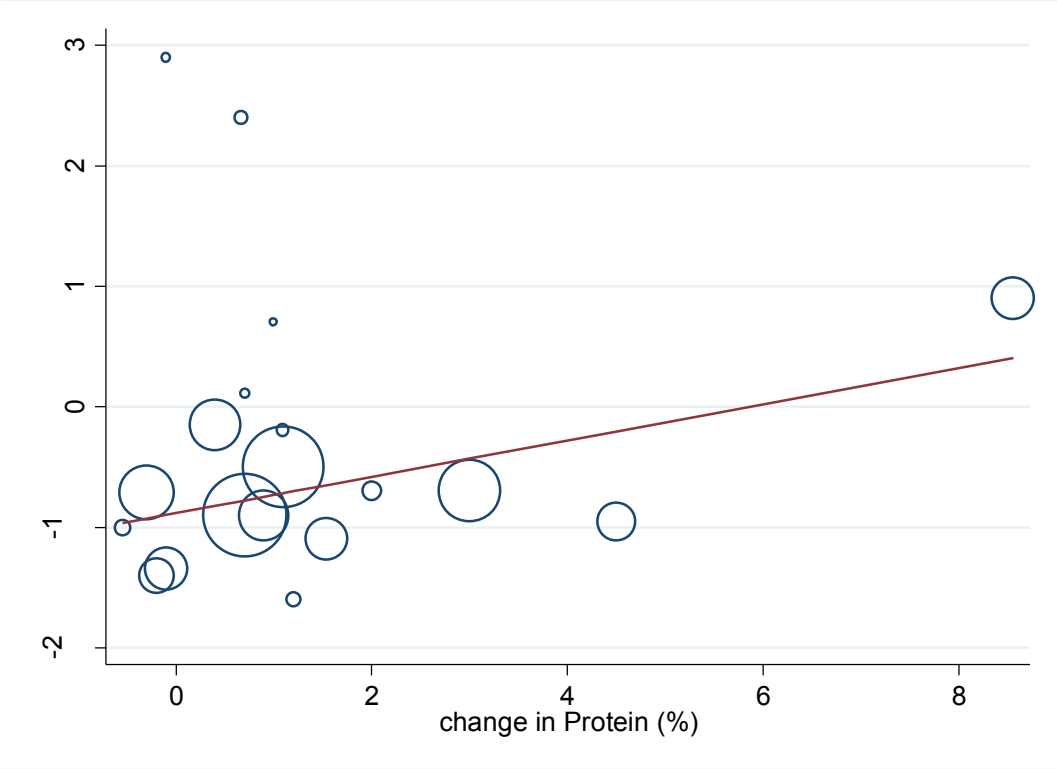
The dashed line represents the pooled estimate for the overall primary analysis of pasta in the context of low-GI dietary patterns on waist circumference. Within subgroup mean differences are the pooled effect estimates represented by a diamond. 95% CIs are represented by the line through the square. The residual I^2 was estimated with the use of the Cochran's Q statistic and indicates the between study heterogeneity unexplained by the variability in response because of the specific between studies factor. $P < 0.05$ indicated that the effect size differed between levels of the subgroup.

*Within and between subgroup analyses could not be performed against HRB since no values for HRB subgroup.
** no values for URB subgroup.
§ Blinding of Participants, Personnel, and Outcome Assessors.

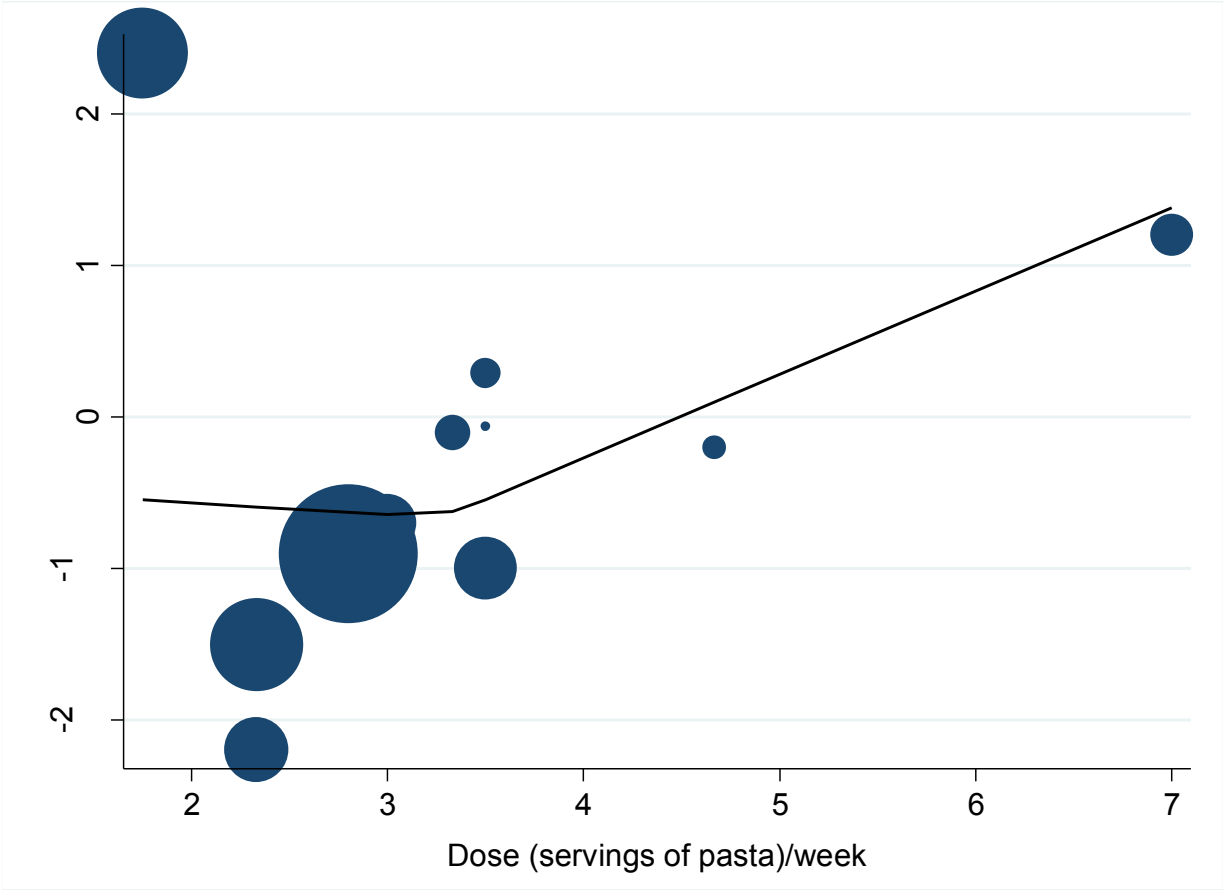
CI, confidence interval; HRB, High Risk of Bias; GI, glycemic index; LRB, Low Risk of Bias; URB, Unclear Risk of Bias



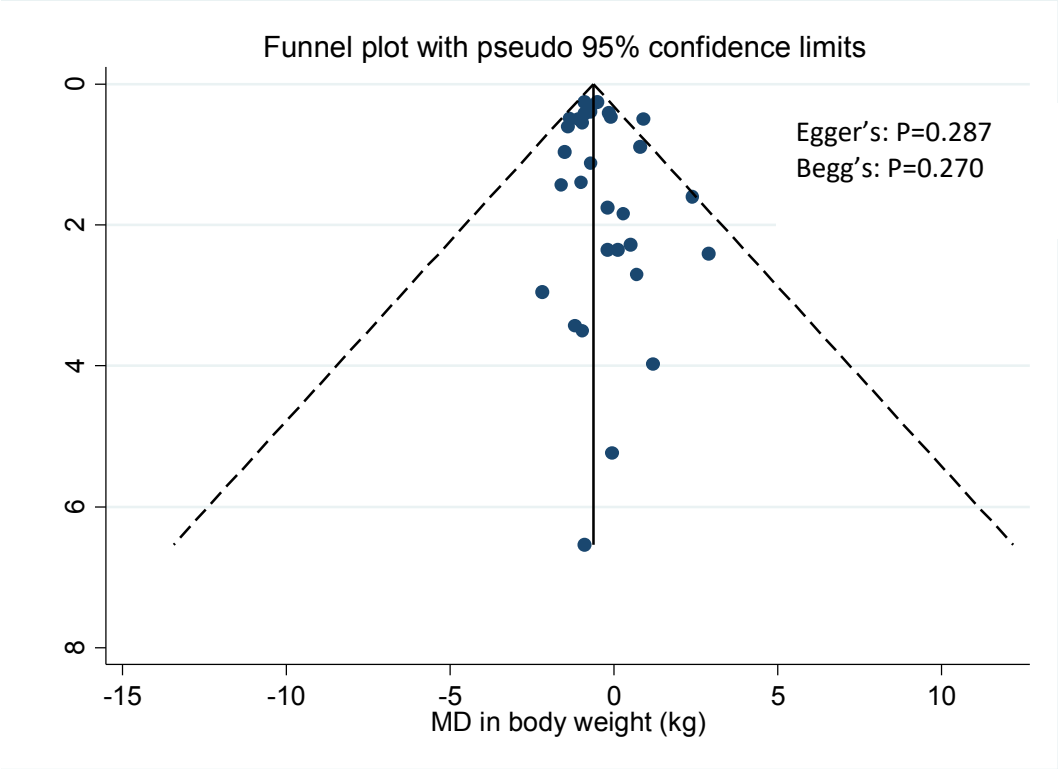
Supplemental Figure S21: Continuous meta-regression of change in carbohydrate intake in the low-GI dietary pattern intervention arms with change in body weight (n=19)
CHO, carbohydrate; MD, mean difference



Supplemental Figure S22: Continuous meta-regression of change in protein intake in the low-GI dietary pattern intervention arms with change in body weight (n=19)
MD, mean difference

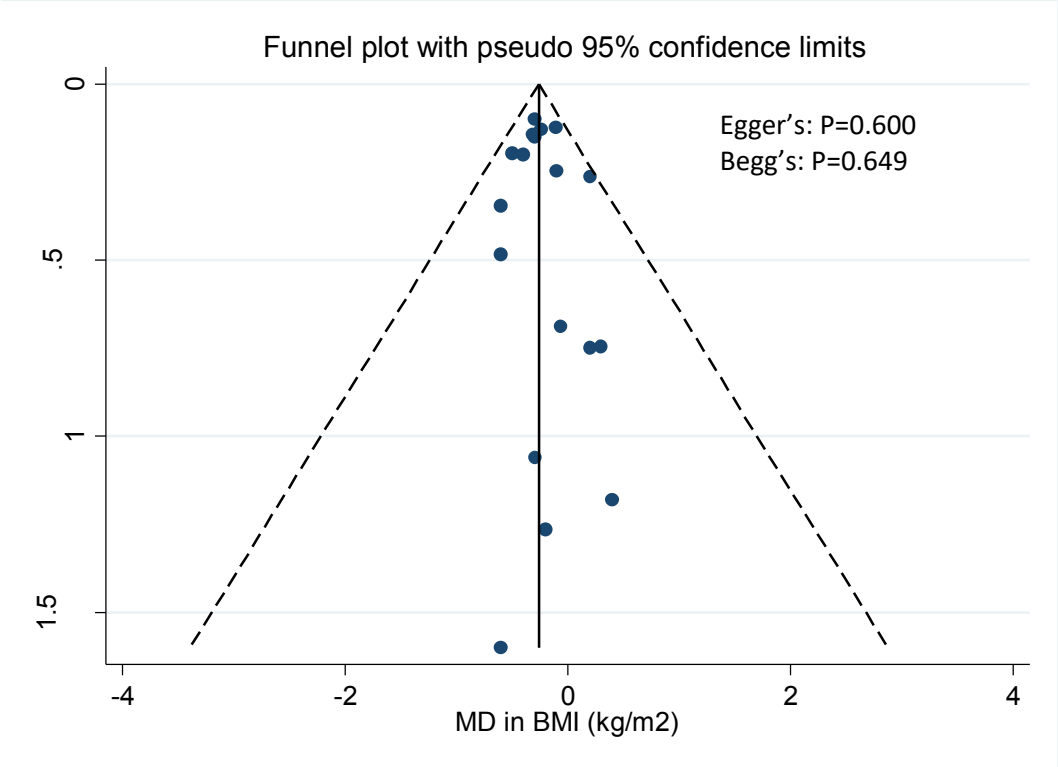


Supplemental Figure S23: Pasta intake dose-response analyses by spline curve modeling (MKSPLINE procedure)



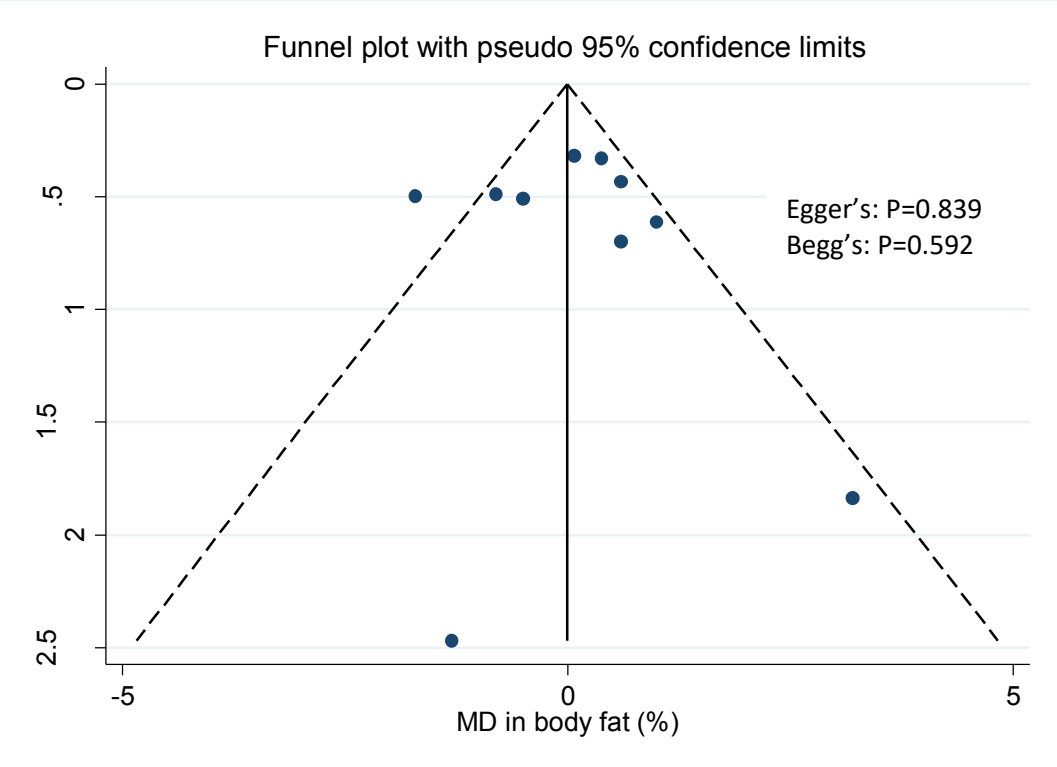
Supplemental Figure S24: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on body weight (kg)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.



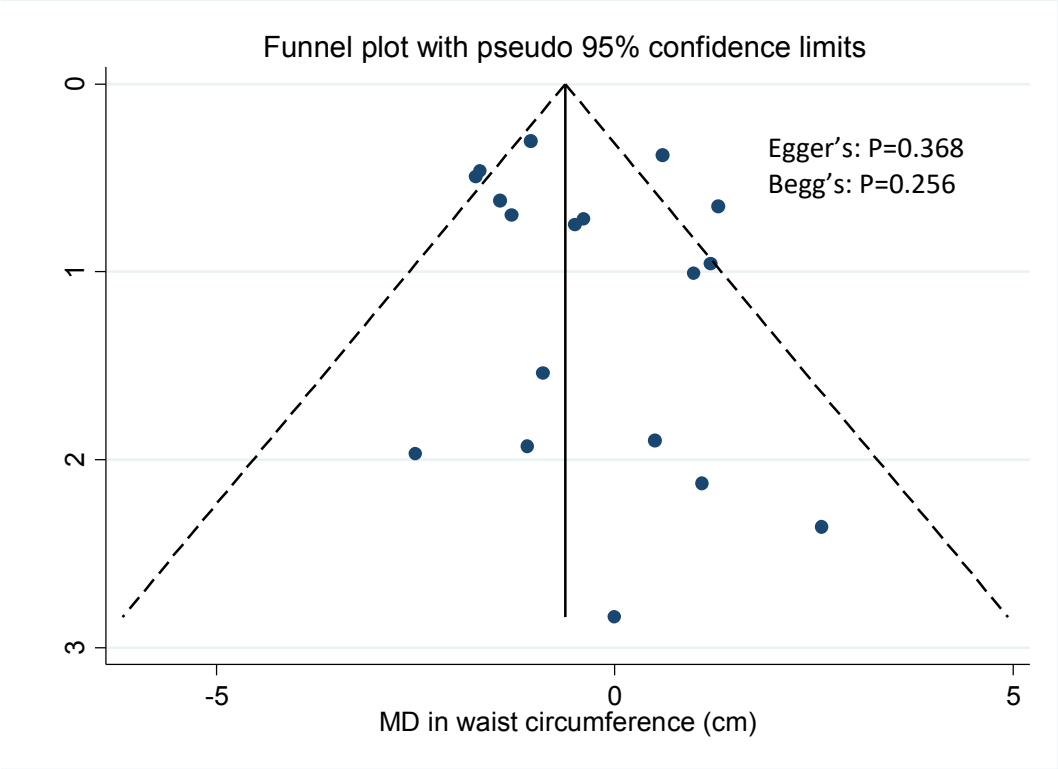
Supplemental Figure S25: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on BMI (kg/m^2)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.



Supplemental Figure S26: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on body fat (%)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.



Supplemental Figure S27: Funnel plot for the effect of pasta in the context of low-GI dietary patterns on waist circumference (cm)

The solid line represents the overall pooled estimate for all studies included in the meta-analysis expressed as a weighted mean difference. Dashed lines represent 95% CIs. P values are derived from the quantitative assessment of publication bias by Egger's and Begg's tests.

Supplemental Table S1: PRISMA Checklist

PRISMA 2009 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.	2-3
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of what is already known.	4
Objectives	4	Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS).	4
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 Table S2
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, Supplemental Table S1
Search	8	Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated.	Supplemental Table S1
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
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
Data items	11	List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and	6, Supplemental

		simplifications made.	Table S2
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.	6-7
Summary measures	13	State the principal summary measures (e.g., risk ratio, difference in means).	7-8
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.	8

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).	9
Additional analyses	16	Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified.	7-10
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.	10, 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.	10-11, Table 1, Supplemental Tables S2-S3
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).	11, 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.	11-13, Figures 2-3, Supplemental Figures S3-S7
Synthesis of results	21	Present results of each meta-analysis done, including confidence intervals and measures of consistency.	11-13, Figures 2-3, Supplemental Figures S3-S7
Risk of bias across studies	22	Present results of any assessment of risk of bias across studies (see Item 15).	11,15 Supplemental

			Table S1, S2, S12, S15, S17, S20
Additional analysis	23	Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]).	12-16, Supplemental Tables S4-S10, Supplemental Figures S8-S26
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).	16-22
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).	19-21
Conclusions	26	Provide a general interpretation of the results in the context of other evidence, and implications for future research.	22-23
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.	26-31

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

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