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Scientific Advisory Committee on Nutrition

Additional analyses performed subsequent to the original systematic reviews to inform the Carbohydrates Working Group deliberations.

Contents

Additional colo-rectal health analyses

Dietary fibre intake and the risk of colo-rectal cancer (report paragraphs 8.26-8.28)

1. Further meta-analyses were performed which included more recent data from the EPIC cohort (Murphy et al 2012), as identified in the update search, and excluding the previous publication from the EPIC cohort that was included in the colo-rectal health review (Bingham et al 2005). The cancer results presented below have been scaled down in the Carbohydrates and Health report to an increment of 7g/day of dietary fibre.

Figure 1. Forest plot of per unit analysis (10g/day) for dietary fibre intake and colo-rectal cancer risk

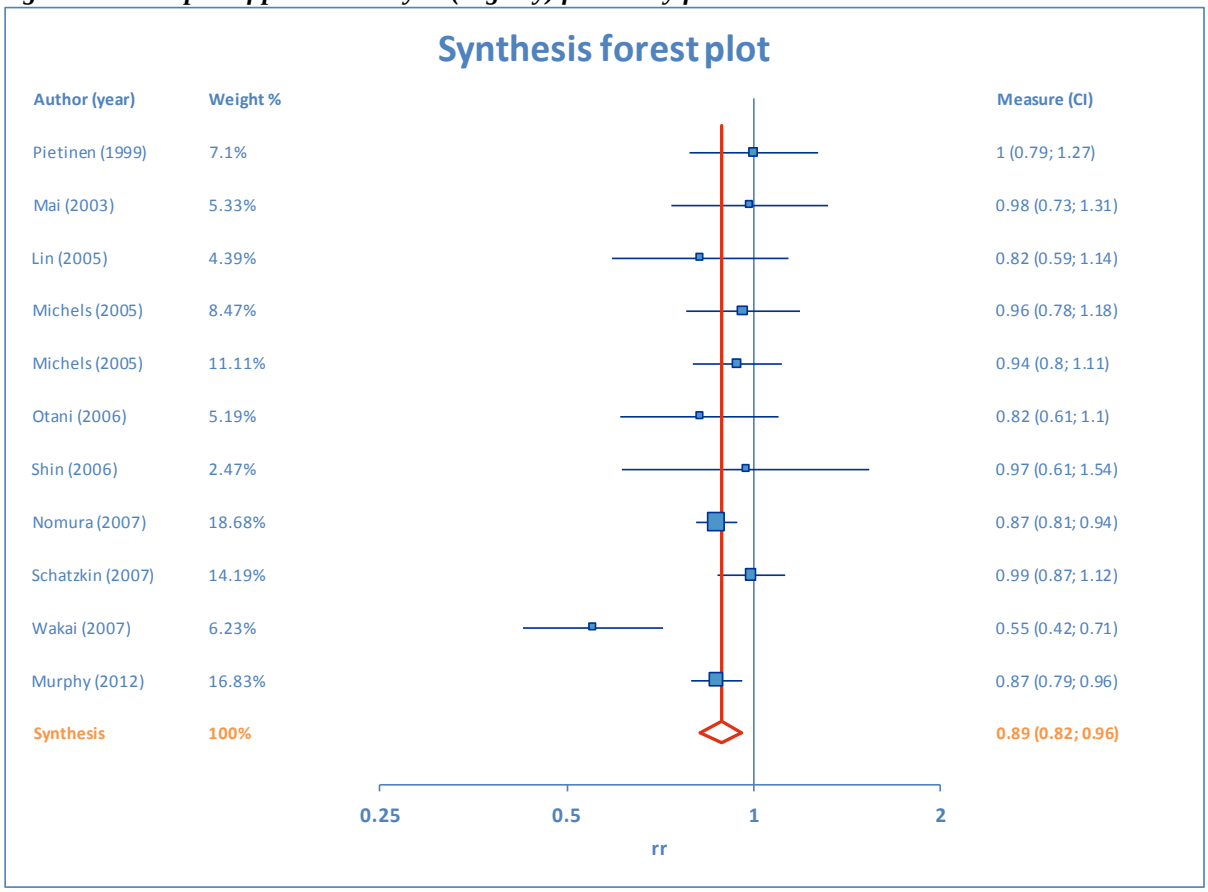


Table 1. Results of per unit meta-analysis (10g/day) for dietary fibre intake and colo-rectal cancer risk

Model	Pooled risk ratio estimate[1]		
	No.[2]	RR (95%CI)	Z (p-value)
Random effect	11	0.89 (0.82-0.96)	-3.06 (p=0.002)

[1] I^2 = 48.0%; p for test of heterogeneity = 0.037

[2] No. of relative risk estimates included in the pooled analysis.

2. Scaled down the per-unit meta-analysis for 7g/d dietary fibre increase in relation to colo-rectal cancer risk: RR 0.92, 95% CI 0.87, 0.97; p=0.002.

Dietary fibre intake and the risk of colon cancer (report paragraphs 8.29-8.30)

Figure 2. Forest plot of per unit analysis (10g/day) for dietary fibre intake and colon cancer risk

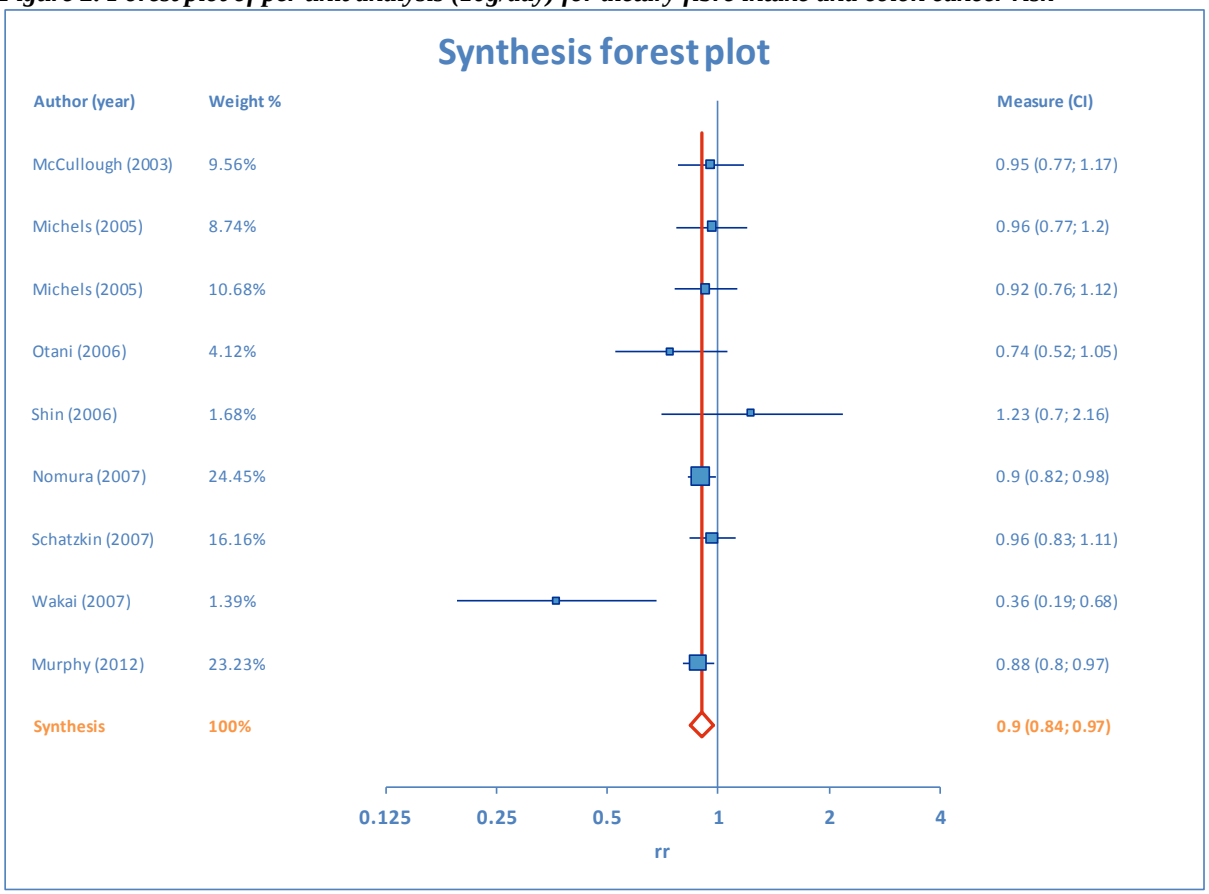


Table 2. Results of per unit meta-analysis (10g/day) for dietary fibre intake and colon cancer risk

Model	Pooled risk ratio estimate[1]		
	No.[2]	RR (95%CI)	Z (p-value)
Random effect	9	0.90 (0.84-0.97)	-2.70 (p=0.007)

[1] I^2 = 34.2%; p for test of heterogeneity = 0.144

[2] No. of relative risk estimates included in the pooled analysis.

3. Scaled down the per-unit meta-analysis for 7g/d dietary fibre increase in relation to colon cancer risk: RR 0.93, 95% CI 0.89, 0.98; p=0.007.

Dietary fibre intake and the risk of rectal cancer (report paragraphs 8.31-8.32)

Figure 3. Forest plot of per unit analysis (10g/day) for dietary fibre intake and rectal cancer risk

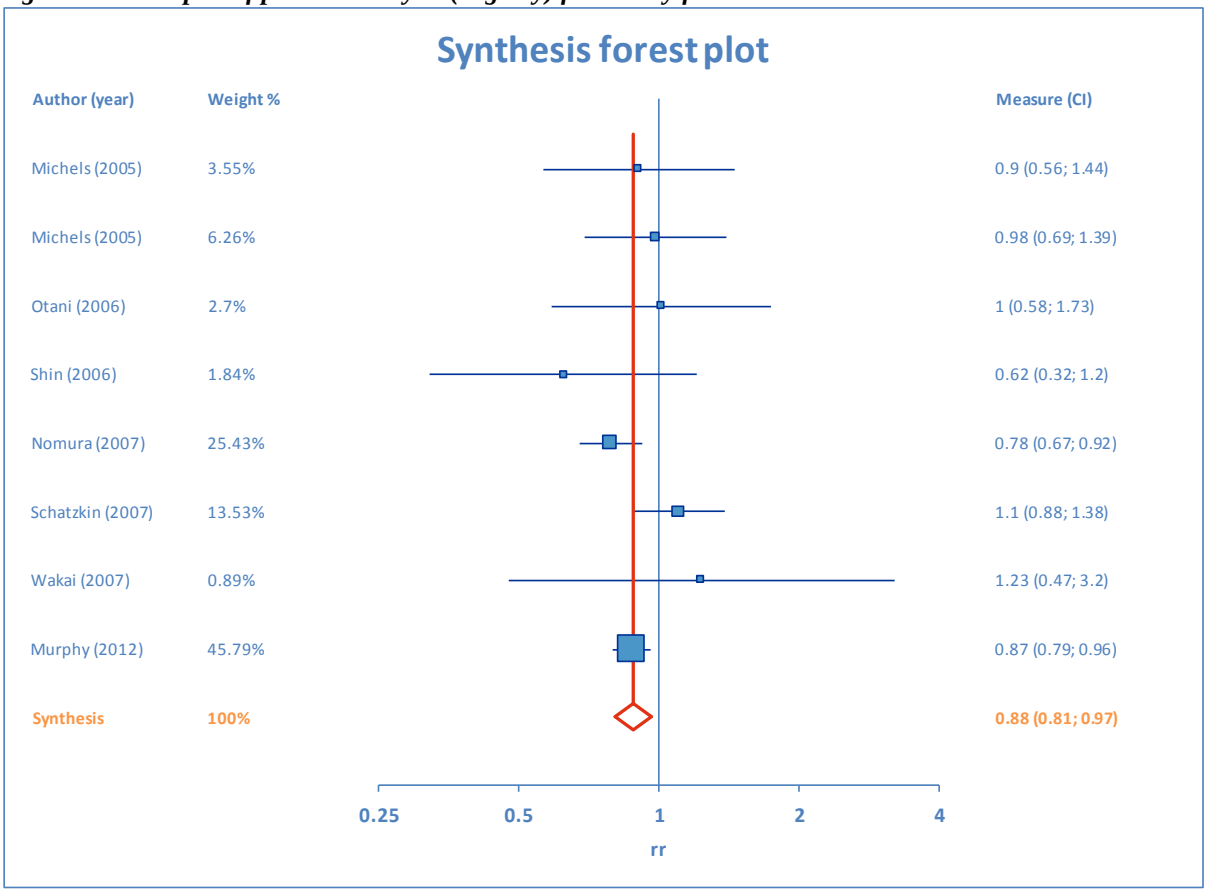


Table 3. Results of per unit meta-analysis (10g/day) for dietary fibre intake and rectal cancer risk

Model	Pooled risk ratio estimate[1]		
	No.[2]	RR (95%CI)	Z (p-value)
Random effect	8	0.88 (0.81-0.96)	-2.69 (p=0.007)

[1] I^2 = 12.1%; p for test of heterogeneity = 0.336

[2] No. of relative risk estimates included in the pooled analysis.

4. Scaled down the per-unit meta-analysis for 7g/d dietary fibre increase in relation to rectal cancer risk: RR 0.91, 95% CI 0.86, 0.97; p=0.007.

Cereal fibre intake and the risk of colo-rectal cancer (report paragraphs 8.114-8.115)

Figure 4. Forest plot of per unit analysis (10g/day) for cereal fibre intake and colo-rectal cancer risk

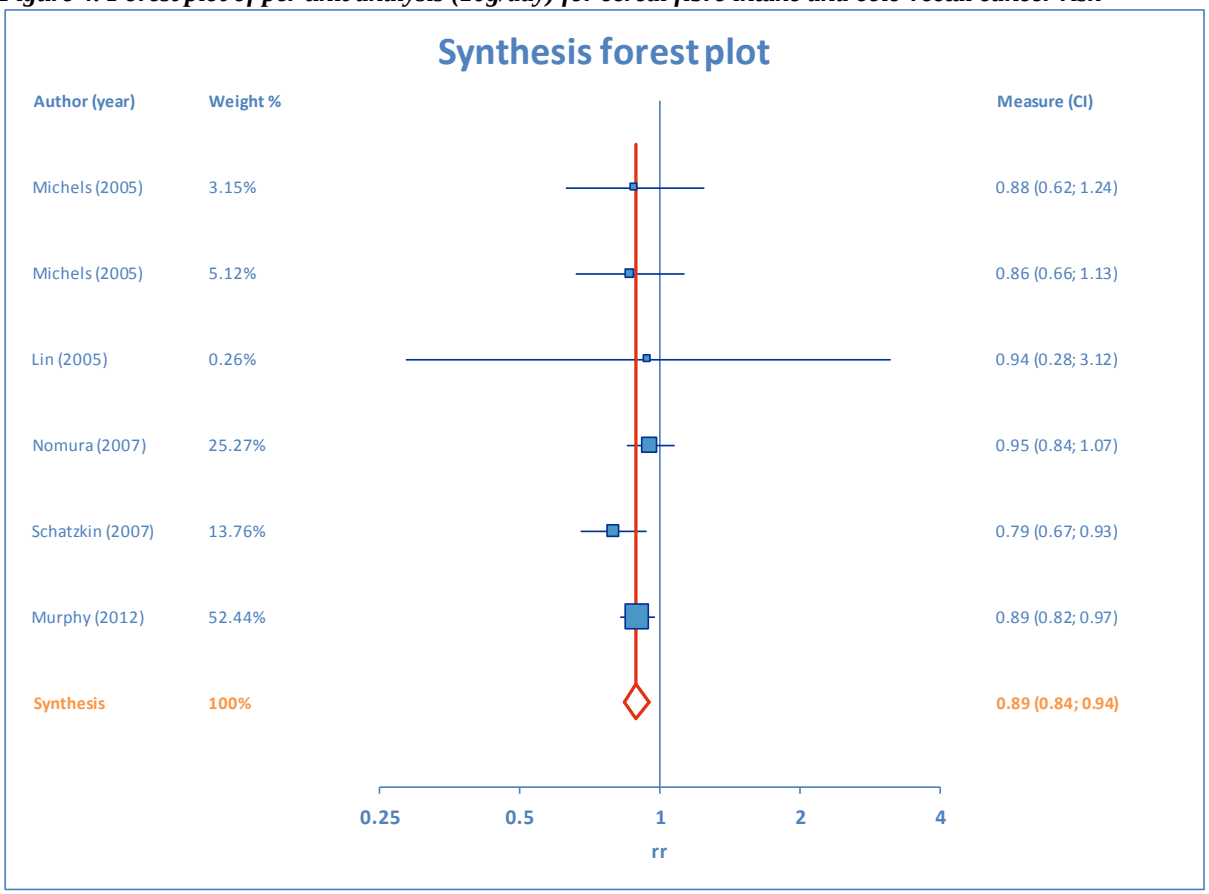


Table 4. Results of per unit meta-analysis (10g/day) for cereal fibre intake and colo-rectal cancer risk

Model	Pooled risk ratio estimate[1]		
	No.[2]	RR (95%CI)	Z (p-value)
Random effect	8	0.89 (0.84-0.94)	-3.12 (p<0.001)

[1] I^2 = 0.00%; p for test of heterogeneity = 0.666

[2] No. of relative risk estimates included in the pooled analysis.

5. Scaled down the per-unit meta-analysis for 7g/d cereal fibre increase in relation to colo-rectal cancer risk: RR 0.92, 95% CI 0.89, 0.96; p=0.001.

Dietary fibre and colo-rectal cancer risk: adjusting studies that determine dietary fibre as NSP to approximate AOAC values (report paragraph A2.11)

6. One study stated that dietary fibre was determined as NSP (Pietinen *et al*., 1999). This provided an estimate for total colo-rectal cancer risk in relation to dietary fibre intakes. This study did not report colon and rectal cancer separately. The RR from this study was scaled down by a factor of 1.3 to give 10 g AOAC value from a 10 g NSP value.

7. In the EPIC cohort study (Bingham *et al*., 2005; Murphy *et al*., 2012) the AOAC method was used for all countries, except in the UK and Greece, where the NSP method was used. The fibre variable used in their analyses was obtained from the EPIC nutrient data base; in which the nutritional composition of foods across the different countries had been standardised. The earlier EPIC report (Bingham *et al*., 2005) was used for analyses of cereal, vegetable, fruit and legume fibre in relation to colo-rectal cancer, as the later paper (Murphy *et al*., 2012) did not report on these dietary fibre constituents. It is not possible to adjust the dietary fibre intake data within the EPIC cohort study.

8. Only the meta-analysis of dietary fibre intake in relation to total colo-rectal cancer risk needed to be re-analysed to adjust NSP for AOAC values for Pietinen *et al*., 1999. Figure 5 and Table 5**Error! Reference source not found.** show the forest plot and meta-analysis with the adjusted intake data. The assigned weight to the Pietinen *et al*., 1999 study increases slightly in the adjusted analysis, as the variation is reduced due to the RR being scaled down by a factor of 1.3. This has very little impact on the overall pooled risk ratio estimate (compare with Figure 1 and Table 1 above).

Figure 5 Forest plot of per unit analysis (10g AOAC/day) for dietary fibre intake and colo-rectal cancer risk

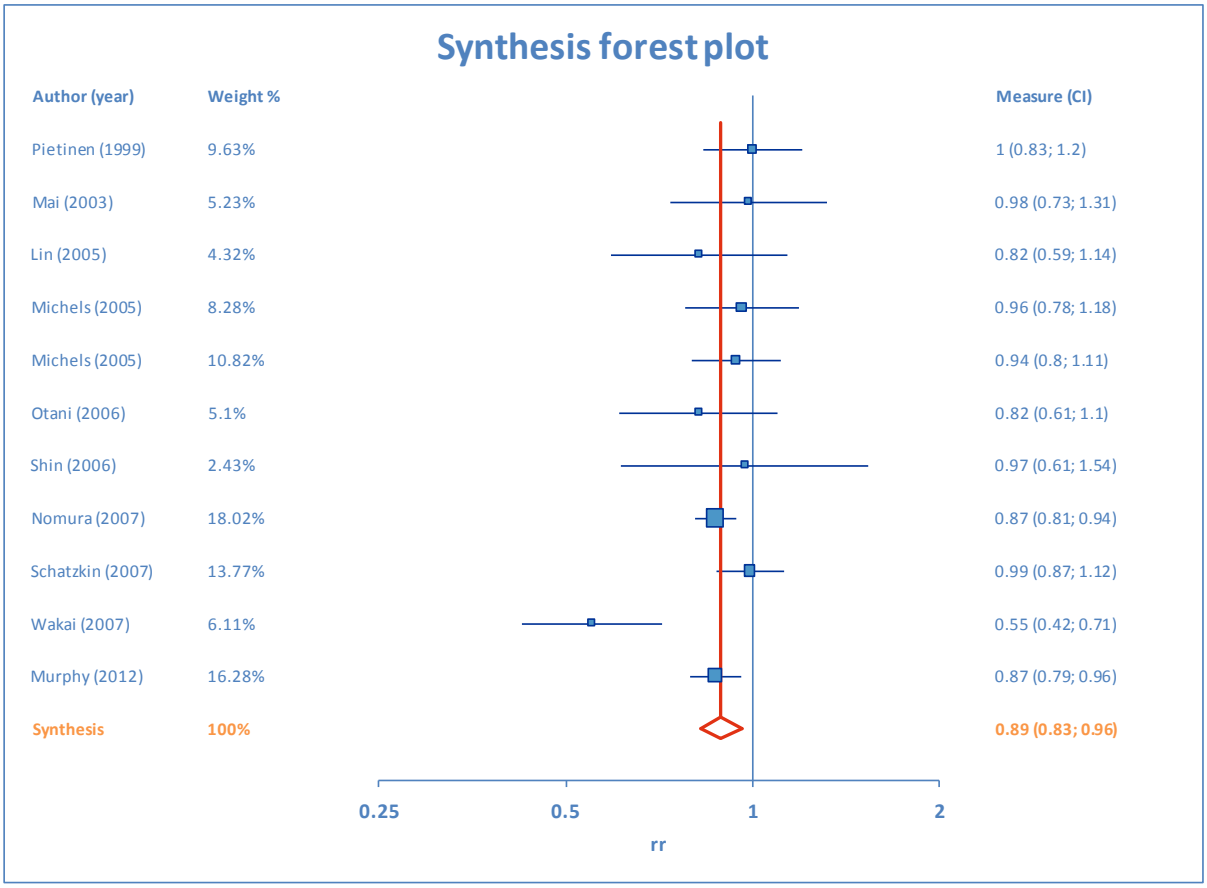


Table 5. Results of per unit meta-analysis (10g AOAC/day) for dietary fibre intake and colo-rectal cancer risk

Model	Pooled risk ratio estimate[1]		
	No.[2]	RR (95%CI)	Z (p-value)
Random effect	11	0.89 (0.83-0.96)	-2.89 (p=0.003)

[1] I^2 = 54.9%; p for test of heterogeneity = 0.014

[2] No. of RR estimates included in pooled analysis.

9. Scaled down the per-unit meta-analysis for 7g/d dietary fibre increase in relation to colo-rectal cancer risk: RR 0.92, 95% CI 0.88, 0.97; p=0.003. This has very little impact on the overall pooled risk ratio estimate (compare with Figure 1 and Table 1 above).

Non-digestible oligosaccharide or inulin supplementation and calcium absorption in adults (report paragraphs 9.35-9.37)

10. Meta-analyses conducted based on the evidence detailed in Chapter 9.

Figure 6. Forest plot of the effect of non-digestible oligosaccharides or inulin supplementation on fractional calcium absorption in adults

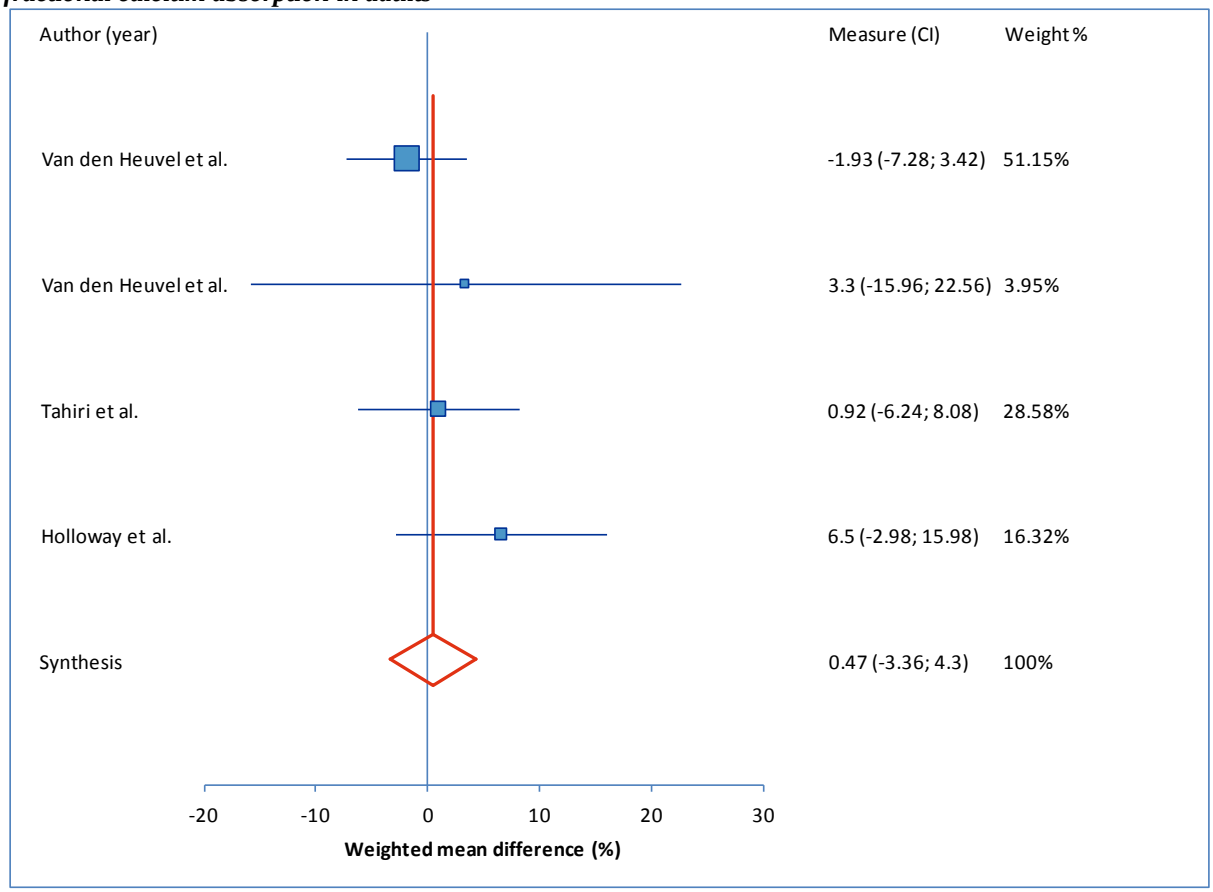


Table 6. Results of meta-analysis for non-digestible oligosaccharides or inulin supplementation on calcium absorption in adults

Model	Pooled mean difference estimate[1]		
	No.[2]	% (95%CI)	Z (p-value)
Random effect	4	0.47 (-3.36-4.29)	0.24(p=0.81)

[1] I^2 = 0.00% (95% CI 0.00-84.69%); p for test of heterogeneity = 0.489

[2] No. of mean difference estimates included in the pooled analysis.

Non-digestible oligosaccharide or inulin supplementation and calcium absorption in children and adolescents (report paragraphs 9.68-9.69)

Figure 7. Forest plot of the effect of non-digestible oligosaccharides or inulin supplementation on fractional calcium absorption in children and adolescents

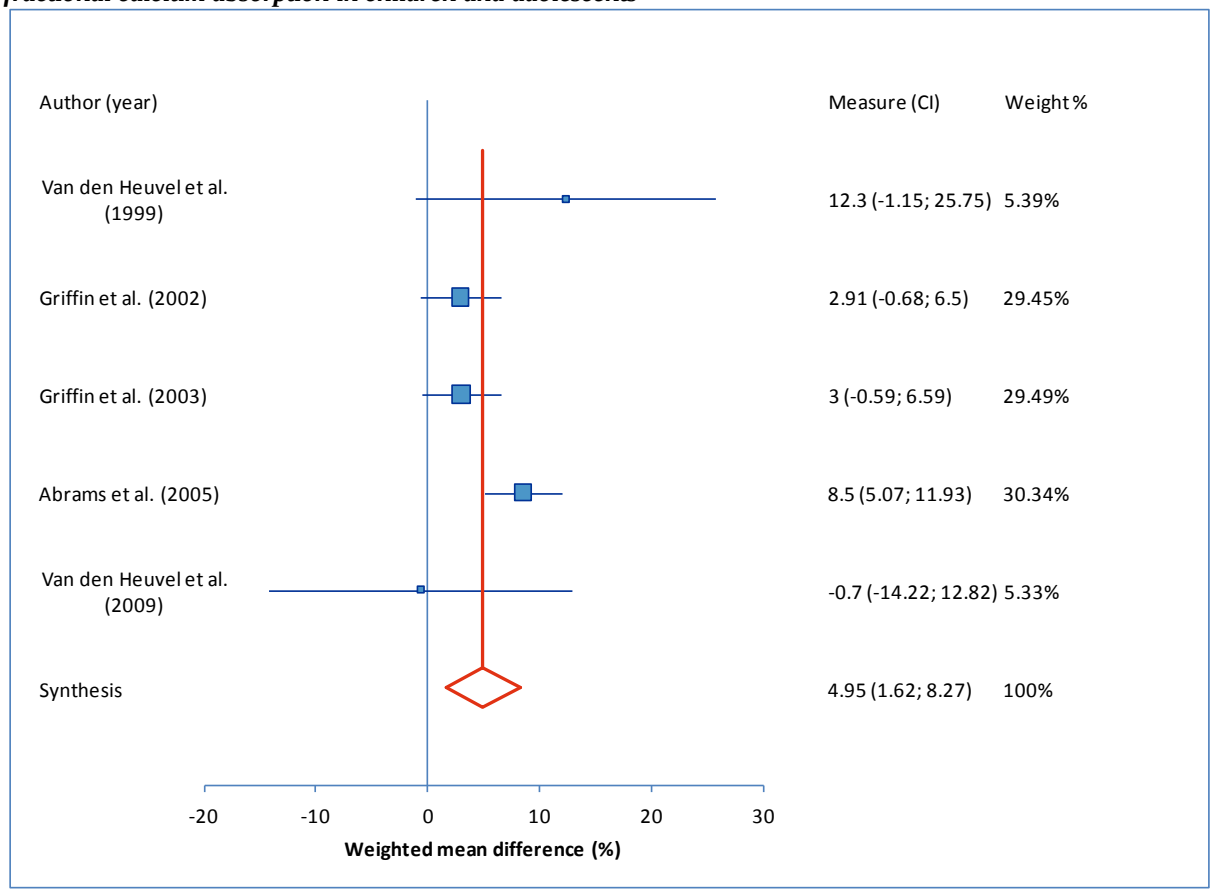


Table 7. Results of meta-analysis for non-digestible oligosaccharides or inulin supplementation on calcium absorption in children and adolescents

Model	Pooled mean difference estimate[1]		
	No.[2]	% (95%CI)	Z (p-value)
Random effect	5	4.95 (1.62-8.27)	2.726(p=0.003)

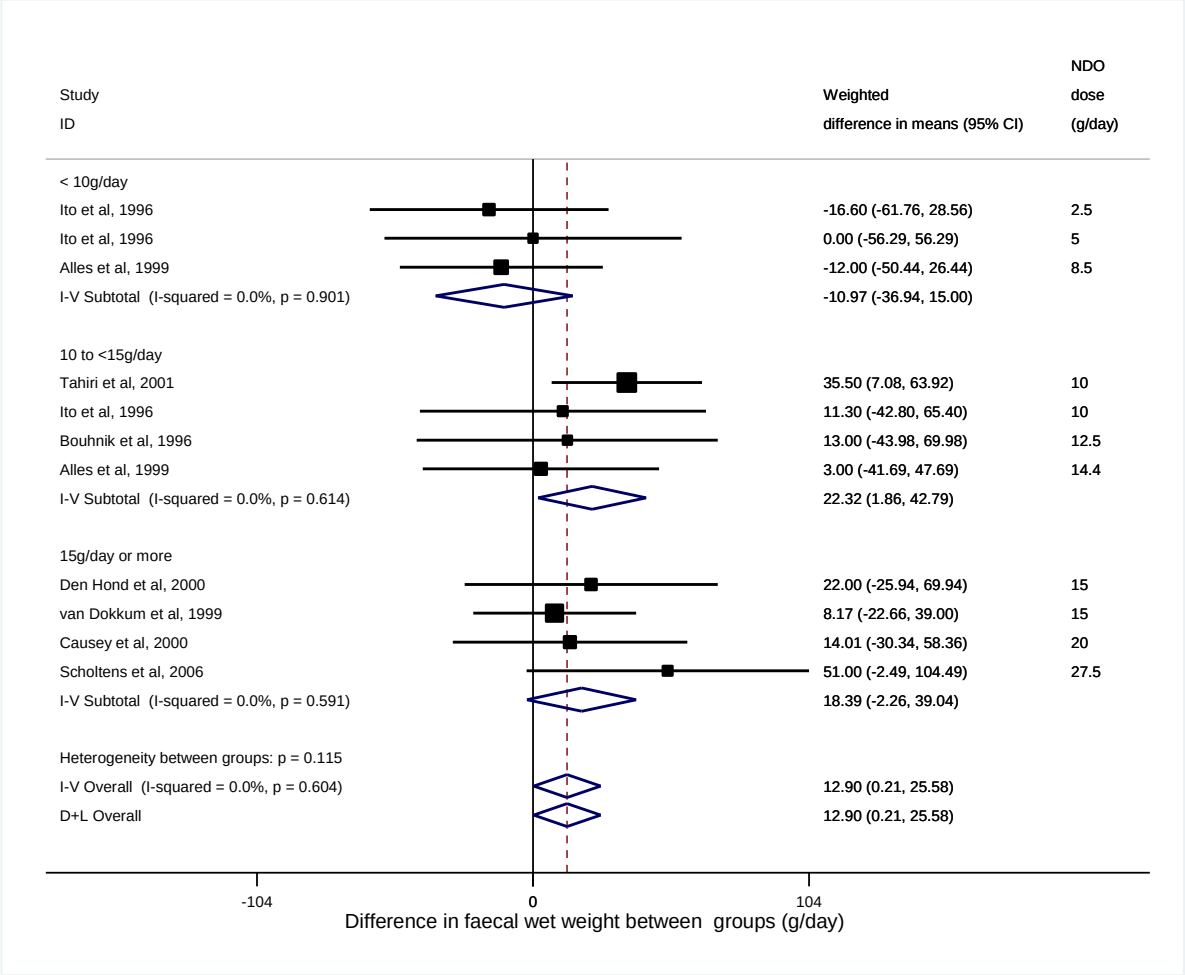
[1] I^2 = 51.79% (95% CI 0.00-82.29%); p for test of heterogeneity = 0.081

[2] No. of mean difference estimates included in the pooled analysis.

Investigation of the dose-response relationship between dietary fibre and faecal wet weight and, where possible, intestinal transit time

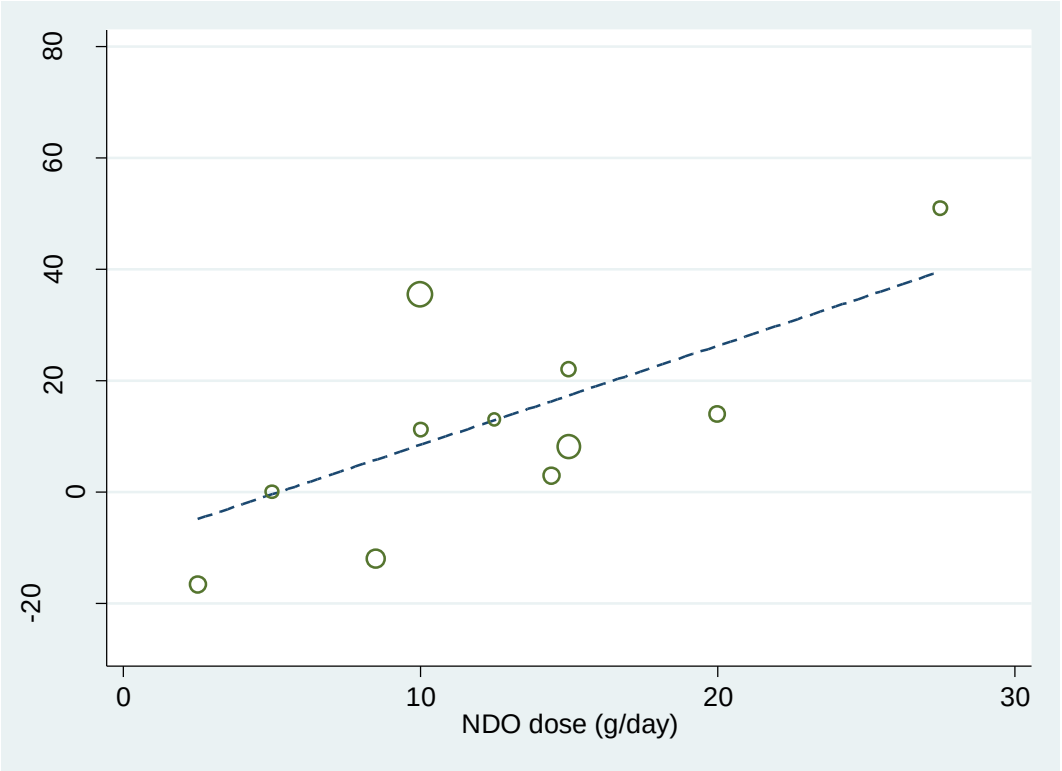
Non-digestible oligosaccharides (report paragraph 9.24)

Figure 8. Sub-group analysis of non-digestible oligosaccharide (FOS, GOS & inulin) intake and faecal weight by exposure category



11. The test for heterogeneity between sub-groups is not significant (p=0.115), indicating no significant dose-response relationship in the data.

Figure 9. Meta-regression plot and analysis of non-digestible oligosaccharide (FOS, GOS & inulin) intake in relation to faecal wet weight



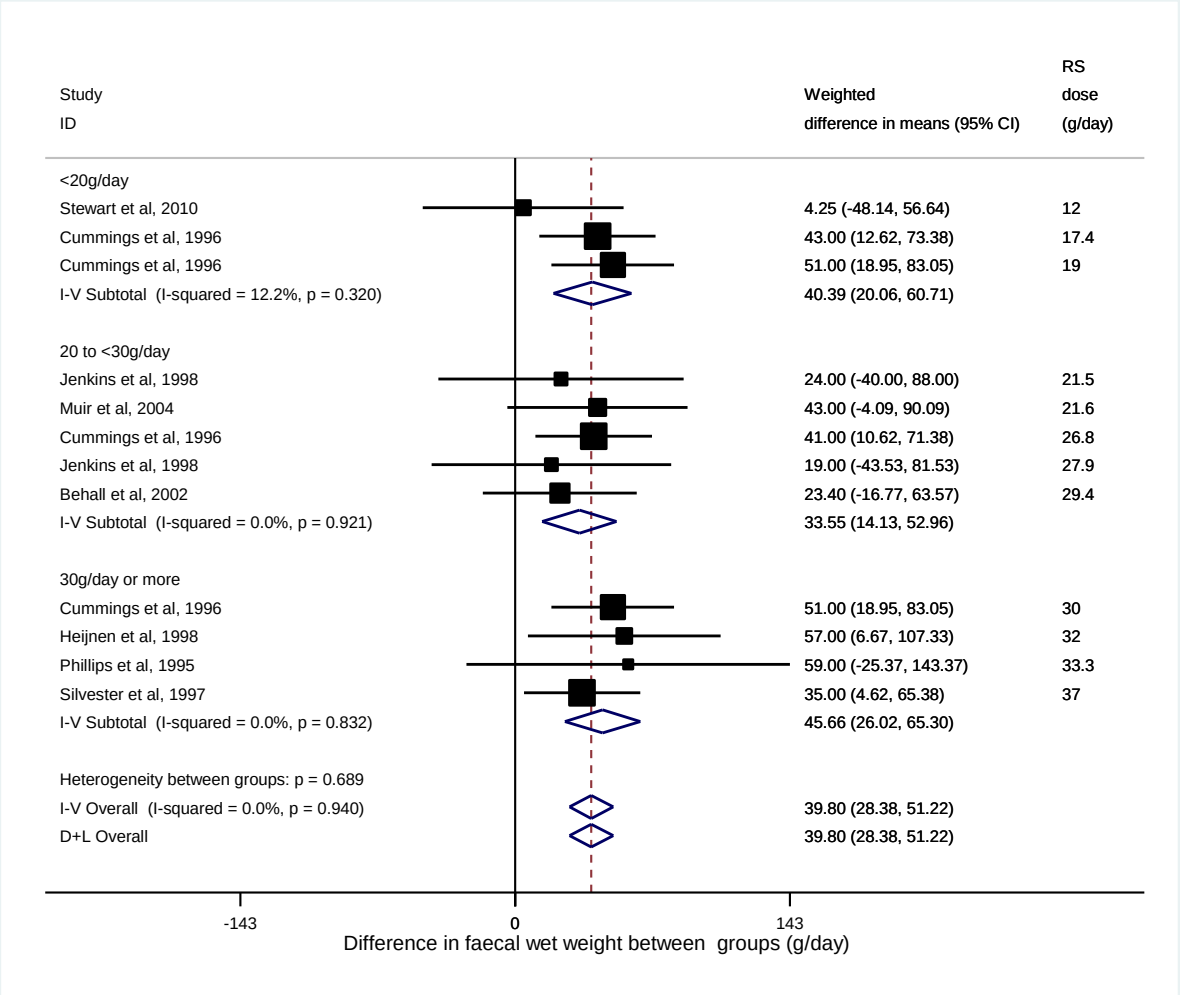

```
Meta-regression                                   Number of obs  =      11
REML estimate of between-study variance           tau2           =   25.87
% residual variation due to heterogeneity         I-squared_res  =   0.00%
Proportion of between-study variance explained    Adj R-squared  = -44.67%
With Knapp-Hartung modification
```

md	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
NDO	1.81137	1.150715	1.57	0.150	-.7917282	4.414469
_cons	-9.918389	15.85427	-0.63	0.547	-45.78324	25.94646

12. The meta-regression analysis is not significant (p=0.150), indicating no significant linear dose-response relationship in the data. A limitation with the meta-regression analysis is that it fits a linear regression line, but does not determine non-linear relationship

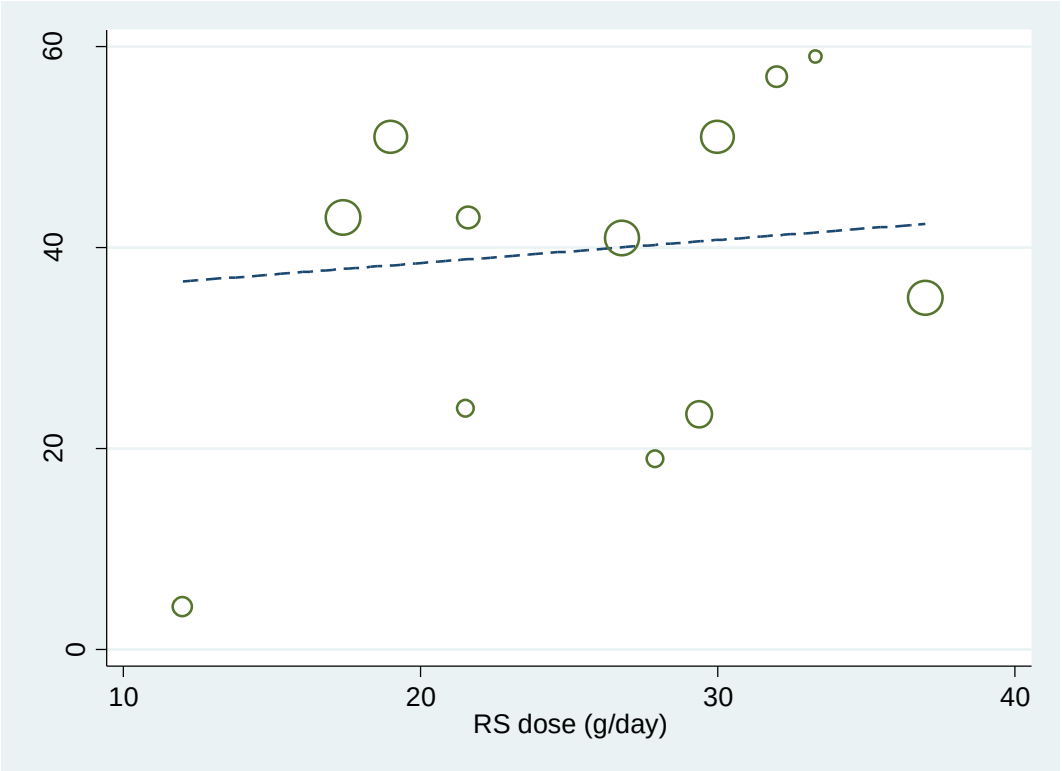
Resistant starch (report paragraph 9.45)

Figure 10. Sub-group analysis of resistant starch (RS1, 2 & 3) intake and faecal weight by exposure category



13. The test for heterogeneity between subgroups is not significant (p=0.689), indicating no significant dose-response relationship in the data.

Figure 11. Meta-regression plot and analysis of resistant starch (RS1, 2 & 3) intake in relation to faecal wet weight




```
Meta-regression                                 Number of obs  =        12
REML estimate of between-study variance         tau2           =         0
% residual variation due to heterogeneity       I-squared_res  =    0.00%
Proportion of between-study variance explained  Adj R-squared  =        .%
With Knapp-Hartung modification
```

md	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
RS	.2294103	.817321	0.28	0.785	-1.591694	2.050515
_cons	33.88574	21.86565	1.55	0.152	-14.83397	82.60544

14. The meta-regression analysis is not significant (p=0.785), indicating no significant linear dose-response relationship in the data.

Wheat fibre (report paragraph 8.99)

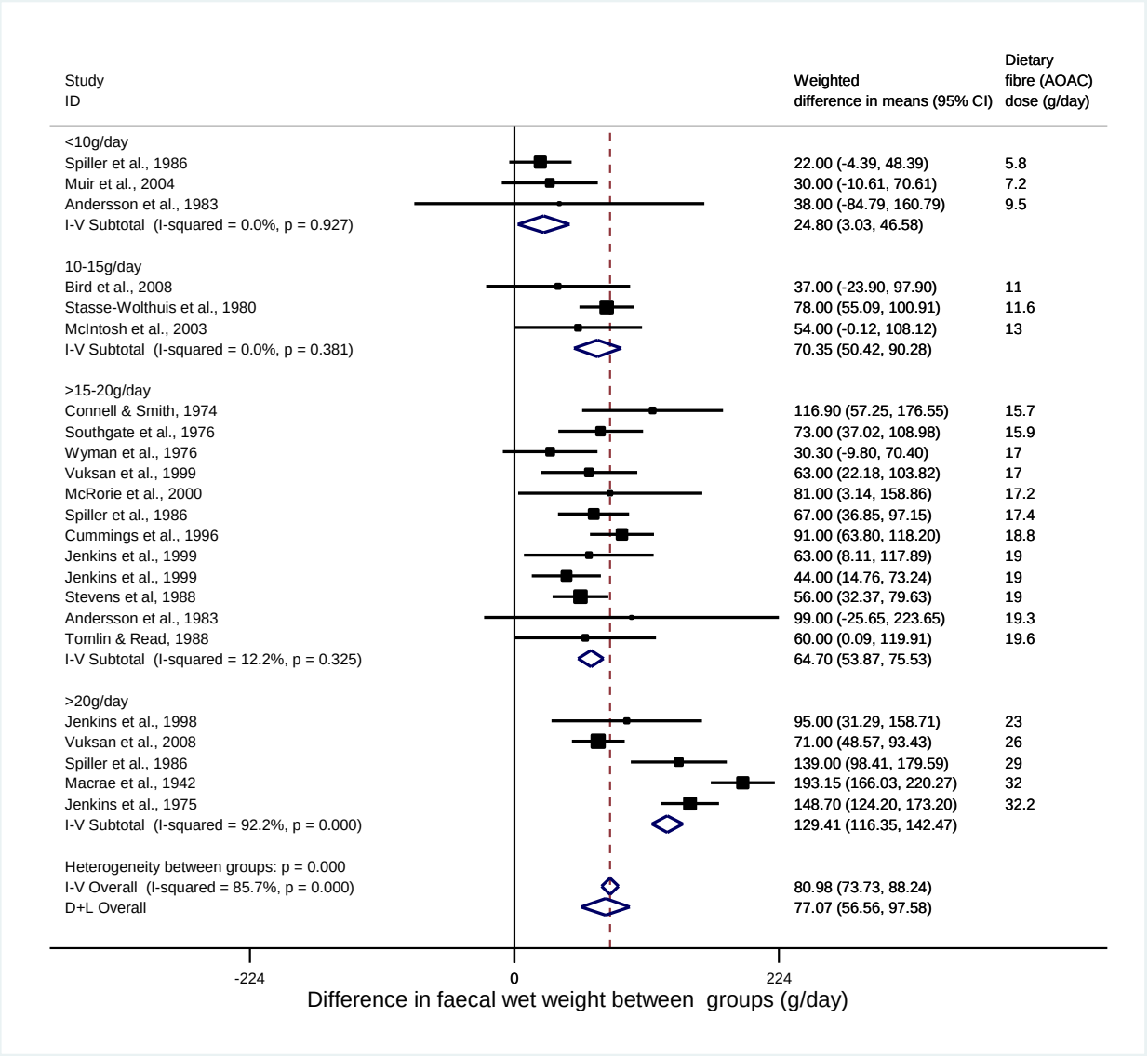
15. In the trials measuring faecal wet weight several different analytical techniques were used to determine the dietary fibre content of wheat fibre. All dietary fibre values for wheat fibre have been converted to AOAC values based on published comparisons between the different techniques. The dietary fibre analytical techniques other than AOAC used in the trials were crude fibre, neutral detergent fibre (Van Soest), Southgate method and NSP. Values were converted to NSP then to AOAC values.

16. Neutral detergent fibre (Van Soest) determines insoluble fibre only and crude fibre determines part of the insoluble fibre. For kidney beans the crude fibre measure is 0.6 that of the neutral detergent fibre (Lunn & Buttriss, 2007). For the conversion of wheat fibre/bran as measured by crude fibre a ratio of 0.43 to NSP was used (AWT, 2005). The values from the McCance and Widdowson food composition tables 5th edition provide Southgate and NSP dietary fibre values for wheat bran and the 7th edition provides NSP and AOAC dietary fibre values for wheat bran. For wheat bran the Southgate value is 1.089 that for NSP and the NSP value is 0.799 that of the AOAC value. The values have been adjusted accordingly to estimate the AOAC value for wheat fibre in each trial, where this is not given.

AWT, Arbeitsgemeinschaft Wirkstoffe in der Tierernährung (2005) *Enzyme in der Tierernährung* (Enzymes in Animal Nutrition), 55pp.

Lunn, J & Buttriss, JL (2007) Carbohydrates and dietary fibre. *British Nutrition Foundation Nutrition Bulletin*, **32**, 21–64

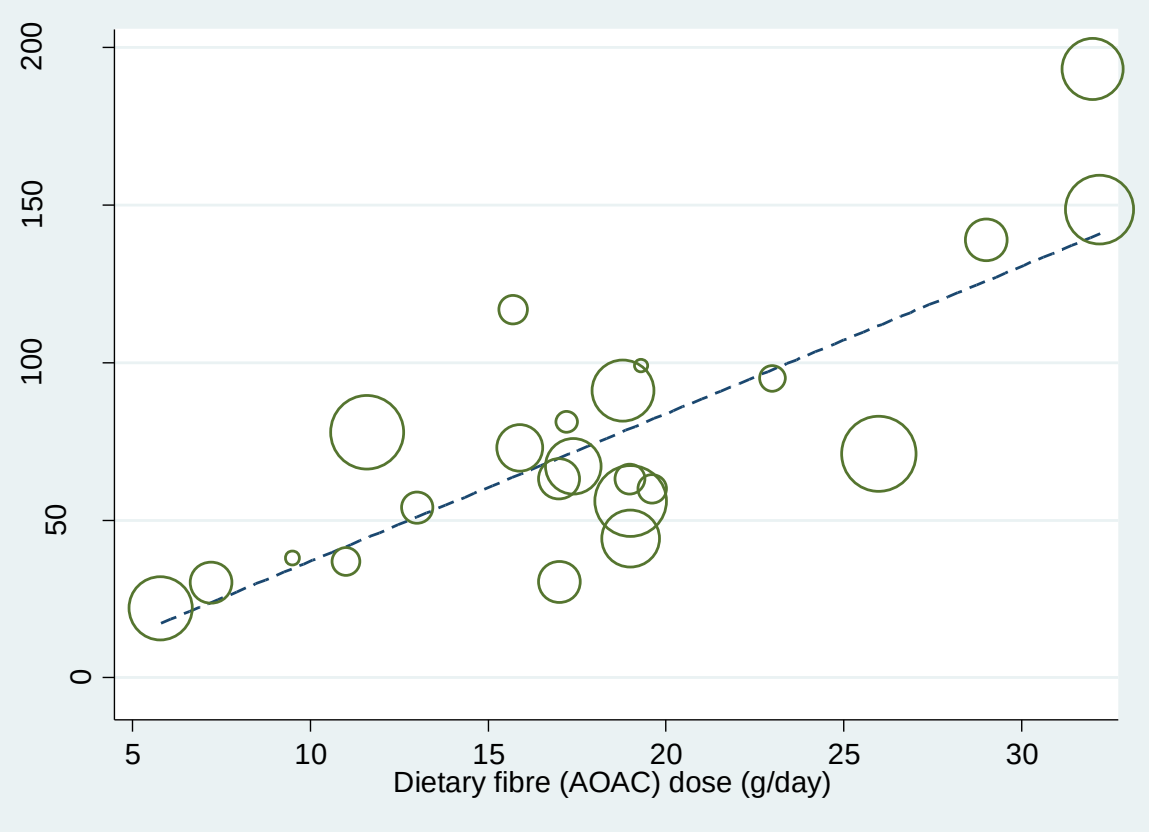
Figure 12. Sub-group analysis of wheat fibre intake and faecal weight by exposure category (AOAC values)



17. The forest plot shows the individual trials in order of ascending dose (right column) and grouped into subgroups of <10g/day, 10-15g/day, >15g-20g/day and >20g/day AOAC dietary fibre. The dose is the mean difference in intake between intervention and control group.

18. The test for heterogeneity between sub-groups is significant ($p<0.001$), indicating a significant dose-response relationship in the data. This is investigated further in the meta-regression analysis below.

Figure 13. Meta-regression plot and analysis of wheat fibre intake in relation to faecal wet weight



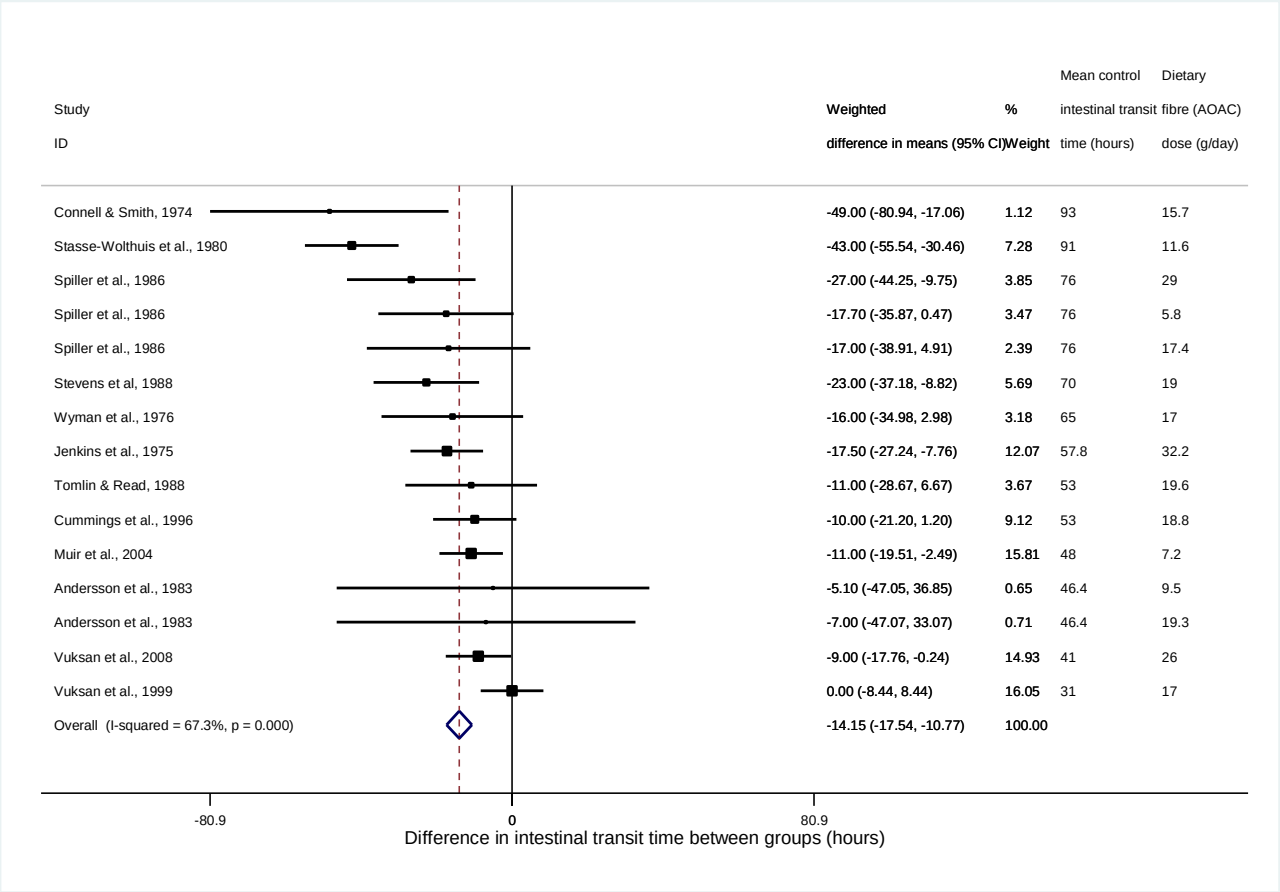

```
Meta-regression                                   Number of obs  =       23
REML estimate of between-study variance           tau2           =    489.7
% residual variation due to heterogeneity         I-squared_res  =   61.85%
Proportion of between-study variance explained    Adj R-squared  =   69.71%
With Knapp-Hartung modification
```

md	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
dose	4.84749	.8639021	5.61	0.000	3.050907	6.644073
_cons	-12.5989	17.42783	-0.72	0.478	-48.84205	23.64424

19. The meta-regression analysis is significant (p<0.001), indicating a significant linear dose-response relationship in the data. A 1g increase in wheat fibre intake results in a 4.8g (95%CI 3.0, 6.6) increase in faecal weight.

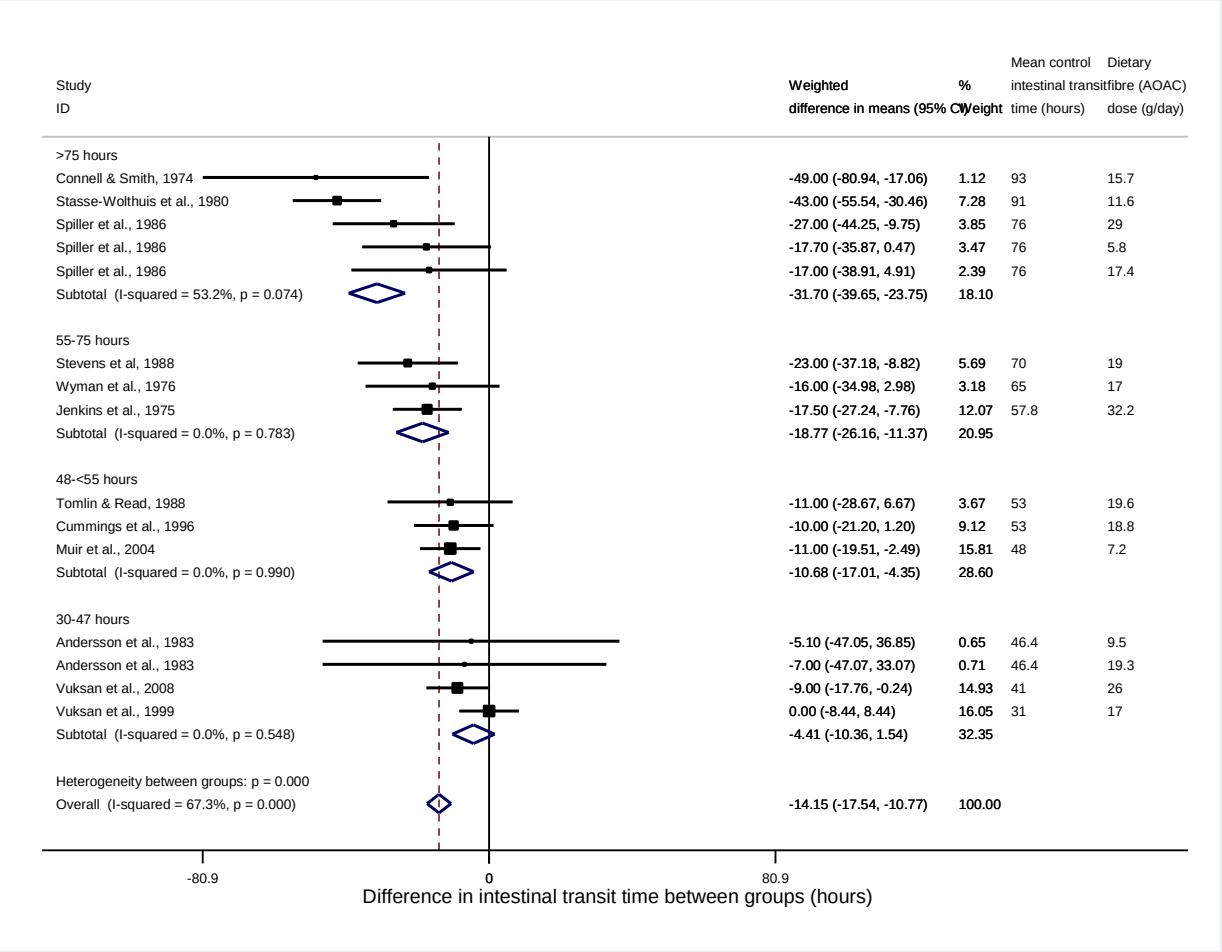
20. The colo-rectal health review estimated the effect on faecal wet weights as broadly equating to a 4g increase in faecal wet weight per 1g wheat fibre.

Figure 14. Forest plot showing the effect of wheat fibre on intestinal transit time, in order of descending mean control intestinal transit time



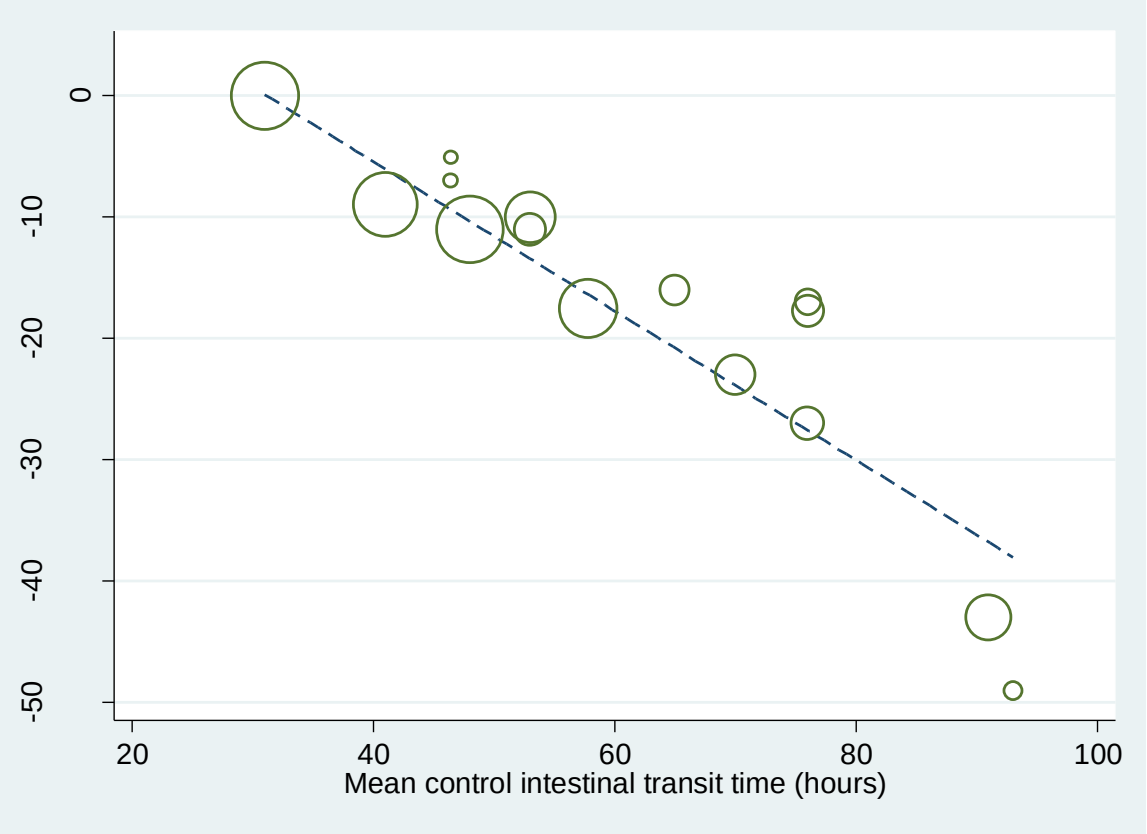
21. The forest plot shows the individual trials in order of descending mean control intestinal transit time (right column). The effect of wheat fibre on decreasing intestinal transit time is significant ($p<0.001$), but the heterogeneity is high ($I^2=67\%$) and this is due to variation in the initial transit time (as determined by control values) modifying the response to wheat fibre. This is investigated further in the meta-regression analysis below.

Figure 15. Sub-group analysis of randomised controlled trials investigating the modifying effect of initial transit time on the effect of wheat fibre on intestinal transit time



22. A sub-group analysis within the meta-analysis was performed by categorising the differences in intestinal transit time into four groups based on initial transit time (>75 hours; 55-75 hours; 48-55 hours; and 30-47 hours). The test for heterogeneity between sub-groups was significant ($p<0.001$), indicating a significant modifying effect of initial transit time on the effect of wheat fibre on intestinal transit time.

Figure 16. Meta-regression showing the modifying effect of initial transit time on the effect of wheat fibre on intestinal transit time




```
Meta-regression                                   Number of obs  =      15
REML estimate of between-study variance           tau2           =       0
% residual variation due to heterogeneity         I-squared_res  =   0.00%
Proportion of between-study variance explained    Adj R-squared  = 100.00%
Joint test for all covariates                     Model F(2,12)  =   19.15
With Knapp-Hartung modification                   Prob > F       =  0.0002
```

md	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
dose	-.0791916	.2139272	-0.37	0.718	-.5452988	.3869157
mean0	-.6184224	.0999872	-6.19	0.000	-.8362759	-.4005689
_cons	20.82544	7.22623	2.88	0.014	5.080841	36.57005

23. The meta-regression includes two variables: wheat fibre dose and control group values (variable mean0 above). It is only possible to graph one variable, which is shown above for mean control intestinal transit time in relation to the effect of wheat fibre on total intestinal transit time. There is no linear dose-response relationship between wheat fibre and transit time in the data (p=0.718), but the effect of initial transit time (as determined by mean control group values) on the intestinal transit time response to wheat fibre is significant (p<0.001).

24. There are insufficient trials to enable the dose-response relationship to be investigated within a sub-set of trials in which subjects have a similar initial intestinal transit time.

Additional cardio-metabolic health analyses

Meta-regression of trials investigating sugars intake in relation to energy intake (report paragraph A9.1)

25. The exposure measure (mean difference in percentage energy intake as sugars) and outcome measure (mean difference in total energy intake) are inter-related. A meta-regression of eleven trials investigating sugars intake in relation to energy intake was performed using exposure data as a comparison of end of intervention if the outcome measure with variance data was reported thus and change from baseline if the outcome measure with variance data was reported thus.

Outcome data (weighted mean difference in total energy intake)

26. The outcome measure with variance data were those reported in the trial, except for Drummond *et al*., 2003, which was analysed using imputed variance data for change from baseline value comparisons (to avoid misrepresentation of results – see Annex 9 in report). Except for that one trial, this approach did not require imputation of further variance data.

Exposure data (mean difference in percentage energy as sugars)

27. The mean difference in sugars intake was calculated from a comparison of the end of intervention data in seven trials (Drummond & Kirk, 1998; Poppitt *et al.*, 2002; Raben *et al.*, 2002; Brynes *et al.*, 2003; Reid *et al.*, 2010; Aeberli *et al.*, 2011; Reid *et al.*, 2014). Two trials only reported the amount of sugars subjects were supplemented with and this was assumed to represent the difference in intakes between the control and intervention groups (Reid *et al.*, 2007; Njike *et al.*, 2011). For two trials the mean difference in sugars intake was calculated from a comparison of change from baseline data (Saris *et al.*, 2000; Drummond *et al.*, 2003).

28. Four trials involved multiple interventions and only the most comparable groups, with the exception of sugars intake, have been used in the analyses (Poppitt *et al.*, 2002; Brynes *et al.*, 2003; Aeberli *et al.*, 2011; Njike *et al.*, 2011). In one trial the intervention groups comprised a high and low GI group, a high fat and high sucrose group (Brynes *et al.*, 2003). The data from the high sucrose group were compared to the high GI group, as these two groups had the most similar dietary intakes with the exception of sucrose intake – the low GI diet had a higher dietary fibre content and the high fat had a lower carbohydrate content. Another trial compared moderate fructose, moderate glucose, high fructose, high glucose, high sucrose and low fructose intervention groups (Aeberli *et al.*, 2011). For the analyses the outcome/exposure data from the high sucrose group were compared to the low fructose group, as these two groups had the greatest difference in sugars intake and there was no specific low sucrose group. The data for sucrose, free glucose and free fructose were combined to give sugars exposure values for each group.

29. A third trial comprised a control diet, a low-fat, complex carbohydrate diet and a low-fat, simple carbohydrate diet (Poppitt *et al.*, 2002). For the analyses the

outcome/exposure data from the low-fat, simple carbohydrate group were compared to the low-fat, complex carbohydrate group, as these two groups had the most similar dietary intakes with the exception of sugars intake. A fourth trial comprised a control diet, a sugar-free cocoa and a sugar-sweetened cocoa group (Njike *et al.*, 2011). For the analyses the outcome/exposure data from the sugar-free cocoa group were compared to the sugar-sweetened cocoa group, as these two groups had the most similar dietary intakes with the exception of sugars intake.

30. To convert grams of sugar to percentage energy. values have been used for individual sugars as follows: 15.7 kJ/g glucose, 15.2kJ/g fructose, 16.3 kJ/g sucrose (Elia & Cummings, 2007). In two trials, which used sucrose as the intervention, only the weight for total sugars intake was reported (Reid *et al.*, 2010; Reid *et al.*, 2014); a value of 16.0 kJ/g was used to convert grams of total sugar to percentage energy.

Difference in sugar intake calculations for each trial

(Drummond & Kirk, 1998)
End of intervention data
% energy NMES 8.1 vs. 10
Difference = 1.9 % total energy

(Saris *et al.*, 2000)
Only change from baseline data reported
% energy simple carbohydrates 7.2 vs. -3.5
Difference = 10.7 % total energy

(Poppitt *et al.*, 2002)
End of intervention data
Six month combined intervention data used for outcome measure as this was the only measure with variance; therefore, six month combined data used for exposure measure.
% energy simple carbohydrates 17.6 vs. 28.9
Difference = 11.3 % total energy

(Raben *et al.*, 2002)
End of intervention data
% energy sucrose 27 vs. 4
Difference = 23 % total energy

(Brynes *et al.*, 2003)
End of intervention data
Sucrose 132g vs. 46g
132 x 16.3kJ = 2.15MJ; 46 x 16.3kJ = 0.75MJ
Difference = (2.15/9.9 x 100) – (0.75/9.02 x 100) = 13.4 % total energy

(Drummond *et al.*, 2003)
Change from baseline data with variance computed
% energy NMES Change from 10 to 10.5 vs. Change from 11.4 to 9.0: 0.5 vs. -2.4
Difference = 2.9% total energy

(Reid *et al.*, 2007)
End of intervention data. No sugars intake data, but the sucrose supplements provided 105g/day sucrose
105g x 16.3kJ/g = 1.71 MJ/day.
End of intervention total energy intake of sucrose group = 8.722MJ/day
Assumed difference = 1.71/8.722 x 100 = 19.6% total energy

(Reid *et al.*, 2010)
End of intervention data
Total sugars 196.65g vs. 105.06g
196.65 x 16.0kJ = 3.15MJ; 105.06 x 16.0kJ = 1.68MJ
Difference = (3.15/9.3 x 100) – (1.68/8.22 x 100) = 13.4 % total energy

(Aeberli *et al.*, 2011)
End of intervention data
The data for sucrose, free glucose and free fructose were combined to give sugars intake
Low fructose = (53.1g x 16.3kJ) + (8g x 15.7kJ) + (7.2g x 15.2kJ) = 1.10MJ
High sucrose = (130.4g x 16.3 kJ) + (14g x 15.7kJ) + (13.4 x 15.2kJ) = 2.55 MJ
Difference = (2.55/10.86 x 100) – (1.10/9.79 x 100) = 12.2 % total energy

(Njike *et al.*, 2011)
End of intervention data. No sugars intake data, but the sucrose-sweetened drinks provided 91 g/d
91g x 16.3kJ = 1.48MJ
End of intervention total energy intake of sucrose group = 1991.4 kcal x 4.184 = 8.33MJ/day
Assumed difference = 1.48/8.33 x 100 = 17.8% total energy

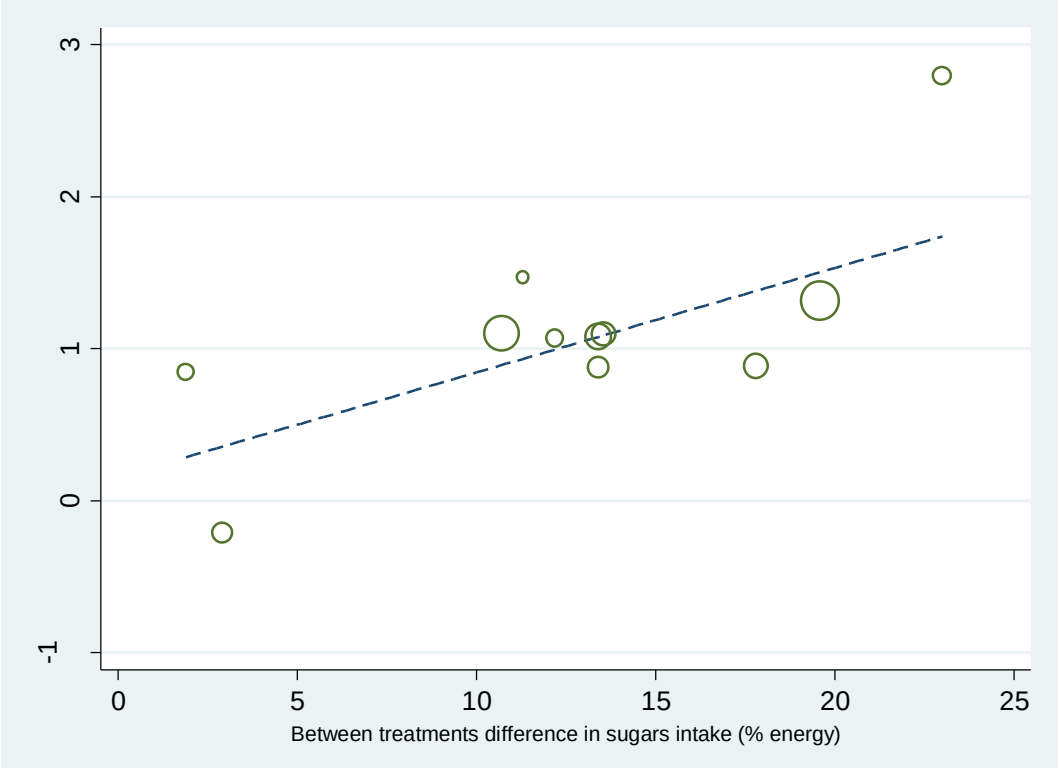
(Reid *et al.*, 2014)
End of intervention data
Total sugars 177.5g vs. 88.4g
177.5 x 16.0kJ = 2.84MJ; 88.4 x 16.0kJ = 1.41MJ
Difference = (2.84/9.091 x 100) – (1.41/7.996 x 100) = 13.55 % total energy

Meta-regression analysis

31. The regression coefficient obtained from a meta-regression analysis describes how the outcome variable (weighted mean difference in total energy intake; kJ/day) changes with a unit increase in the explanatory variable (mean difference in % energy from sugars/day).

32. The meta-regression analysis shows a significant linear dose-response relationship with a 69kJ (95%CI 3,135kJ; p=0.042) change in total energy intake per one percentage change in energy from sugars (see **Error! Not a valid bookmark self-reference.**).

Figure 17. Meta-regression plot and analysis of trials investigating sugars intake in relation to energy intake using exposure data as a comparison of end of intervention if the outcome measure with variance data was reported thus and change from baseline if the outcome measure with variance data was reported thus




```
Meta-regression                                 Number of obs  =      11
REML estimate of between-study variance         tau2           =       0
% residual variation due to heterogeneity       I-squared_res  =   0.00%
Proportion of between-study variance explained  Adj R-squared  =      .%
With Knapp-Hartung modification
```

md	Coef.	Std. Err.	t	P>|t|	[95% Conf.	Interval]
sugars	.0687964	.0290051	2.37	0.042	.0031823	.1344106
_cons	.1559747	.4353611	0.36	0.728	-.8288805	1.14083

Higher carbohydrate, lower fat diets and fasting total cholesterol:HDL-cholesterol ratio (report paragraphs 5.40-5.41)

33. The mean difference values used in the Cardio-metabolic review were incorrect for one of the trials (Howard *et al.*, 2006). In the review the value on the forest plot (figure 2.43) for the mean difference for the Howard et al 2006 paper is reported as -0.10 95% CI -0.15, -0.03, but in the paper it is reported as -0.04, 95% CI -0.13, 0.5. Therefore, the data have been re-analysed and the pooled estimate given below has been used in the Carbohydrates and Health report.

Figure 18. Forest plot displaying randomised controlled trials investigating the effect of higher carbohydrate, lower fat diets on fasting total cholesterol:HDL-cholesterol ratio

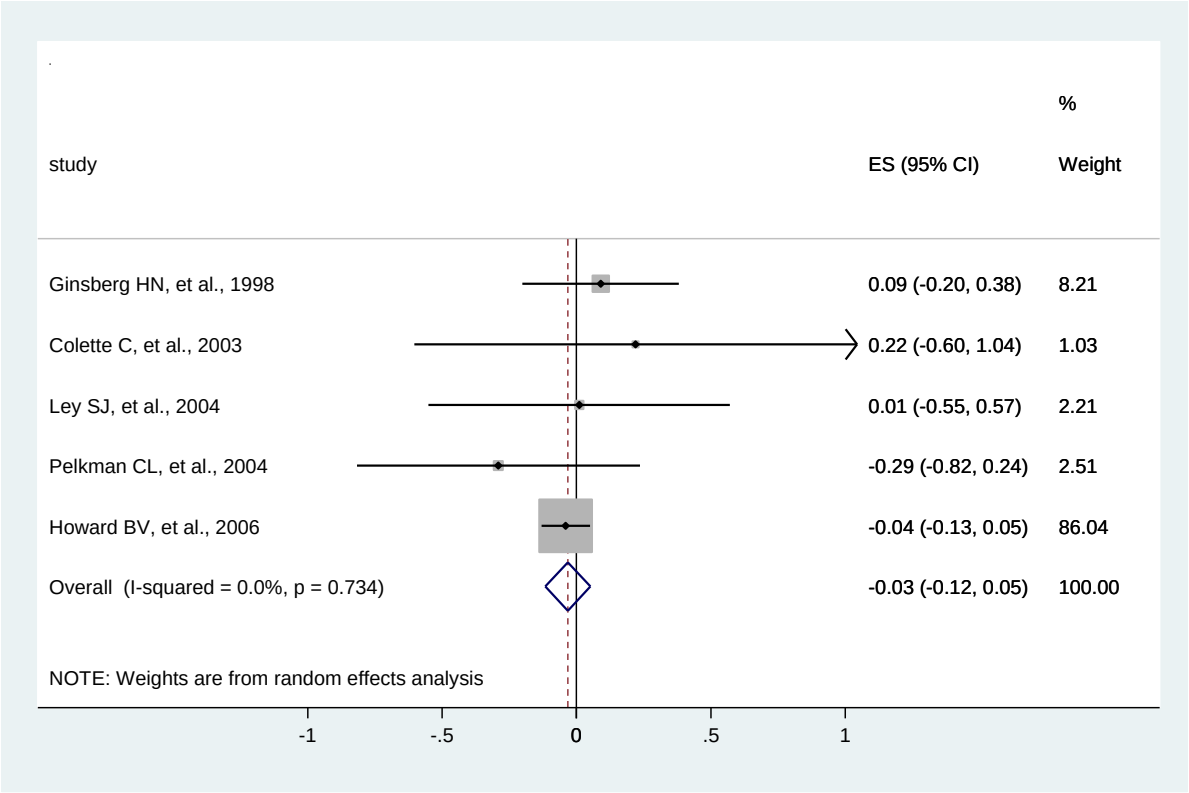


Table 8. Results of meta-analysis for higher carbohydrate, lower fat diets on fasting total cholesterol:HDL-cholesterol ratio

Model	Pooled mean difference estimate[1]		
	No.[2]	MD (95%CI)	Z (p-value)
Random effect	5	-0.03 (-0.12, 0.05)	0.75 (p=0.455)

[1] I^2 = 0.0%; p for test of heterogeneity = 0.734

[2] No. of mean difference estimates included in the pooled analysis.

Cereal fibre (excluding oat fibre) in relation to energy intake (report paragraphs 8.95-8.96)

34. One randomised controlled trial included in the original meta-analysis examined the relationship between cocoa bran and energy intake (Jenkins *et al.*, 2000). As cocoa bran is a legume fibre and not a cereal fibre the meta-analysis was performed with this trial excluded. Four randomised controlled trials were included in a meta-analysis (Tredger *et al.*, 1991; Sanders & Reddy, 1992; Jenkins *et al.*, 1999; Vuksan *et al.*, 1999).

35. No significant effect is demonstrated for cereal fibre (excluding oat fibre) consumption on energy intake (-269kJ, 95% CI (-826, 288 kJ; p=0.34).

Figure 19. Forest plot displaying randomised controlled trials investigating cereal fibre intake (excluding oat fibre) in relation to energy intake (KJ/day)

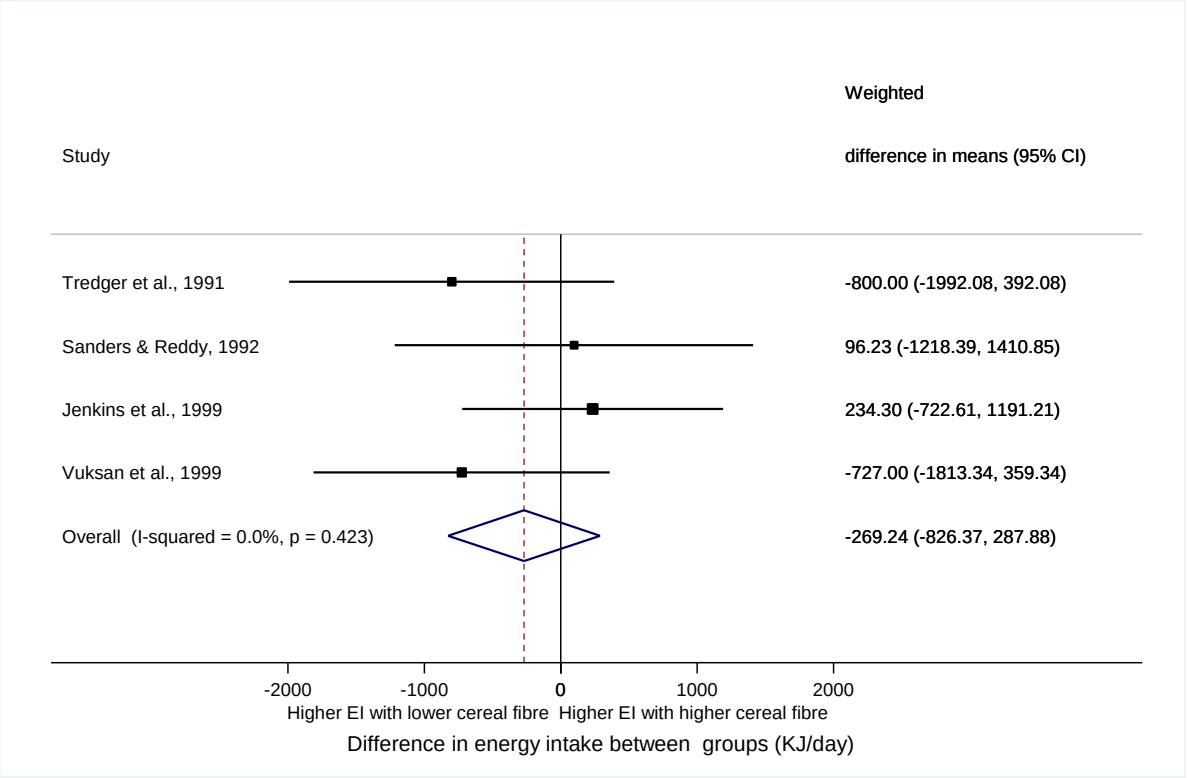


Table 9. Results of meta-analysis for cereal fibre intake (excluding oat fibre) in relation to energy intake (KJ/day)

Model	Pooled mean difference estimate[1]		
	No.[2]	MD (95%CI)	Z (p-value)
Random effect	4	-269KJ (-826, 288)	0.95 (p=0.344)

[1] I^2 = 0.0%; p for test of heterogeneity = 0.423

[2] No. of mean difference estimates included in the pooled analysis.

Glycaemic index and glycaemic load trials (report paragraphs 10.6 – 10.54)

36. In the cardio-metabolic health review, trials investigating glycaemic index (GI) and glycaemic load (GL) were combined into a single meta-analysis for each health outcome. To assist with interpreting the evidence, the trials were subsequently categorised into GI and GL studies and included in separate meta-analyses. Details of the conclusions drawn from these analyses can be found in Chapter 10 of the Carbohydrates and Health report.

37. The difference between these two types of trial is that the glycaemic index trials do not vary carbohydrate quantity, but change the quality to modify the GI. The GL trials reduce carbohydrate intake, resulting in an increased proportion of fat, including saturated fatty acids, and/or protein intake, as well as changing the carbohydrate quality to modify the glycaemic index. Both dietary strategies modify glycaemic index and glycaemic load, but the latter strategy modifies glycaemic load more and the former glycaemic index more.

38. In relation to the fasting lipid concentrations, one trial reported these as change from baseline (McMillan-Price *et al.*, 2006), whereas all other trials reported follow-up values. In the meta-analyses and corresponding forest plots, a positive number on the x-axis indicates that high GI/GL is greater than low GI/GL. For the weight loss meta-analyses and corresponding forest plots, both experimental groups have negative weight values, i.e. weight loss, and a positive value means less weight loss in the high GI/GL group.

Glycaemic index trials

Blood pressure (report paragraphs 10.12-10.13)

Figure 20. Forest plot for glycaemic index diets and diastolic blood pressure (mmHg)

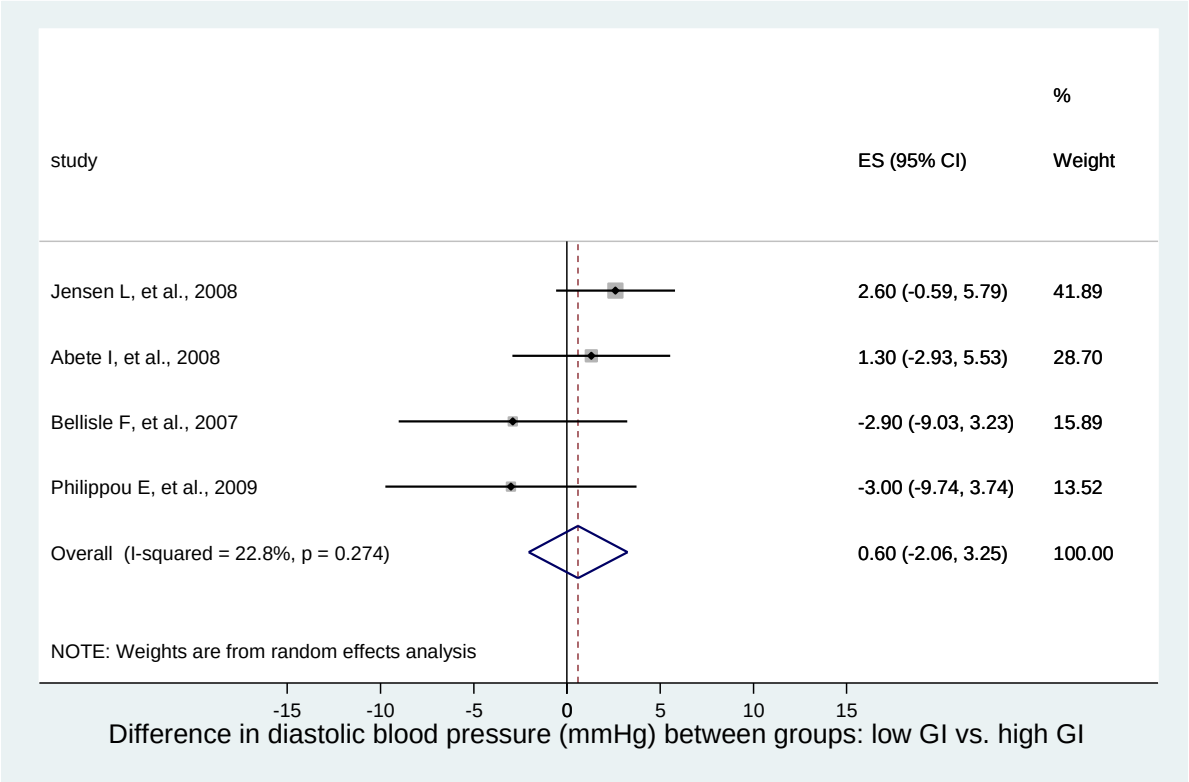


Table 10. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and diastolic blood pressure.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmHg (95%CI)	Z (p-value)
Random effect	4	0.60 (-2.06-3.25)	0.44 (p=0.660)

[1] I^2 = 22.8%; p for test of heterogeneity = 0.274

[2] No. of mean difference estimates included in the pooled analysis.

Figure 21. Forest plot for glycaemic index diets and systolic blood pressure (mmHg)

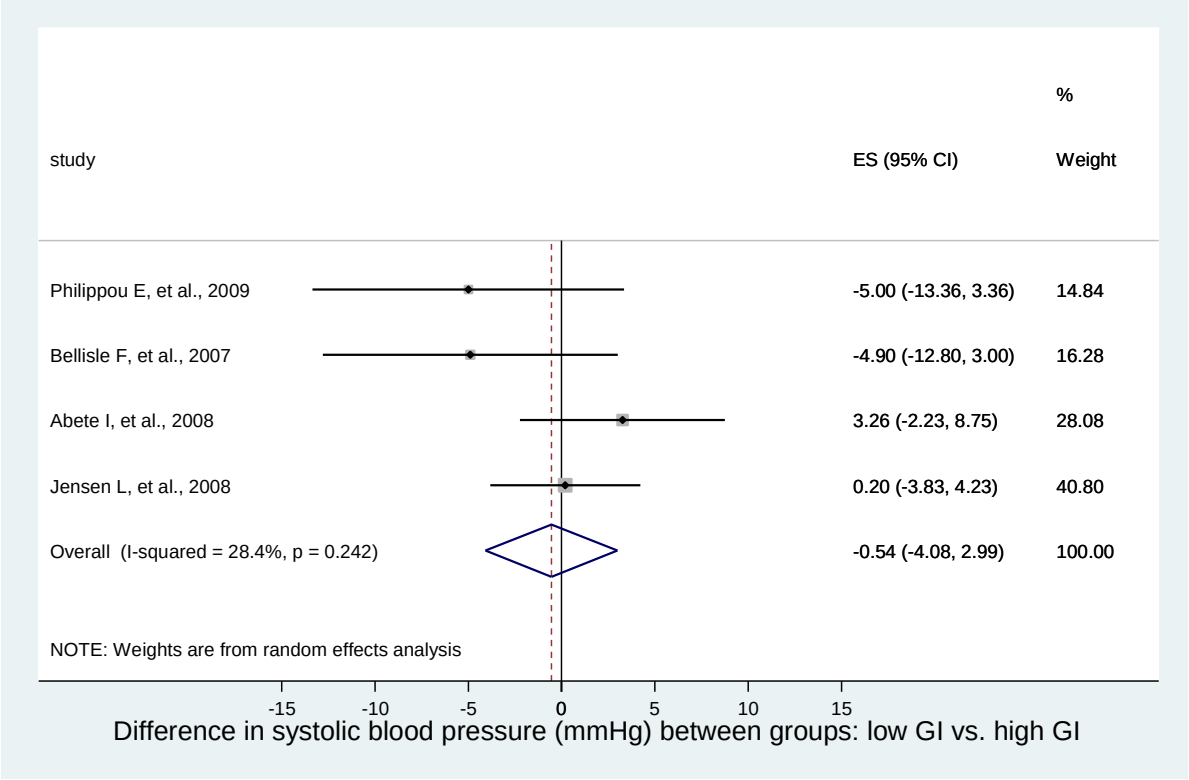


Table 11. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and systolic blood pressure.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmHg (95%CI)	Z (p-value)
Random effect	4	-0.54 (-4.08-2.99)	0.30 (p=0.764)

[1] I^2 = 28.4%; p for test of heterogeneity = 0.242

[2] No. of mean difference estimates included in the pooled analysis.

Fasting blood lipids (report paragraphs 10.14-10.20)

Figure 22. Forest plot for glycaemic index diets and fasting total cholesterol (mmol/L)

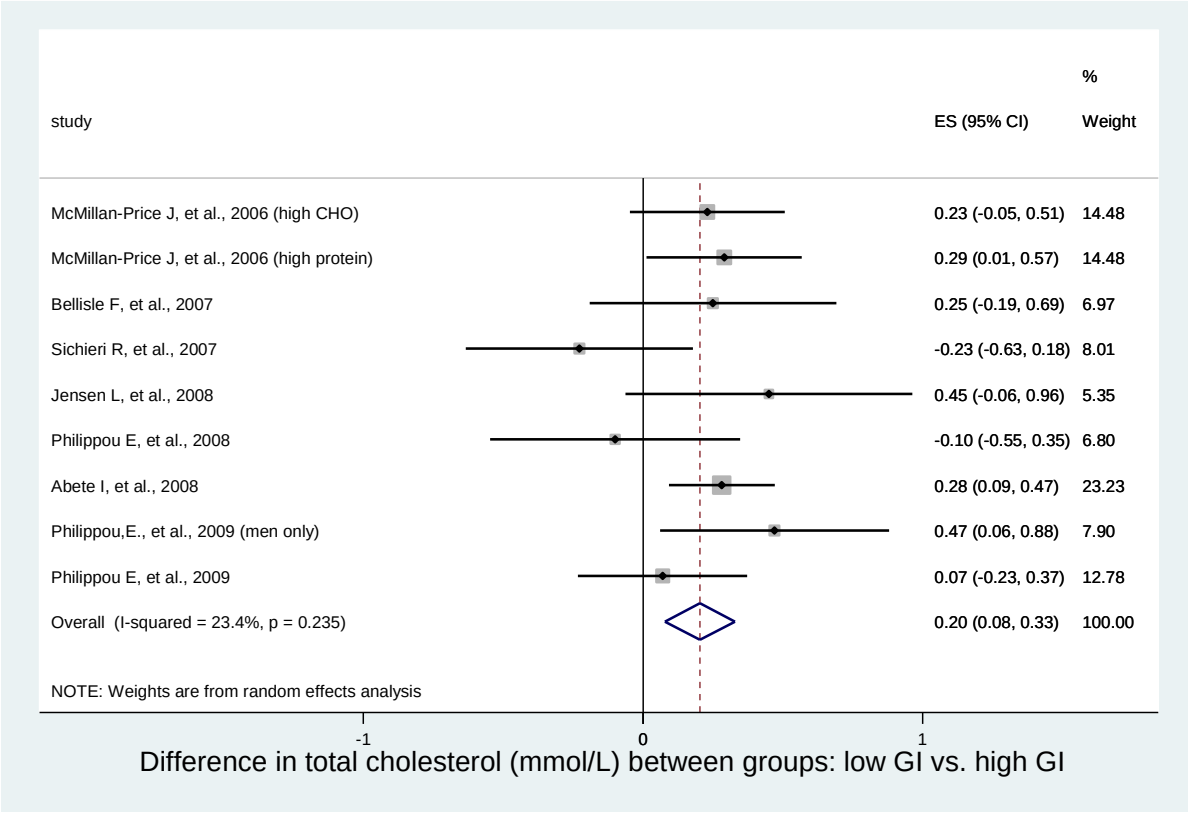


Table 12. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and fasting total cholesterol concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	9	0.20 (0.08-0.33)	3.17 (p=0.002)

[1] I^2 = 23.4%; p for test of heterogeneity = 0.235

[2] No. of mean difference estimates included in the pooled analysis.

Figure 23. Forest plot for glycaemic index diets and fasting LDL-cholesterol (mmol/L)

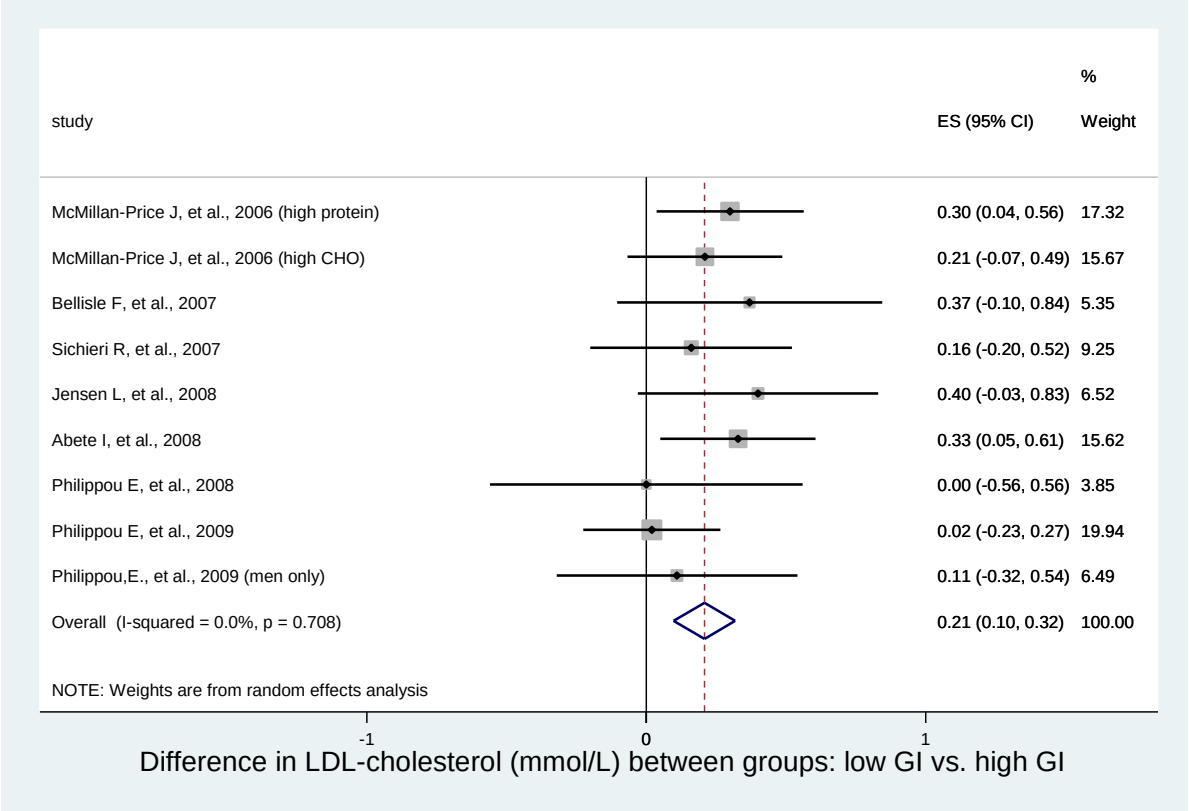


Table 13. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and fasting LDL-cholesterol concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	9	0.21 (0.10-0.32)	3.71 (p=0.000)

[1] I^2 = 0%; p for test of heterogeneity = 0.708

[2] No. of mean difference estimates included in the pooled analysis.

Figure 24. Forest plot of the total cholesterol and LDL-cholesterol trials above, showing mean difference in weight loss between low and high glycaemic index diets (kg) (report paragraph 10.16)

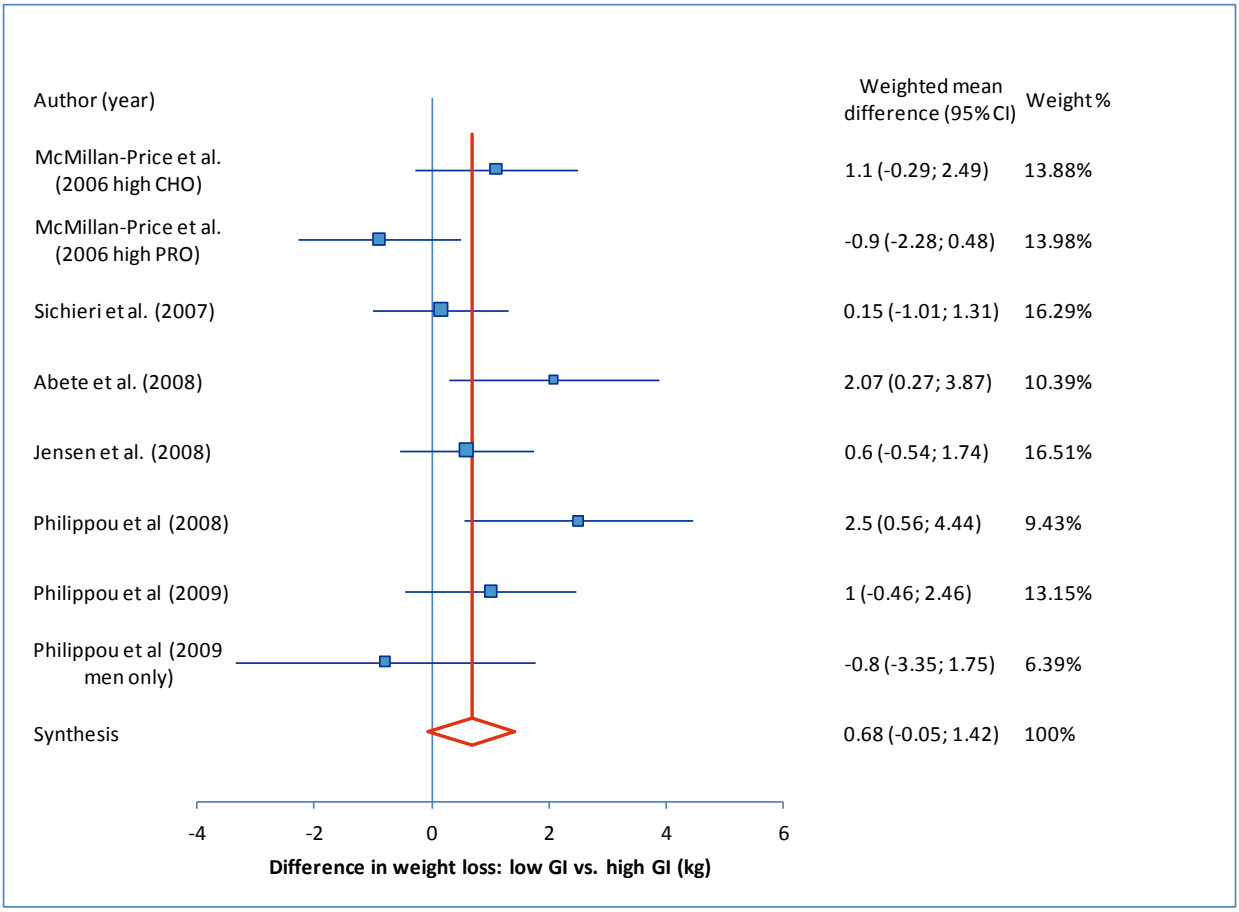


Table 14. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and mean difference in weight loss.

Model	Pooled mean difference estimate[1]		
	No.[2]	kg (95%CI)	Z (p-value)
Random effect	8	0.68 (-0.05-1.42)	1.81 (p=0.069)
Fixed effect	8	0.62 (0.10-1.13)	2.34 (p=0.019)

[1] I^2 = 47.60%; p for test of heterogeneity = 0.064

[2] No. of mean difference estimates included in the pooled analysis.

39. One trial does not report the weight loss from baseline, but gives total weights from which it is not possible to calculate the mean difference with variation. The difference in the mean weight loss values in this trial is -4.0kg for the low GI diet and -4.5kg for the high GI diet (Bellisle *et al.*, 2007).

Figure 25. Forest plot for glycaemic index diets and fasting HDL-cholesterol (mmol/L)

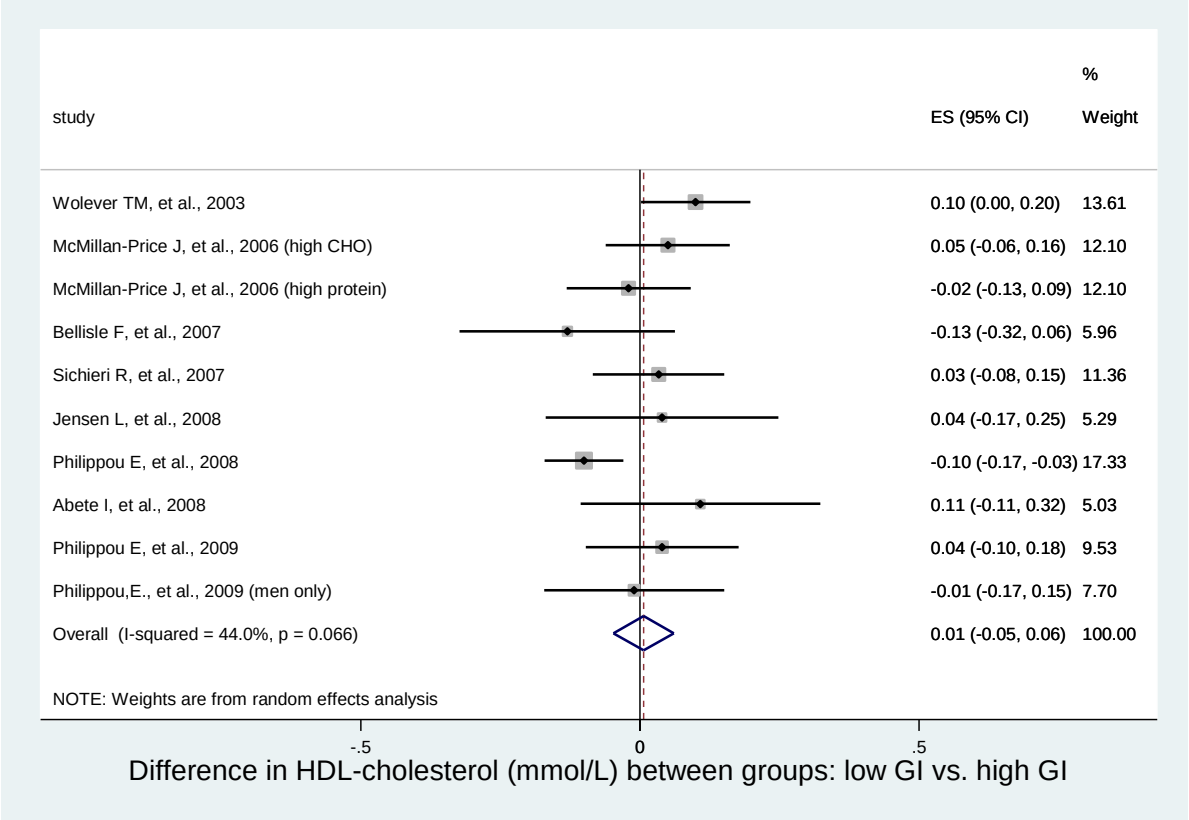


Table 15. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and fasting HDL-cholesterol concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	10	0.01 (-0.05-0.06)	0.24 (p=0.810)

[1] I^2 = 44.0%; p for test of heterogeneity = 0.066

[2] No. of mean difference estimates included in the pooled analysis.

Figure 26. Forest plot for glycaemic index diets and fasting triacylglycerol (mmol/L)

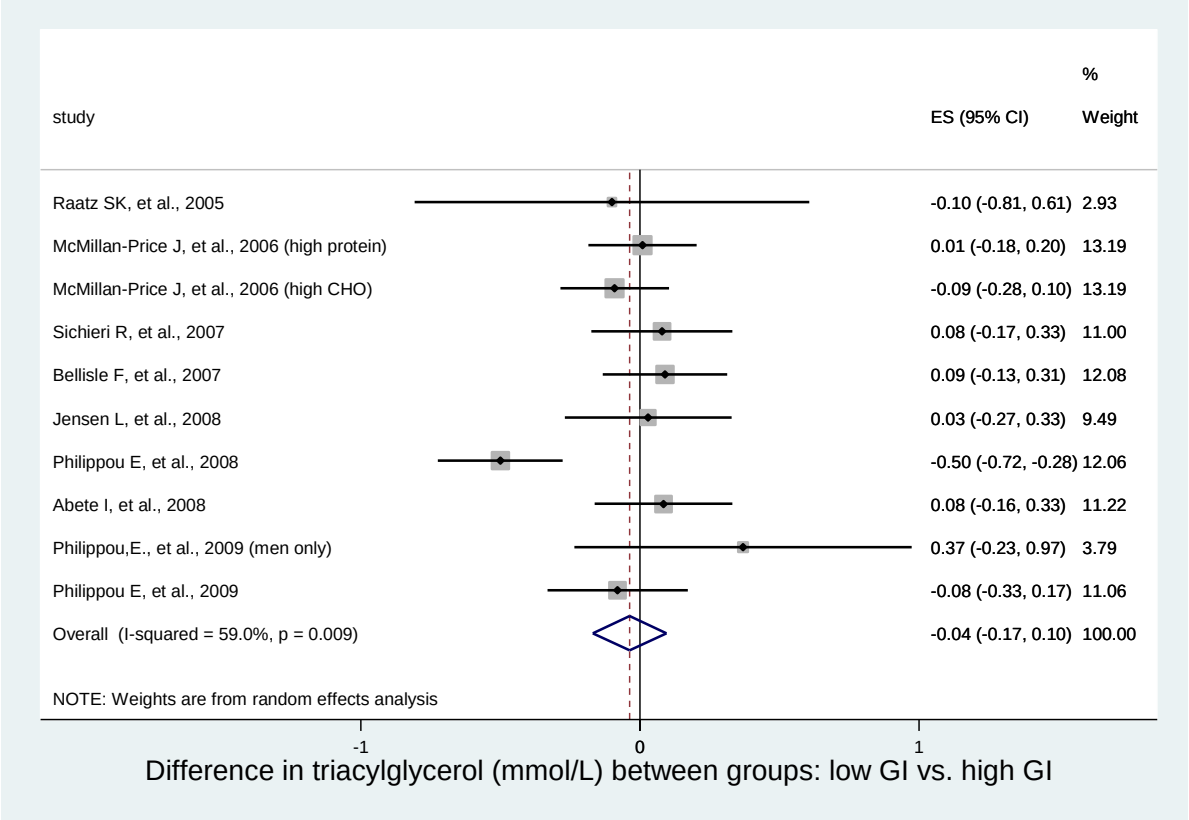


Table 16. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and fasting triacylglycerol concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	10	-0.04 (-0.17-0.10)	0.55 (p=0.586)

[1] I^2 = 59%; p for test of heterogeneity = 0.009

[2] No. of mean difference estimates included in the pooled analysis.

Figure 27. Forest plot for glycaemic index diets and fasting total cholesterol:HDL-cholesterol ratio

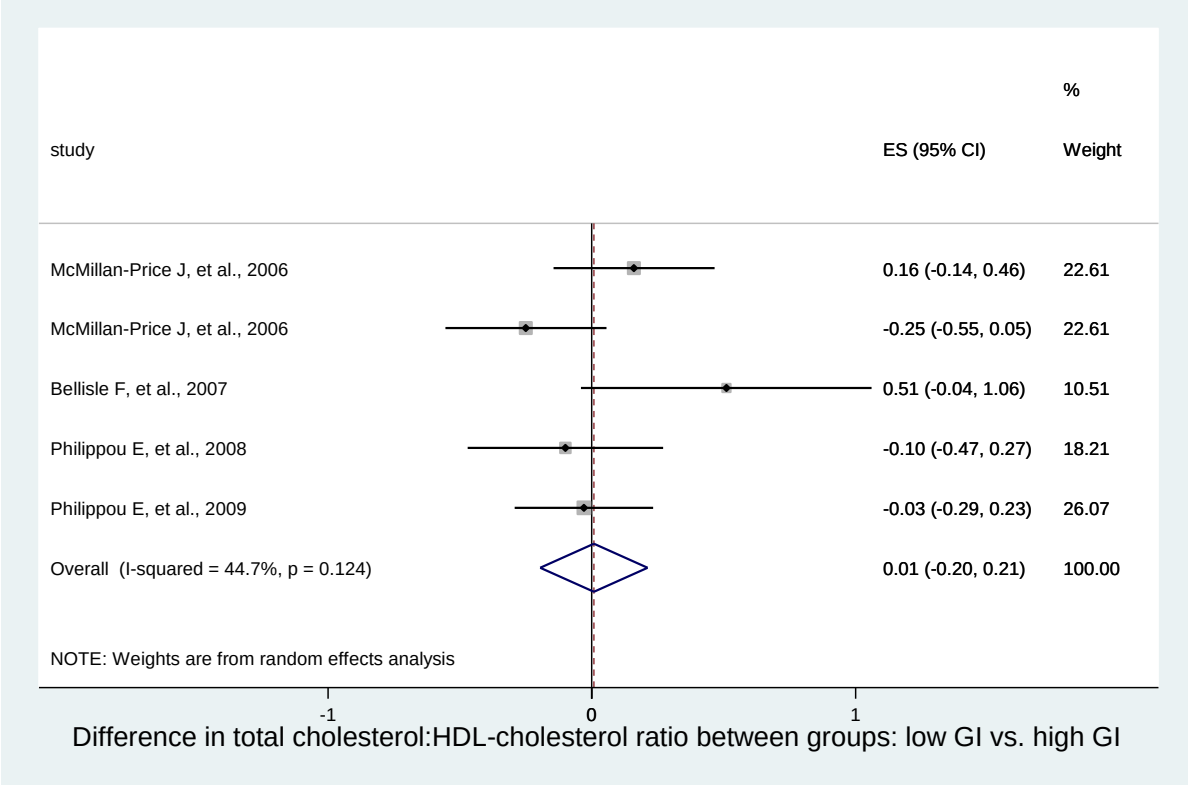


Table 17. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and fasting total cholesterol:HDL-cholesterol ratio.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD (95%CI)	Z (p-value)
Random effect	5	0.01 (-0.20-0.21)	0.07 (p=0.945)

[1] I^2 = 44.7% (95% CI 0.00-72.78); p for test of heterogeneity = 0.124

[2] No. of mean difference estimates included in the pooled analysis.

Figure 28. Forest plot for glycaemic index diets and fasting non-esterified fatty acids (mmol/L)

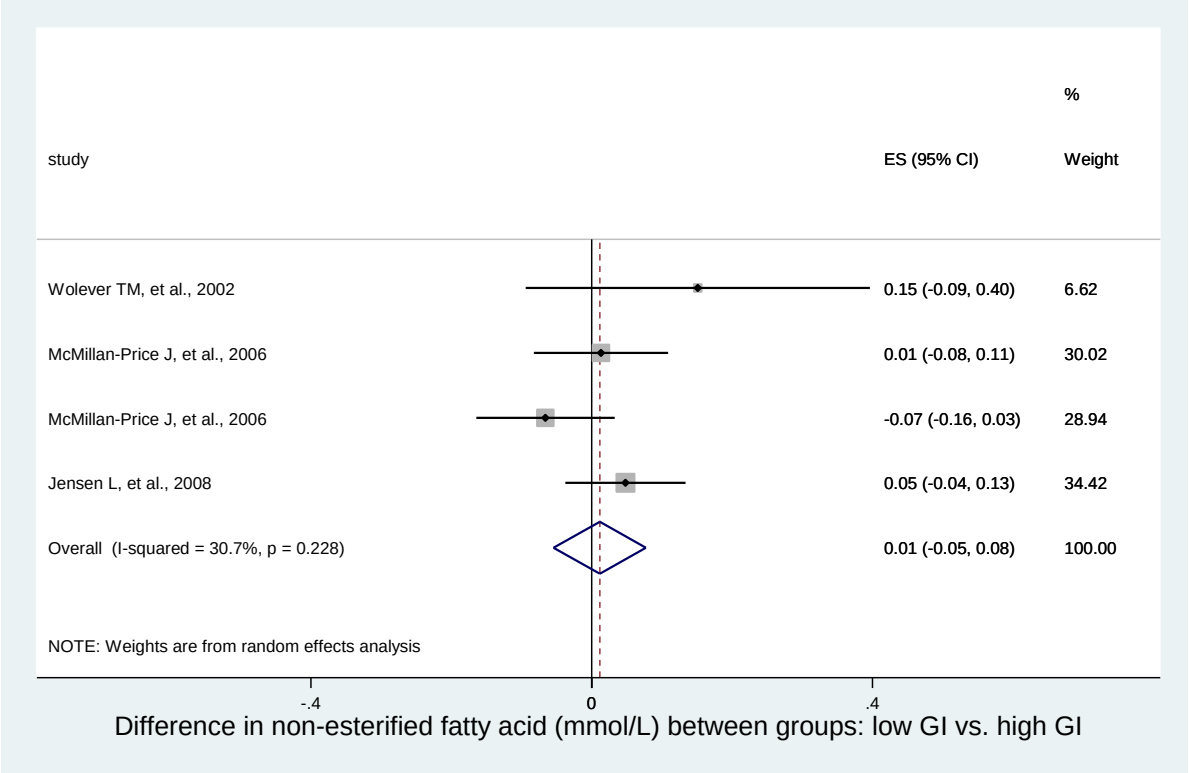


Table 18. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and fasting non-esterified fatty acid concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	4	0.01 (-0.05-0.08)	0.34 (p=0.736)

[1] I^2 = 30.7% (95% CI 0.00-72.78); p for test of heterogeneity = 0.228

[2] No. of mean difference estimates included in the pooled analysis.

Metabolic measures (report paragraphs 10.27-10.28)

Figure 29. Forest plot for glycaemic index diets and fasting blood glucose concentration (mmol/L)

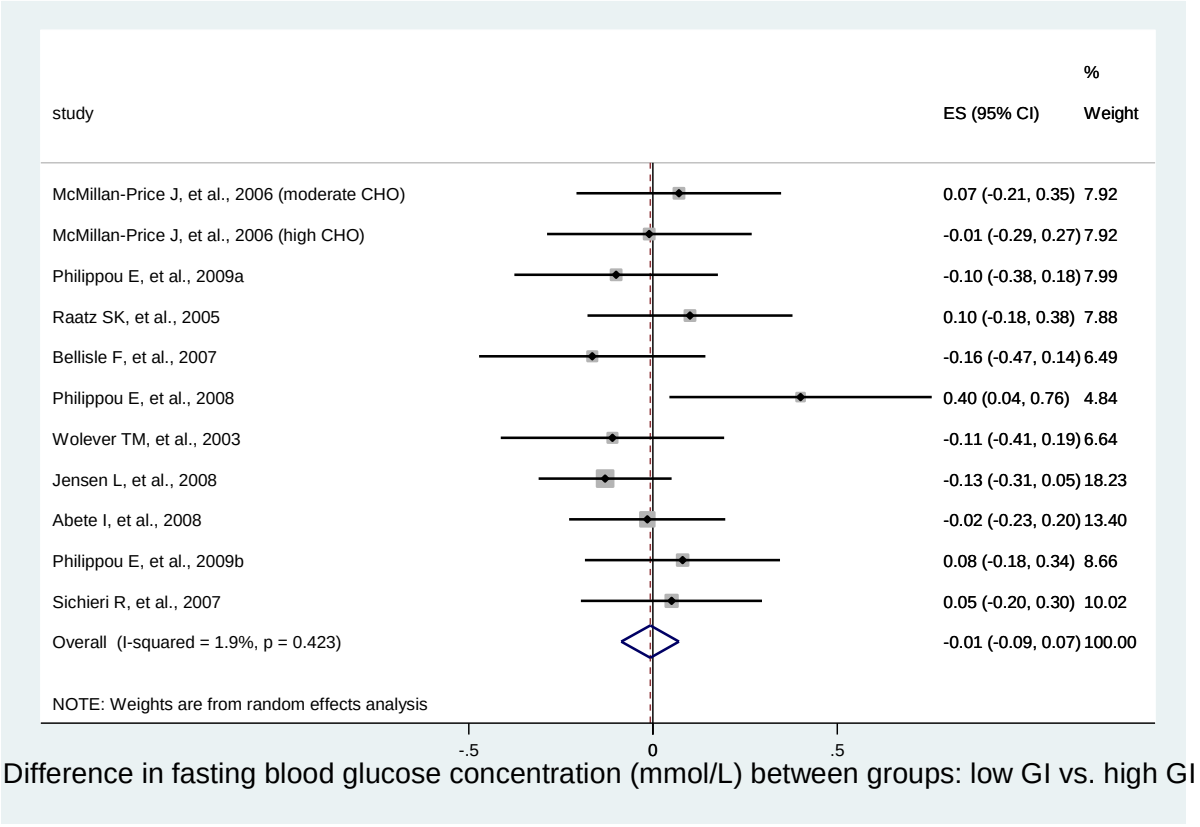


Table 19. Results of meta-analysis for low glycaemic index diet vs. high glycaemic index diet and fasting blood glucose concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	11	-0.01 (-0.09-0.07)	0.20 (p=0.845)

[1] I^2 = 1.9%; p for test of heterogeneity = 0.423

[2] No. of mean difference estimates included in the pooled analysis.

Glycaemic load trials

Blood pressure (report paragraphs 10.37-10.38)

Figure 30. Forest plot for glycaemic load diets and diastolic blood pressure (mmHg)

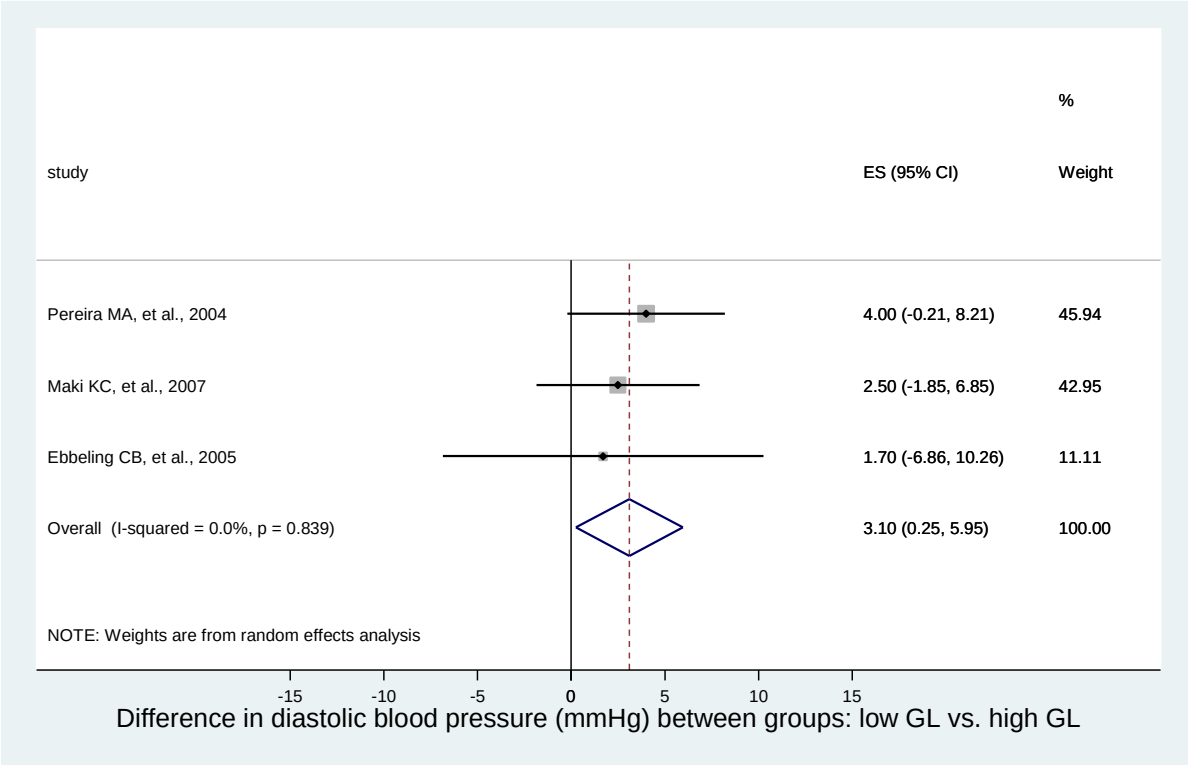


Table 20. Results of meta-analysis for low glycaemic load diet vs. high glycaemic load diet and diastolic blood pressure.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmHg (95%CI)	Z (p-value)
Random effect	3	3.10 (0.25-5.95)	2.13 (p=0.033)

[1] I^2 = 0.0%; p for test of heterogeneity = 0.839

[2] No. of mean difference estimates included in the pooled analysis.

Figure 31. Forest plot for glycaemic load diets and systolic blood pressure (mmHg)

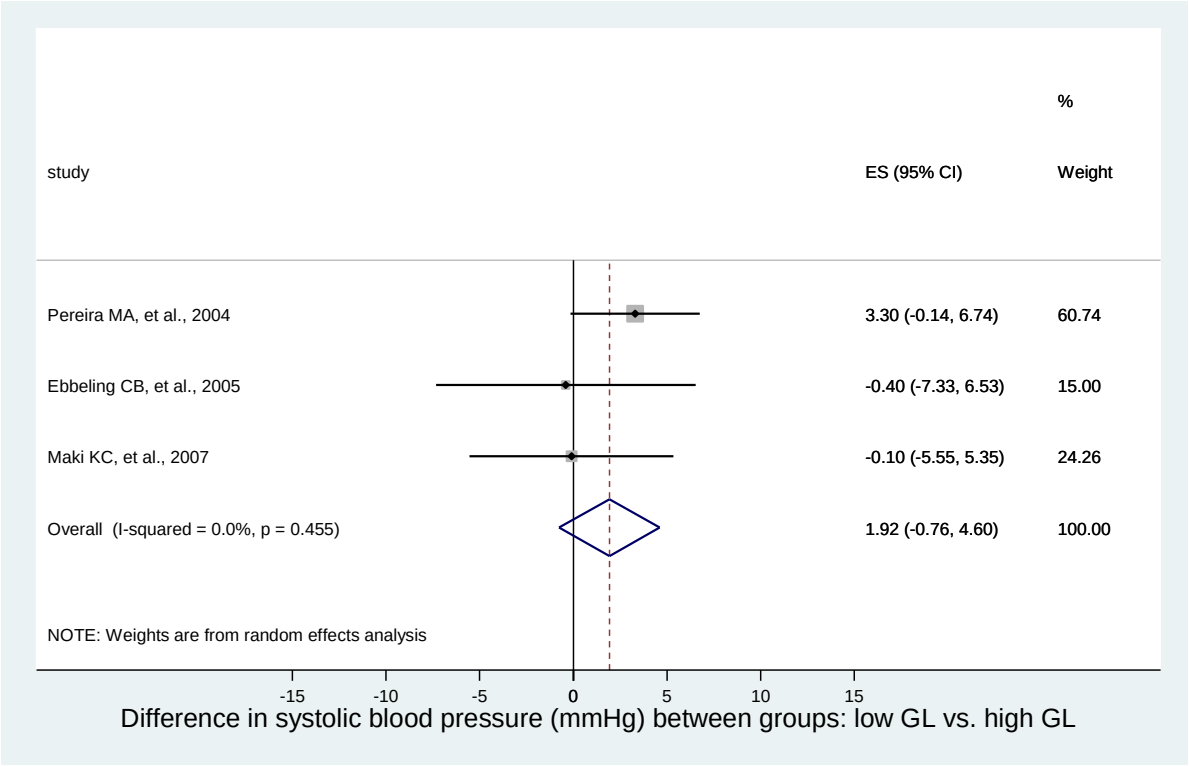


Table 21. Results of meta-analysis for low glycaemic load diet vs. high glycaemic load diet and systolic blood pressure.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmHg (95%CI)	Z (p-value)
Random effect	3	1.92 (-0.76-4.60)	1.40 (p=0.161)

[1] I^2 = 0.0%; p for test of heterogeneity = 0.455

[2] No. of MD estimates included in pooled analysis.

Fasting Blood lipids (report paragraphs 10.39-10.40)

Figure 32. Forest plot for glycaemic load diets and fasting total cholesterol (mmol/L)

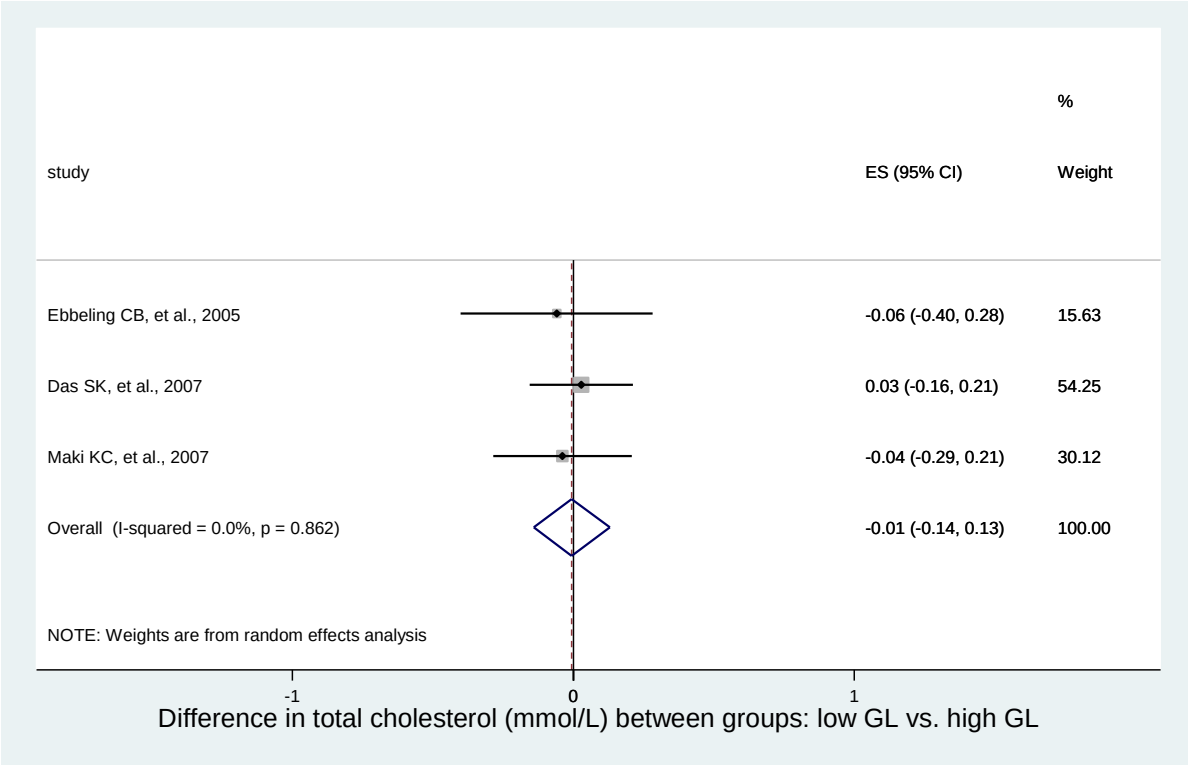


Table 22. Results of meta-analysis for low glycaemic load diet vs. high glycaemic load diet and fasting total cholesterol concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	3	-0.01 (-0.14-0.13)	0.08 (p=0.936)

[1] I^2 = 0%; p for test of heterogeneity = 0.862

[2] No. of mean difference estimates included in the pooled analysis.

Figure 33. Forest plot for glycaemic load diets and fasting LDL-cholesterol (mmol/L)

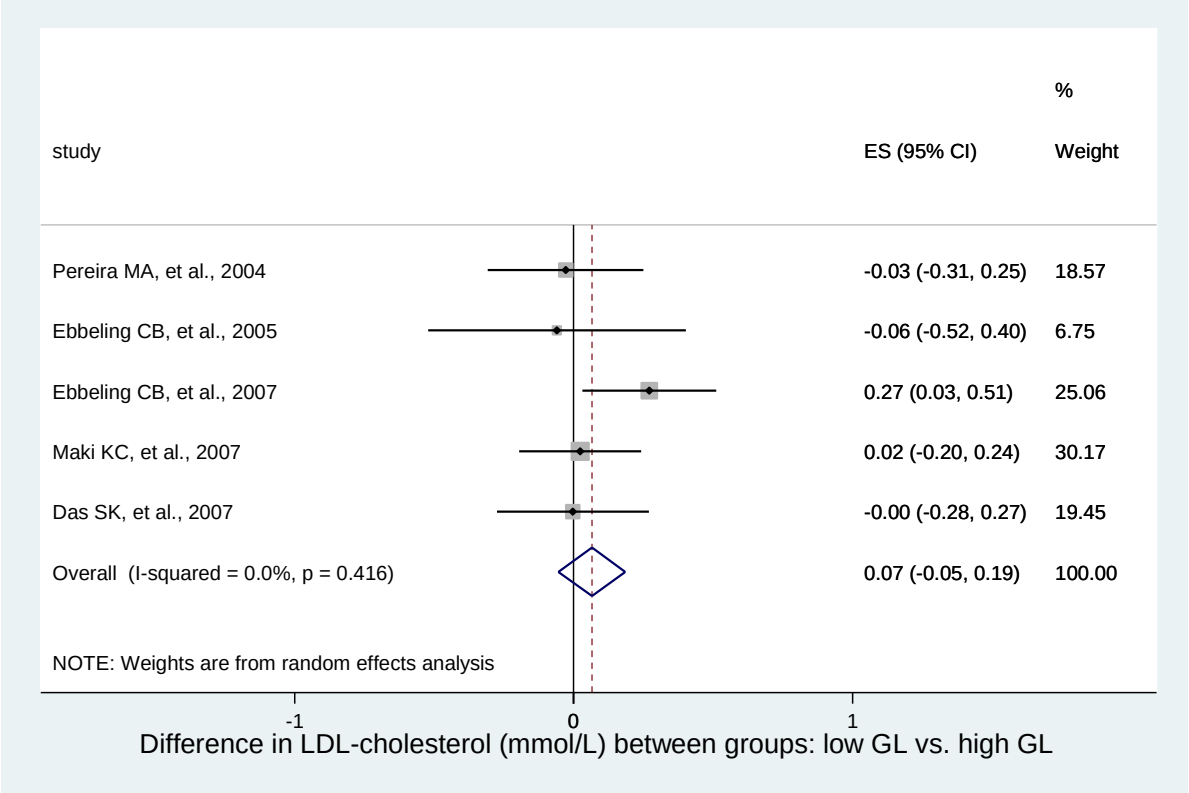


Table 23. Results of meta-analysis for low glycaemic load diet vs. high glycaemic load diet and fasting LDL-cholesterol concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	5	0.07 (-0.05-0.19)	1.06 (p=0.287)

[1] I^2 = 0%; p for test of heterogeneity = 0.416

[2] No. of mean difference estimates included in the pooled analysis.

Figure 34. Forest plot for glycaemic load diets and fasting HDL-cholesterol (mmol/L)

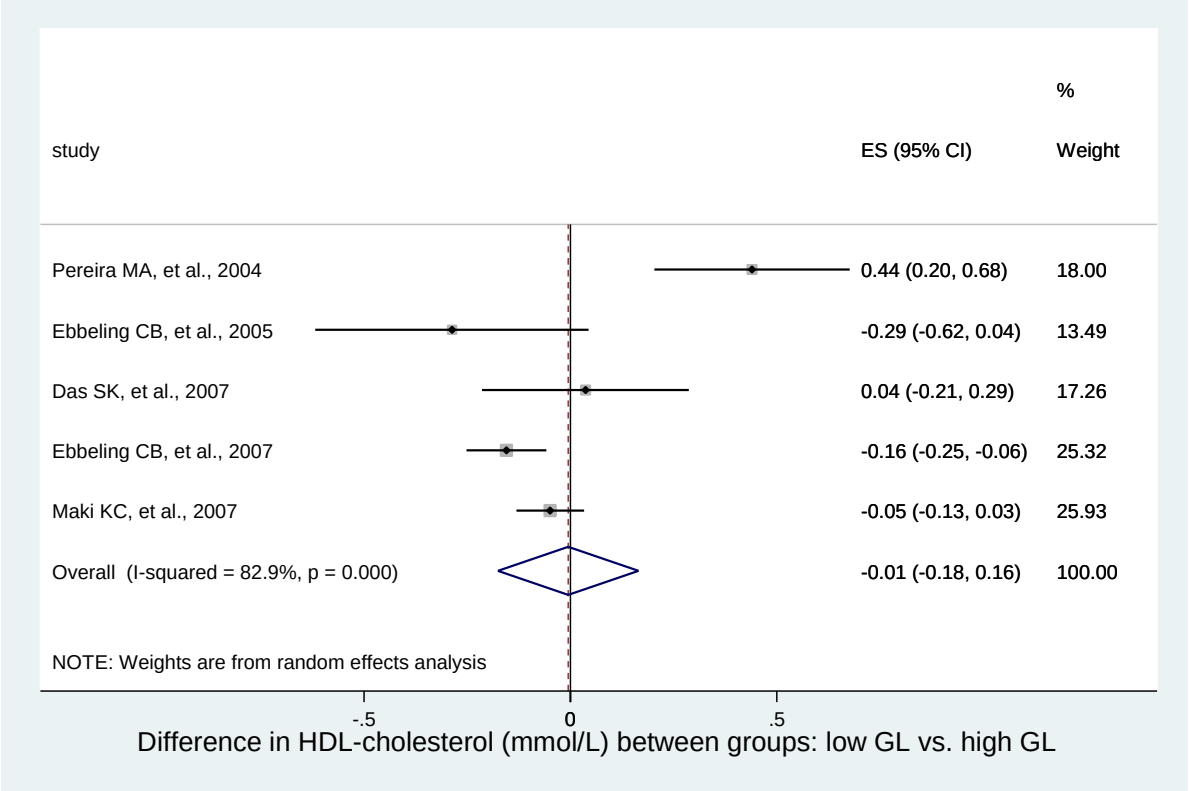


Table 24. Results of meta-analysis for low glycaemic load diet vs. high glycaemic load diet and fasting HDL-cholesterol concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	5	-0.01 (-0.18-0.16)	0.06 (p=0.951)

[1] I^2 = 82.9%; p for test of heterogeneity = 0.000

[2] No. of mean difference estimates included in the pooled analysis.

Figure 35. Forest plot for glycaemic load diets and fasting triacylglycerol (mmol/L)

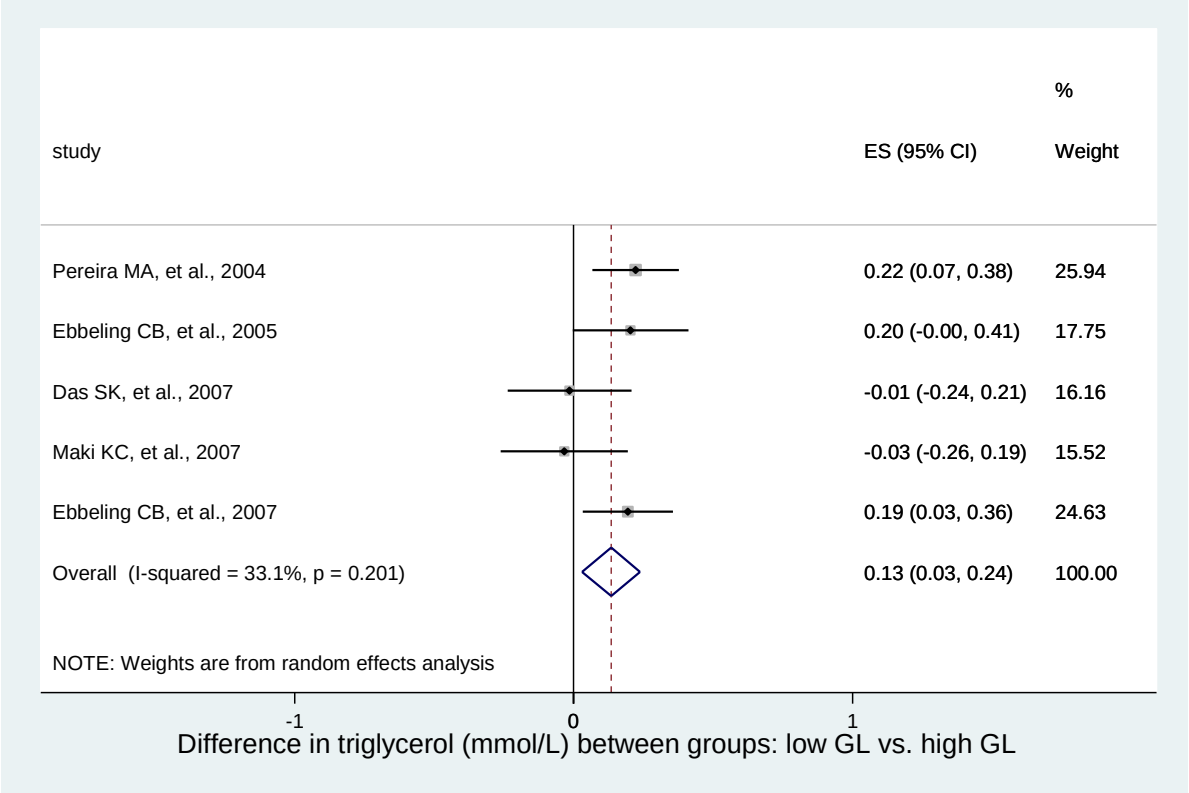


Table 25. Results of meta-analysis for low glycaemic load diet vs. high glycaemic load diet and fasting triacylglycerol concentration.

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	5	0.13 (0.03-0.24)	2.53 (p=0.012)

[1] I^2 = 33.1%; p for test of heterogeneity = 0.201

[2] No. of mean difference estimates included in the pooled analysis.

One trial reported on glycaemic load diets and fasting total cholesterol:HDL-cholesterol ratio (Maki *et al.*, 2007) and no trials reported on fasting non-esterified fatty acids.

Figure 36. Forest plot of trials above, showing mean difference in weight loss between low and high glycaemic load diets (kg)

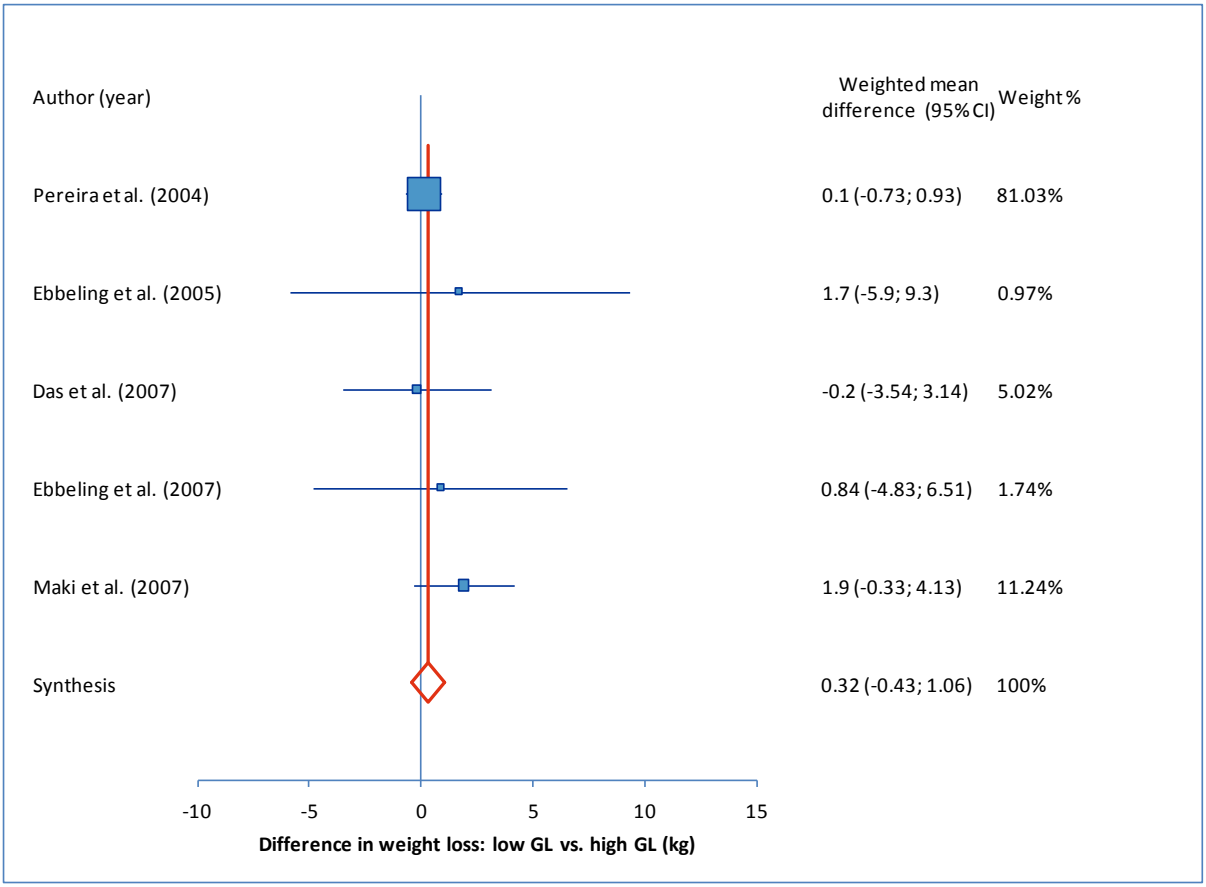


Table 26. Results of meta-analysis for low glycaemic load diet vs. high glycaemic load diet and mean difference in weight loss.

Model	Pooled mean difference estimate[1]		
	No.[2]	kg (95%CI)	Z (p-value)
Random effect	5	0.32 (-0.43-1.06)	0.82 (p=0.41)

[1] I^2 = 0.0%; p for test of heterogeneity = 0.655

[2] No. of mean difference estimates included in the pooled analysis.

N.B. data extracted from a figure for one trial (Ebbeling *et al.*, 2007)

Metabolic measures (report paragraphs 10.47-10.48)

Figure 37. Forest plot for glycaemic load diets and fasting blood glucose concentration (mmol/L)

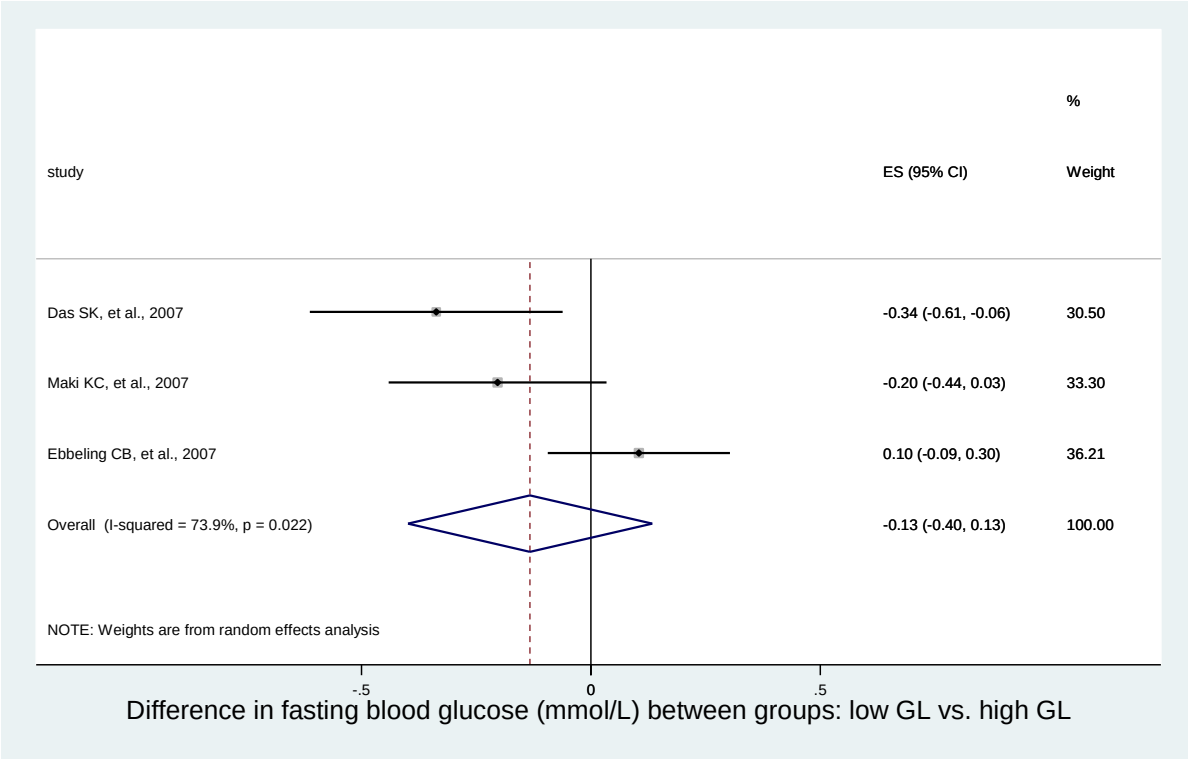


Table 27. Results of meta-analysis for low glycaemic load diet vs. high glycaemic load diet and fasting blood glucose concentration).

Model	Pooled mean difference estimate[1]		
	No.[2]	MD mmol/l (95%CI)	Z (p-value)
Random effect	3	-0.13 (-0.40-0.13)	1.40 (p=0.328)

[1] I^2 = 73.9%; p for test of heterogeneity = 0.022

[2] No. of MD estimates included in pooled analysis.

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