

Table of Contents

Supplementary Table 1: PRISMA Checklist.....	3
Supplementary Table 2. Search strategy for randomized controlled trials assessing the effect of oats and oat β -glucan on glycemic control, insulin sensitivity and beta-cell function	6
Supplementary Table 3. Risk of Bias of trial comparisons on the effect of oat β -glucan on HbA1c	8
Supplementary Table 4. Risk of Bias of trial comparisons on the effect of oat β -glucan on fasting glucose.....	8
Supplementary Table 5. Risk of Bias of trial comparisons on the effect of oat β -glucan on 2h-PG	9
Supplementary Table 6. Risk of Bias of trial comparisons on the effect of oat β -glucan on fasting insulin	9
Supplementary Table 7. Risk of Bias of trial comparisons on the effect of oat β -glucan on HOMA-IR.....	9
Supplementary Table 8. Sensitivity analyses of the use of correlation coefficient of 0.25 and 0.75 for paired analysis in the analysis of the effect of oat β -glucan on HbA1c, fasting glucose, 2h-PG, fasting insulin and HOMA-IR	10
Supplementary Table 9. GRADE assessment for the effect of oat β -glucan on HbA1c, fasting glucose, 2h-PG, fasting insulin and HOMA-IR.....	11
Supplementary Fig. 1. RoB summary on the effect of oat β -glucan on HbA1c	13
Supplementary Fig. 2. RoB summary on the effect of oat β -glucan on fasting glucose	13
Supplementary Fig. 3. RoB summary on the effect of oat β -glucan on 2h-PG.....	14
Supplementary Fig. 4. RoB summary on the effect of oat β -glucan on fasting insulin.....	14
Supplementary Fig. 5. RoB summary on the effect of oat β -glucan on HOMA-IR	15
Supplementary Fig. 6. Pooled effect estimates of oat β -glucan on HbA1c.....	16
Supplementary Fig. 7. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β -glucan on HbA1c	16
Supplementary Fig. 8. Pooled linear and non-linear dose-response relationship between oat β -glucan and HbA1c	17
Supplementary Fig. 9. Pooled effect estimates of oat β -glucan on fasting glucose	18
Supplementary Fig. 10. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β -glucan on fasting glucose	18
Supplementary Fig. 11. Pooled linear and non-linear dose-response relationship between oat β -glucan and fasting glucose	19
Supplementary Fig. 12. Pooled effect estimates of oat β -glucan on 2h-PG	20
Supplementary Fig. 13. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β -glucan on 2h-PG.....	20
Supplementary Fig. 14. Pooled linear dose-response relationship between oat β -glucan and 2h-PG.....	21
Supplementary Fig. 15. Pooled effect estimates of oat β -glucan on fasting insulin	22

Supplementary Fig. 16. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β-glucan on fasting insulin.....	22
Supplementary Fig. 17. Pooled linear dose-response relationship between oat β-glucan and fasting insulin	23
Supplementary Fig. 18. Pooled effect estimates of oat β-glucan on HOMA-IR	24
Supplementary Fig. 19. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β-glucan on HOMA-IR.....	24
Supplementary Fig. 20. Pooled linear dose-response relationship between oat β-glucan and HOMA-IR	25

Supplementary Table 1: PRISMA Checklist

Section and Topic	Item #	Checklist item	Location where item is reported
TITLE			
Title	1	Identify the report as a systematic review.	1
ABSTRACT			
Abstract	2	See the PRISMA 2020 for Abstracts checklist.	1
INTRODUCTION			
Rationale	3	Describe the rationale for the review in the context of existing knowledge.	1, 2
Objectives	4	Provide an explicit statement of the objective(s) or question(s) the review addresses.	2
METHODS			
Eligibility criteria	5	Specify the inclusion and exclusion criteria for the review and how studies were grouped for the syntheses.	2
Information sources	6	Specify all databases, registers, websites, organisations, reference lists and other sources searched or consulted to identify studies. Specify the date when each source was last searched or consulted.	2
Search strategy	7	Present the full search strategies for all databases, registers and websites, including any filters and limits used.	2, Table S2
Selection process	8	Specify the methods used to decide whether a study met the inclusion criteria of the review, including how many reviewers screened each record and each report retrieved, whether they worked independently, and if applicable, details of automation tools used in the process.	2
Data collection process	9	Specify the methods used to collect data from reports, including how many reviewers collected data from each report, whether they worked independently, any processes for obtaining or confirming data from study investigators, and if applicable, details of automation tools used in the process.	2
Data items	10a	List and define all outcomes for which data were sought. Specify whether all results that were compatible with each outcome domain in each study were sought (e.g. for all measures, time points, analyses), and if not, the methods used to decide which results to collect.	2, 3
	10b	List and define all other variables for which data were sought (e.g. participant and intervention characteristics, funding sources). Describe any assumptions made about any missing or unclear information.	2
Study risk of bias assessment	11	Specify the methods used to assess risk of bias in the included studies, including details of the tool(s) used, how many reviewers assessed each study and whether they worked independently, and if applicable, details of automation tools used in the process.	2
Effect measures	12	Specify for each outcome the effect measure(s) (e.g. risk ratio, mean difference) used in the synthesis or presentation of results.	3
Synthesis methods	13a	Describe the processes used to decide which studies were eligible for each synthesis (e.g. tabulating the study intervention characteristics and comparing against the planned groups for each synthesis (item #5)).	3
	13b	Describe any methods required to prepare the data for presentation or synthesis, such as handling of missing summary statistics, or data conversions.	2, 3
	13c	Describe any methods used to tabulate or visually display results of individual studies and syntheses.	3
	13d	Describe any methods used to synthesize results and provide a rationale for the choice(s). If meta-analysis was performed, describe the model(s), method(s) to identify the presence and extent of statistical heterogeneity, and software package(s) used.	3
	13e	Describe any methods used to explore possible causes of heterogeneity among study results (e.g. subgroup analysis, meta-regression).	3

Section and Topic	Item #	Checklist item	Location where item is reported
	13f	Describe any sensitivity analyses conducted to assess robustness of the synthesized results.	3
Reporting bias assessment	14	Describe any methods used to assess risk of bias due to missing results in a synthesis (arising from reporting biases).	2, 3
Certainty assessment	15	Describe any methods used to assess certainty (or confidence) in the body of evidence for an outcome.	3
RESULTS			
Study selection	16a	Describe the results of the search and selection process, from the number of records identified in the search to the number of studies included in the review, ideally using a flow diagram.	3, Fig. 1
	16b	Cite studies that might appear to meet the inclusion criteria, but which were excluded, and explain why they were excluded.	N/A
Study characteristics	17	Cite each included study and present its characteristics.	3, 4, 6, Table 1
Risk of bias in studies	18	Present assessments of risk of bias for each included study.	6, Table S3-S7, Fig. S1-S5
Results of individual studies	19	For all outcomes, present, for each study: (a) summary statistics for each group (where appropriate) and (b) an effect estimate and its precision (e.g. confidence/credible interval), ideally using structured tables or plots.	6-8, Fig. 2, Fig, S6, S9, S12, S15, S18
Results of syntheses	20a	For each synthesis, briefly summarise the characteristics and risk of bias among contributing studies.	6, Table 1, Table S3-S7, Fig. S1-S5
	20b	Present results of all statistical syntheses conducted. If meta-analysis was done, present for each the summary estimate and its precision (e.g. confidence/credible interval) and measures of statistical heterogeneity. If comparing groups, describe the direction of the effect.	6-8, Fig. 2, Fig, S6, S9, S12, S15, S18
	20c	Present results of all investigations of possible causes of heterogeneity among study results.	6-8, Table S8, Fig. S7, S10, S13, S16, S19
	20d	Present results of all sensitivity analyses conducted to assess the robustness of the synthesized results.	6-8, Table S8, Fig. S7, S10, S13, S16, S19
Reporting biases	21	Present assessments of risk of bias due to missing results (arising from reporting biases) for each synthesis assessed.	6, Table S3-S7, Fig. S1-S5

Section and Topic	Item #	Checklist item	Location where item is reported
Certainty of evidence	22	Present assessments of certainty (or confidence) in the body of evidence for each outcome assessed.	8, Fig. 2, Table S9
DISCUSSION			
Discussion	23a	Provide a general interpretation of the results in the context of other evidence.	8, 9
	23b	Discuss any limitations of the evidence included in the review.	9
	23c	Discuss any limitations of the review processes used.	9
	23d	Discuss implications of the results for practice, policy, and future research.	9
OTHER INFORMATION			
Registration and protocol	24a	Provide registration information for the review, including register name and registration number, or state that the review was not registered.	1, 2
	24b	Indicate where the review protocol can be accessed, or state that a protocol was not prepared.	1, 2
	24c	Describe and explain any amendments to information provided at registration or in the protocol.	N/A
Support	25	Describe sources of financial or non-financial support for the review, and the role of the funders or sponsors in the review.	10
Competing interests	26	Declare any competing interests of review authors.	10
Availability of data, code and other materials	27	Report which of the following are publicly available and where they can be found: template data collection forms; data extracted from included studies; data used for all analyses; analytic code; any other materials used in the review.	11

Supplementary Table 2. Search strategy for randomized controlled trials assessing the effect of oats and oat β -glucan on glycemic control, insulin sensitivity and beta-cell function

MEDLINE 1946 to – 6 June 2021	EMBASE 1947 to – 6 June 2021	Cochrane Central Register of Controlled Trials Through 6 June 2021
1. exp Dietary Fiber/ 2. dietary fiber.mp. 3. exp Avena/ 4. avena sativa.mp. 5. exp beta-Glucans/ 6. beta glucan.mp. 7. b-glucans.mp. 8. 1 or 2 or 3 or 4 or 5 or 6 or 7 9. exp Diabetes Mellitus/ 10. Diabetes type 2.mp. 11. Non insulin dependent diabetes mellitus.mp. 12. NIDDM.mp. 13. Type II diabetes.mp. 14. Type 2 Diabetes.mp. 15. T2DM.mp. 16. metabolic syndrome.mp. 17. exp Hemoglobin A, Glycosylated/ 18. Hemoglobin A1c/ 19. Hemoglobin A1c.mp. 20. hba1c.mp. 21. exp Glucose/ 22. exp Hyperglycemia/ 23. (blood adj3 glucose).mp. 24. glucose blood level/ 25. Glyc?emi*.mp. 26. Hyperglyc?emi*.mp. 27. Hypoglyc?emi*.mp. 28. insulin*.mp. 29. hyperinsulin*.mp. 30. Fasting insulin.mp. 31. Insulin resistance/ 32. HOMA*.mp. 33. Matsuda index.mp. 34. OGTT.mp. 35. FSIGT.mp. 36. euglyc?emic.mp.	1. exp Dietary Fiber/ 2. dietary fiber.mp. 3. exp Avena/ 4. avena sativa.mp. 5. exp beta-Glucans/ 6. beta glucan.mp. 7. b-glucans.mp. 8. 1 or 2 or 3 or 4 or 5 or 6 or 7 9. exp diabetes mellitus/ 10. Diabetes type 2.mp. 11. Non insulin dependent diabetes mellitus.mp. 12. NIDDM.mp. 13. Type II diabetes.mp. 14. Type 2 Diabetes.mp. 15. T2DM.mp. 16. exp metabolic syndrome X/ 17. exp hemoglobin A1c/ 18. Hemoglobin A1c/ 19. Hemoglobin A1c.mp. 20. hba1c.mp. 21. exp glucose/ 22. exp hyperglycemia/ 23. (blood adj3 glucose).mp. 24. glucose blood level/ 25. Glyc?emi*.mp. 26. Hyperglyc?emi*.mp. 27. Hypoglyc?emi*.mp. 28. insulin*.mp. 29. hyperinsulin*.mp. 30. Fasting insulin.mp. 31. Insulin resistance/ 32. HOMA*.mp. 33. Matsuda index.mp. 34. OGTT.mp. 35. FISGT.mp. 36. euglyc?emic.mp.	1. Dietary Fiber/ 2. dietary fiber.mp. 3. avena.mp. 4. avena sativa.mp. 5. beta-glucans/ 6. beta glucan.mp. 7. 1 or 2 or 3 or 4 or 5 or 6 8. Diabetes Mellitus/ 9. Diabetes type 2. ti,ab,kw. 10. Non Insulin dependent diabetes. ti,ab,kw. 11. NIDDM. ti,ab,kw. 12. Type 2 diabetes. ti,ab,kw. 13. T2DM. ti,ab,kw. 14. Adult-onset diabetes. ti,ab,kw. 15. metabolic syndrome.mp. 16. Hemoglobin A, Glycosylated/ 17. HbA1c. ti,ab,kw. 18. hba1c. ti,ab,kw. 19. Glucose/ 20. Hyperglycemia/ 21. (Blood adj3 glucose). ti,ab,kw. 22. Blood Glucose/ 23. Glyc?emi*.ti,ab,kw. 24. Hyperglyc?emi*.ti,ab,kw. 25. Hypoglyc?emi*.ti,ab,kw. 26. insulin*. ti,ab,kw. 27. hyperinsulin*. ti,ab,kw. 28. Fasting insulin. ti,ab,kw. 29. Insulin Resistance/ 30. HOMA*. ti,ab,kw. 31. Matsuda index. ti,ab,kw. 32. OGTT. ti,ab,kw. 33. FSIGT. ti,ab,kw. 34. euglycemic. ti,ab,kw. 35. euglycemic glucose

37. euglyc?emic glucose clamp.mp.	37. euglyc?emic glucose clamp.mp.	clamp. ti,ab,kw.
38. euglyc?emic clamp.mp.	38. euglyc?emic clamp.mp.	36. eyglycemic clamp. ti,ab,kw.
39. glucose clamp.mp.	39. glucose clamp.mp.	37. glucose clamp. ti,ab,kw.
40. beta cell function.mp.	40. beta cell function.mp.	38. beta cell function. ti,ab,kw.
41. beta cell dysfunction.mp.	41. beta cell dysfunction.mp.	39. beta cell dysfunction. ti,ab,kw.
42. insulin secretion index.mp.	42. insulin secretion index.mp.	40. insulin secretion index. ti,ab,kw.
43. ISSI-2.mp.	43. ISSI-2.mp.	41. ISSI-2. ti,ab,kw.
44. 9 or 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29 or 30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42 or 43	44. 9 or 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29 or 30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42 or 43	42. ISSI-2. ti,ab,kw.
45. 8 and 44	45. 8 and 44	43. 9 or 10 or 11 or 12 or 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29 or 30 or 31 or 32 or 33 or 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42
46. clinical trial.mp.	46. clinical trial.mp.	44. 7 and 44
47. clinical trial.pt.	47. clinical tria:.mp.	
48. random:.mp.	48. random:.mp.	
49. 46 or 47 or 48	49. 46 or 47 or 48	
50. 45 and 49	50. 45 and 49	
51. limit 50 to animals	51. limit 50 to animals	
52. 50 not 51	52. 50 not 51	

Supplementary Table 3. Risk of Bias of trial comparisons on the effect of oat β -glucan on HbA1c

Study ID	Experimental	Comparator	Randomization process	Deviations from intended interventions	Missing outcome data	Measurement of the outcome	Selection of the reported result	Overall
Stevens et al 1985	Oat bran in cereal or muffin	No dietary intervention	?	+	+	+	+	!
Kabir et al 2002	Oat β -glucan in cereal	Wheat bread	+	+	+	+	+	+
Liatis 2009	Oat β -glucan enriched bread	Wheat bread	+	+	+	+	+	+
Cugnet-Anceau et al 2010	Oat β -glucan enriched soup	Soup without oat β -glucan	+	+	?	+	+	!
McGeoch et al 2013	Oat products	Standard dietary advice	+	+	+	+	+	+
Ballesteros 2015	40g oatmeal	1 egg	+	+	+	+	+	+
Li et al 2016	50g whole grain oats	No dietary intervention	+	+	+	+	+	+
Li et al 2016	100g whole grain oats	No dietary intervention	+	+	+	+	+	+

+ Low risk
? Some concerns
! High risk

Supplementary Table 4. Risk of Bias of trial comparisons on the effect of oat β -glucan on fasting glucose

Study ID	Experimental	Comparator	Randomization process	Deviations from intended interventions	Missing outcome data	Measurement of the outcome	Selection of the reported result	Overall
Stevens et al 1985	Oat bran in cereal or muffin	No dietary intervention	?	+	+	+	+	!
Kabir et al 2002	Oat β -glucan in cereal	Wheat bread	+	+	+	+	+	+
Liatis 2009	Oat β -glucan enriched bread	Wheat bread	+	+	+	+	+	+
Cugnet-Anceau et al 2010	Oat β -glucan enriched soup	Soup without oat β -glucan	+	+	?	+	+	!
McGeoch et al 2013	Oat products	Standard dietary advice	+	+	+	+	+	+
Ballesteros 2015	40g oatmeal	1 egg	+	+	+	+	+	+
Li et al 2016	50g whole grain oats	No dietary intervention	+	+	+	+	+	+
Li et al 2016	100g whole grain oats	No dietary intervention	+	+	+	+	+	+

+ Low risk
? Some concerns
! High risk

Supplementary Table 5. Risk of Bias of trial comparisons on the effect of oat β -glucan on 2h-PG

Study ID	Experimental	Comparator	Randomization process	Deviations from intended interventions	Missing outcome data	Measurement of the outcome	Selection of the reported result	Overall	
McGeoch 2013	Oat products	Standard dietary advice	1	+	+	+	+	+	Low risk
Li 2016	50g whole grain oats	No dietary intervention	1	+	+	+	+	+	Some concerns
Li 2016	100g whole grain oats	No dietary intervention	1	+	+	+	+	+	High risk

Supplementary Table 6. Risk of Bias of trial comparisons on the effect of oat β -glucan on fasting insulin

Study ID	Experimental	Comparator	Randomization process	Deviations from intended interventions	Missing outcome data	Measurement of the outcome	Selection of the reported result	Overall	
Kabir et al 2002	Oat β -glucan in cereal	Wheat bread	+	+	+	+	+	+	Low risk
Liatis et al 2009	Oat β -glucan enriched bread	Wheat bread	+	+	+	+	+	+	Some concerns
McGeoch et al 2013	Oat products	Standard dietary advice	+	+	+	+	+	+	High risk
Ballestros et al 2015	40g oatmeal	1 egg	+	+	+	+	+	+	

Supplementary Table 7. Risk of Bias of trial comparisons on the effect of oat β -glucan on HOMA-IR

Study ID	Experimental	Comparator	Randomization process	Deviations from intended interventions	Missing outcome data	Measurement of the outcome	Selection of the reported result	Overall	
Liatis 2009	Oat β -glucan enriched bread	Wheat bread	+	+	+	+	+	+	Low risk
McGeoch 2013	Oat products	Standard dietary advice	+	+	+	+	+	+	Some concerns
Ballestros 2015	40g oatmeal	1 egg	+	+	+	+	+	+	High risk
Li 2016	50g whole grain oats	No dietary intervention	+	+	+	+	+	+	
Li 2016	100g whole grain oats	No dietary intervention	+	+	+	+	+	+	

Supplementary Table 8. Sensitivity analyses of the use of correlation coefficient of 0.25 and 0.75 for paired analysis in the analysis of the effect of oat β -glucan on HbA1c, fasting glucose, 2h-PG, fasting insulin and HOMA-IR

MD [95% CI], P, I ² , P _Q			
Outcome	Correlation coefficient used in primary analysis	Correlation coefficient used in the sensitivity analysis	
	0.5	0.25	0.75
HbA1c (%)	-0.47 [-0.80 to -0.13], P _{MD} =0.006, I ² =81.60%, P _Q <0.001	-0.49 [-0.84 to -0.14], P _{MD} = 0.006, I ² =80.88%, P _Q <0.001	-0.43 [-0.72 to -0.14], P _{MD} =0.004, I ² =83.88%, P _Q <0.001
Fasting glucose (mmol/L)	-0.75 [-1.20 to -0.31], P _{MD} <0.001, I ² =45.99%, P _Q =0.073	-0.81 [-1.26 to -0.35], P _{MD} <0.001, I ² =41.25%, P _Q =0.103	-0.66 [-1.08 to -0.24], P _{MD} =0.002, I ² =57.65%, P _Q =0.021
2h-PG (mmol/L)	-0.42 [-0.70 to -0.14], P _{MD} =0.003, I ² =94.68%, P _Q <0.001	-0.63 [-0.99 to -0.27], P _{MD} <0.001, I ² =94.20%, P _Q <0.001	-0.27 [-0.47 to -0.07], P _{MD} =0.008, I ² =94.99%, P _Q <0.001
Fasting insulin (pmol/L)	-4.30 [11.96 to 3.35], P _{MD} =0.271, I ² =64.45%, P _Q =0.038	-5.01 [-13.07 to 3.05], P _{MD} =0.222, I ² =55.57%, P _Q =0.080	-1.27 [-7.72 to 5.19], P _{MD} =0.703, I ² =80.50%, P _Q =0.002
HOMA-IR	-0.88 [-1.55 to -0.20], P _{MD} =0.011, I ² =56.42%, P _Q =0.057	-0.89 [-1.58 to -0.20], P _{MD} =0.012, I ² =56.41%, P _Q =0.057	-0.86 [-1.52 to -0.20], P _{MD} =0.011, I ² =56.43%, P _Q =0.057

Supplementary Table 9. GRADE assessment for the effect of oat β -glucan on HbA1c, fasting glucose, 2h-PG, fasting insulin and HOMA-IR

No. of Trial Comparisons	Design	Quality Assessment						No. of Participants	Pooled Mean Difference (95% CI)	Certainty
		Risk of Bias	Inconsistency	Indirectness	Imprecision	Publication Bias	Other			
HbA1c (%)										
8	RCT	not serious	serious ¹	not serious	serious ²	undetected ³	none	407	-0.47 (-0.80 to -0.13)	⊕⊕ Low
Fasting Glucose (mmol/L)										
8	RCT	not serious	not serious	not serious	serious ⁴	undetected ³	dose ⁵	407	-0.75 (-1.20 to -0.31)	⊕⊕⊕⊕ High
2h Postprandial Glucose (mmol/L)										
3	RCT	not serious	serious ⁶	not serious	serious ⁷	undetected ³	none	246	-0.42 (-0.70 to -0.14)	⊕⊕ Low
Fasting Insulin (pmol/L)										
4	RCT	not serious	not serious ⁸	not serious	serious ⁹	undetected ³	none	110	-4.30 (-11.96 to 3.35)	⊕⊕⊕ Moderate
HOMA-IR										
5	RCT	not serious	not serious ¹⁰	not serious	serious ¹¹	undetected ³	none	316	-0.88 (-1.55 to -0.20)	⊕⊕⊕ Moderate

All outcomes started with high certainty of evidence since all studies were randomized controlled trials and then downgraded or upgraded based on pre-specified criteria. Criteria for downgrades included risk of bias (downgraded if the majority of trials were considered to be at high risk of bias); inconsistency (downgraded if there was substantial unexplained heterogeneity [$I^2 \geq 50.00\%$, $P < 0.100$]; indirectness (downgraded if there were factors absent or present relating to the participants, interventions, or outcomes that limited the generalizability of the results); imprecision (downgraded if the 95% confidence interval crossed the minimally important difference [MID]); and publication bias. Criteria for upgrades included a significant dose-response gradient.

¹Downgraded for serious inconsistency, as $I^2 = 81.60\%$, $P_Q < 0.001$

²Downgraded for serious imprecision, as the 95% confidence interval (-0.80 to -0.13%) overlaps the MID for HbA1c which was set at 0.3%

³Publication bias was not assessed because ≤ 10 trial comparisons were available

⁴Downgraded for serious imprecision, as the 95% confidence interval (-1.20 to -0.31mmol/L) overlaps the MID for fasting glucose which was set at 0.5mmol/L

⁵Upgraded for significant linear dose response (slope=-0.39 [95% CI: -0.64 to -0.14], $P < 0.001$)

⁶Downgraded for serious inconsistency, as $I^2 = 94.68\%$, $P_Q < 0.001$. Although the evidence of substantial heterogeneity was explained by the removal of McGeoch et al. during the sensitivity analysis ($I^2 < 0.01\%$, $P_Q = 0.605$), there were insufficient trial comparisons to warrant not downgrading for inconsistency.

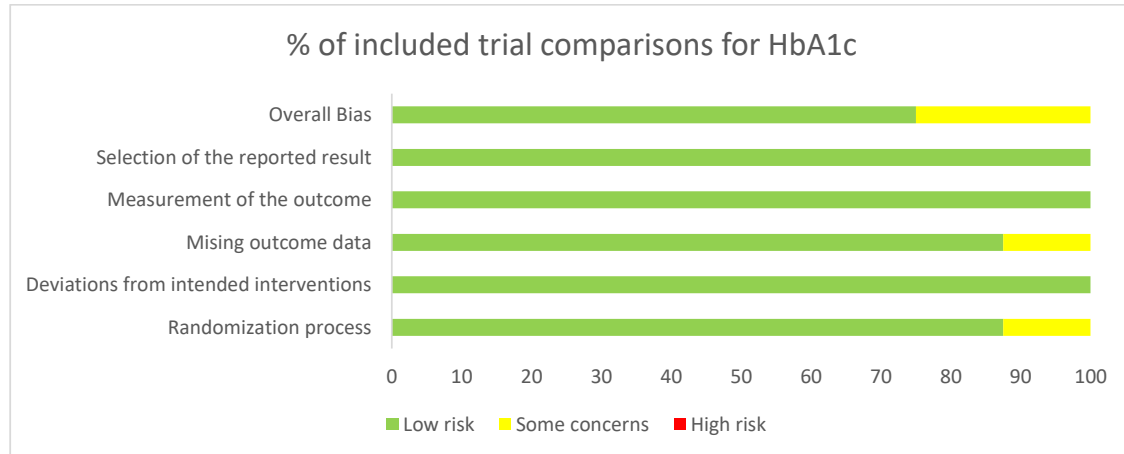
⁷Downgraded for serious imprecision, as the 95% confidence interval (-0.70 to -0.14mmol/L) overlaps the MID for 2h-PG which was set at 0.5mmol/L

⁸No downgrade for serious inconsistency as the presence of substantial heterogeneity ($I^2 = 64.45\%$, $P_Q = 0.038$) was explained by the removal of Liatis et al. ($I^2 = 39.42\%$, $P_Q = 0.192$) during sensitivity analysis.

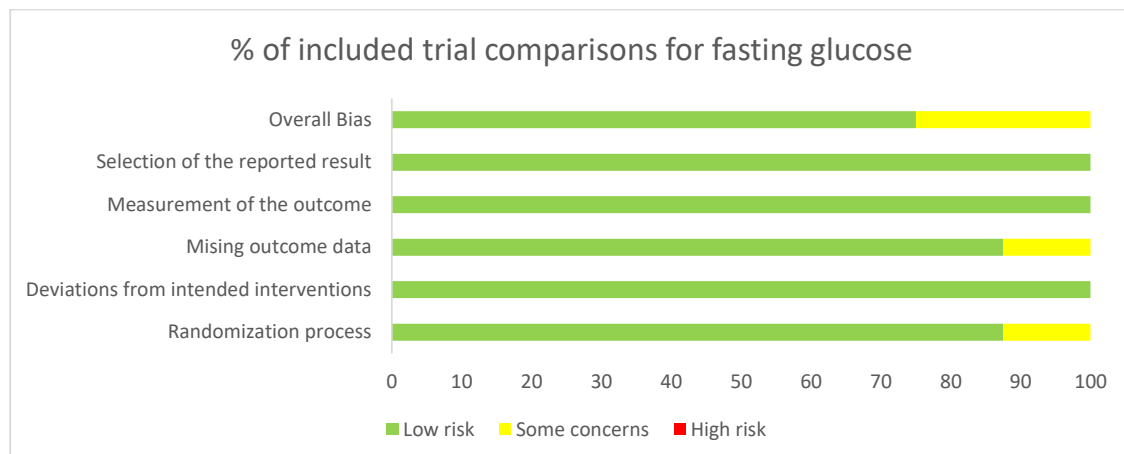
⁹Downgraded for serious imprecision, as the 95% confidence interval (-11.96 to 3.35pmol/L) overlaps the MID for fasting insulin which was set at 5pmol/L

¹⁰No downgrade for serious inconsistency as the presence of substantial heterogeneity ($I^2=56.42\%$, $P_Q=0.057$) was explained by the removal of Liatis et al. ($I^2=41.82\%$, $P_Q=0.161$) during sensitivity analysis.

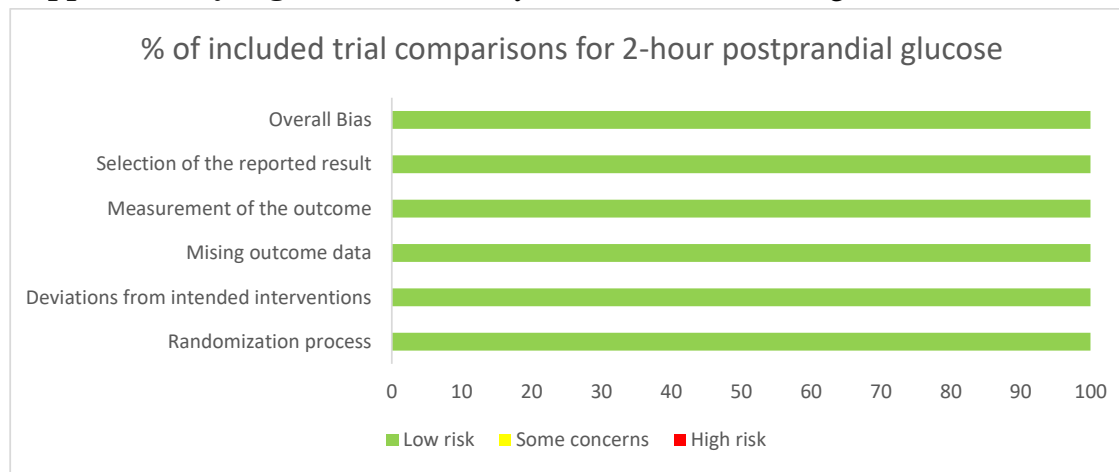
¹¹Downgraded for serious imprecision, as the 95% confidence interval (-1.55 to -0.20) overlaps the MID for HOMA-IR which was set at 1

Supplementary Fig. 1. RoB summary on the effect of oat β -glucan on HbA1c

Coloured bars represent the proportion of studies assessed as having a low risk of bias (green), some concerns (yellow) and a high risk of bias (red) for the 5 domains above according to Cochrane Risk of Bias 2.0 tool in the 8 included trial comparisons.

Supplementary Fig. 2. RoB summary on the effect of oat β -glucan on fasting glucose

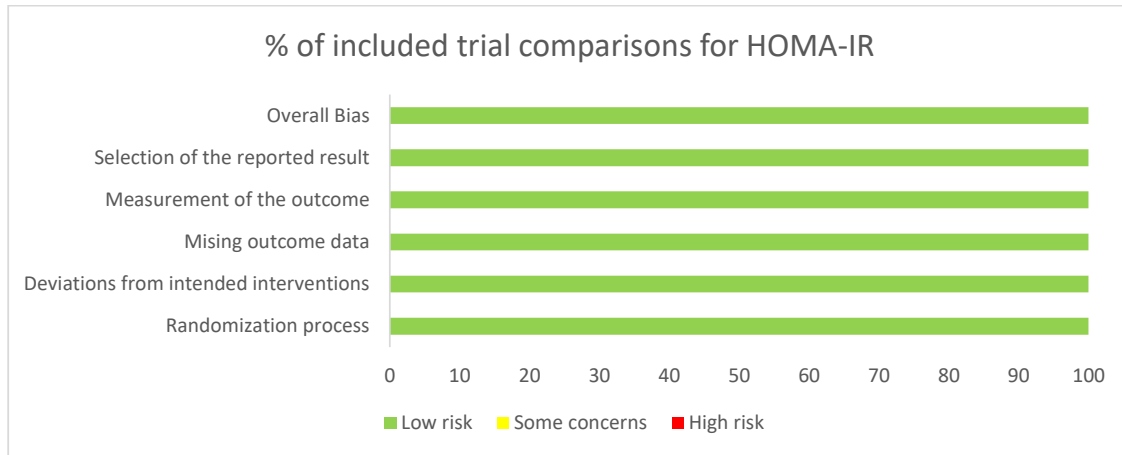
Coloured bars represent the proportion of studies assessed as having a low risk of bias (green), some concerns (yellow) and a high risk of bias (red) for the 5 domains above according to Cochrane Risk of Bias 2.0 tool in the 8 included trial comparisons.

Supplementary Fig. 3. RoB summary on the effect of oat β -glucan on 2h-PG

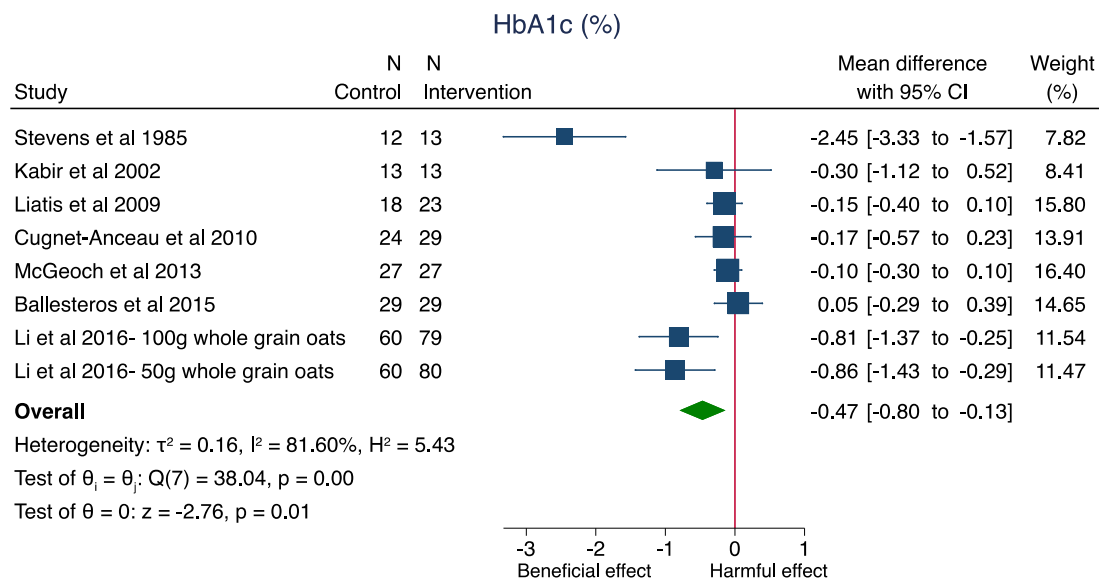
Coloured bars represent the proportion of studies assessed as having a low risk of bias (green), some concerns (yellow) and a high risk of bias (red) for the 5 domains above according to Cochrane Risk of Bias 2.0 tool in the 3 included trial comparisons.

Supplementary Fig. 4. RoB summary on the effect of oat β -glucan on fasting insulin

Coloured bars represent the proportion of studies assessed as having a low risk of bias (green), some concerns (yellow) and a high risk of bias (red) for the 5 domains above according to Cochrane Risk of Bias 2.0 tool in the 4 included trial comparisons.

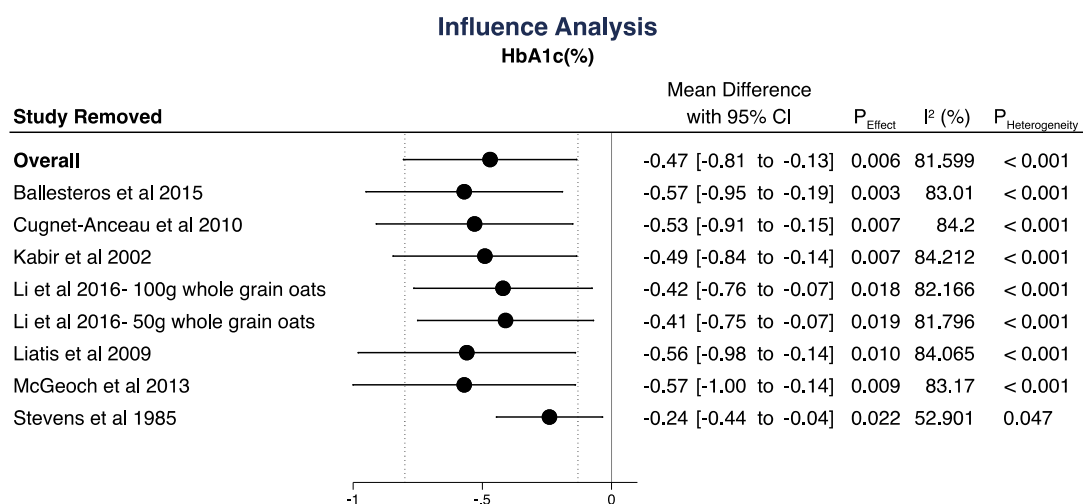
Supplementary Fig. 5. RoB summary on the effect of oat β -glucan on HOMA-IR

Coloured bars represent the proportion of studies assessed as having a low risk of bias (green), some concerns (yellow) and a high risk of bias (red) for the 5 domains above according to Cochrane Risk of Bias 2.0 tool in the 5 included trial comparisons.

Supplementary Fig. 6. Pooled effect estimates of oat β -glucan on HbA1c

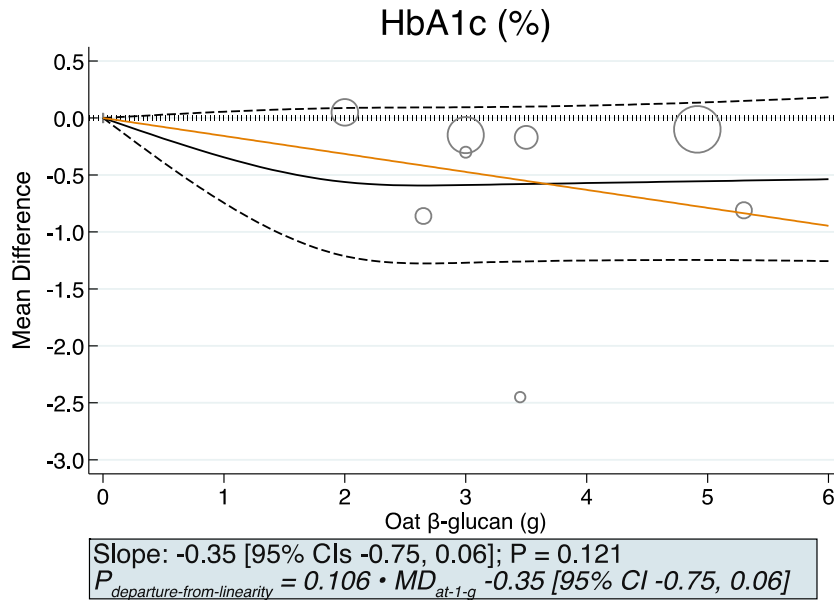
Random-effects DerSimonian-Laird model

The total pooled effect estimate is represented by the green diamond. Data are expressed as MDs with 95% CIs using the generic inverse variance method modelled by random effects (DerSimonian Laird). Heterogeneity was assessed using the Cochrane Q statistic and quantified using the I^2 statistic, where $p < 0.100$ and $I^2 \geq 50.00\%$ were used as evidence of significant substantial heterogeneity.

Supplementary Fig. 7. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β -glucan on HbA1c

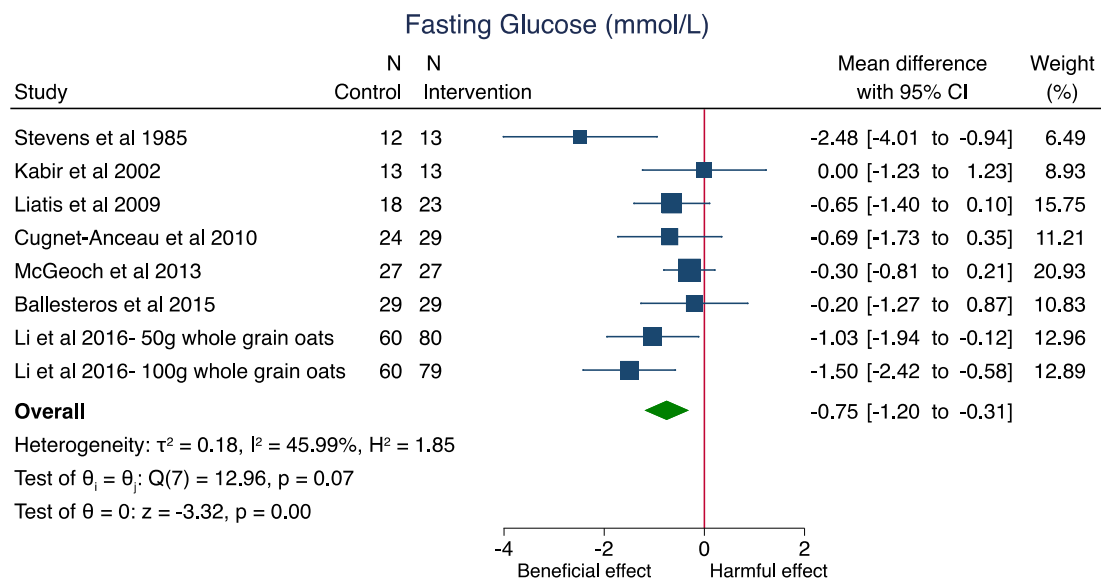
Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

Supplementary Fig. 8. Pooled linear and non-linear dose-response relationship between oat β -glucan and HbA1c



Individual trial comparisons are represented by the circles, with the weight of the comparison in the analysis represented by the size of the circle. The solid, orange line represents the linear dose response modelled by random effect with restricted maximum likelihood methods. The solid, black line and the dashed line represent the non-linear dose response and 95% CIs, respectively, which was modelled with restricted cubic splines with 3 knots.

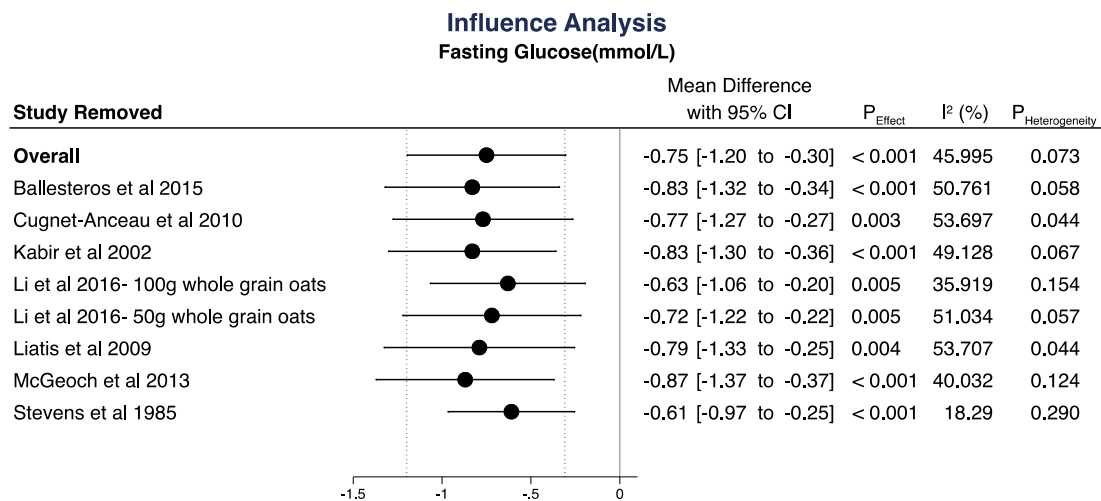
Supplementary Fig. 9. Pooled effect estimates of oat β -glucan on fasting glucose



Random-effects DerSimonian-Laird model

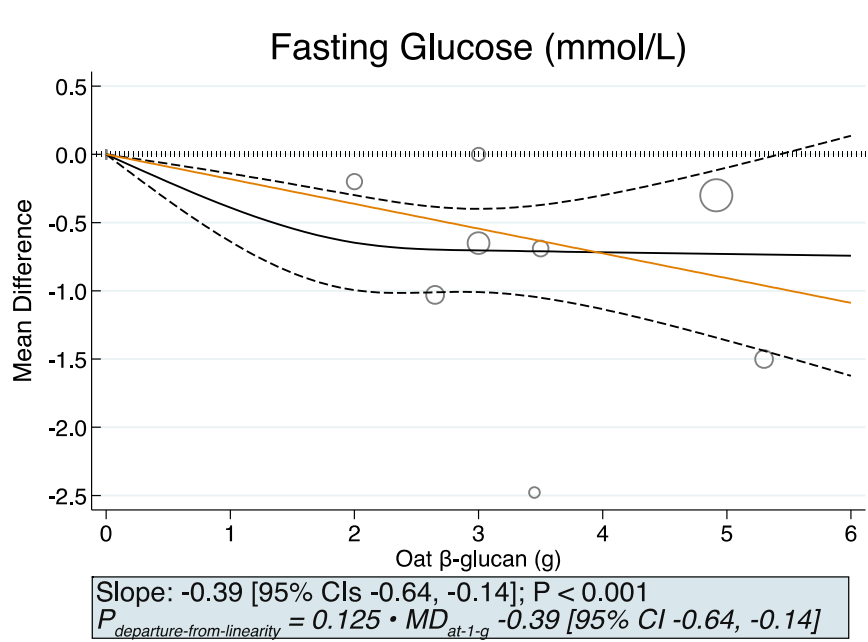
The total pooled effect estimate is represented by the green diamond. Data are expressed as MDs with 95% CIs using the generic inverse variance method modelled by random effects (DerSimonian Laird). Heterogeneity was assessed using the Cochrane Q statistic and quantified using the I^2 statistic, where $p < 0.100$ and $I^2 \geq 50.00\%$ were used as evidence of significant substantial heterogeneity.

Supplementary Fig. 10. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β -glucan on fasting glucose

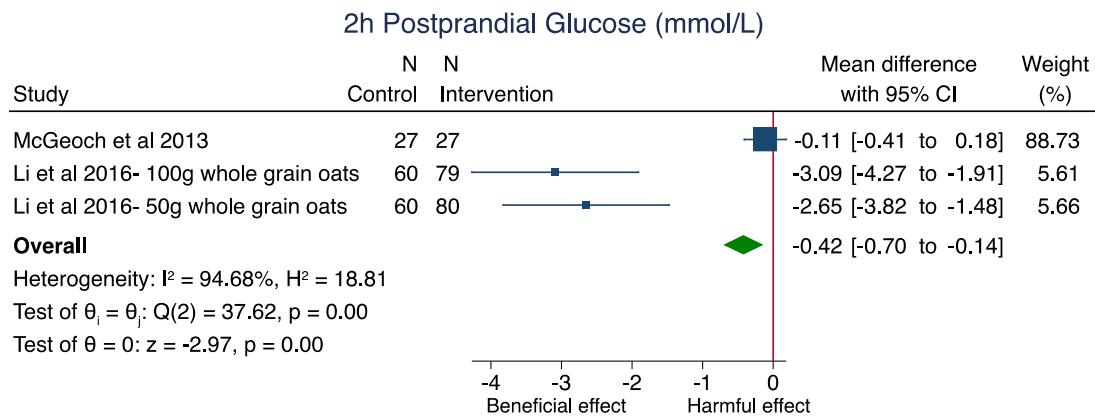


Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

Supplementary Fig. 11. Pooled linear and non-linear dose-response relationship between oat β -glucan and fasting glucose



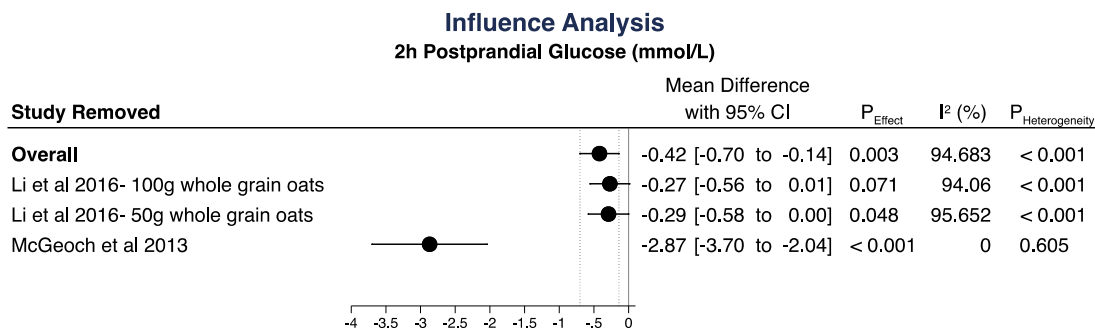
Individual trial comparisons are represented by the circles, with the weight of the comparison in the analysis represented by the size of the circle. The solid, orange line represents the linear dose response modelled by random effect with restricted maximum likelihood methods. The solid, black line and the dashed line represent the non-linear dose response and 95% CIs, respectively, which was modelled with restricted cubic splines with 3 knots.

Supplementary Fig. 12. Pooled effect estimates of oat β -glucan on 2h-PG

Fixed-effects inverse-variance model

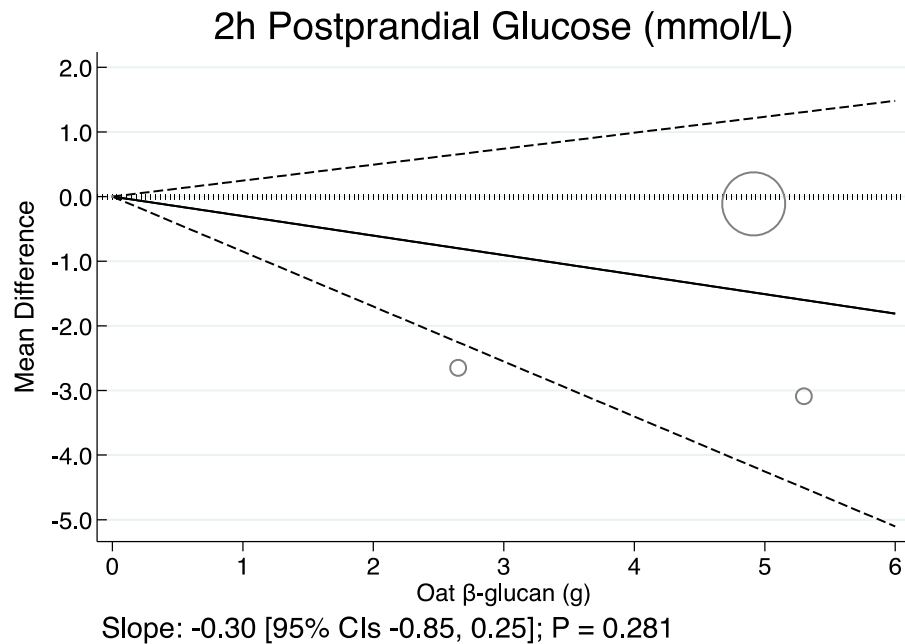
The total pooled effect estimate is represented by the green diamond. Data are expressed as MDs with 95% CIs using the generic inverse variance method modelled by fixed effects.

Heterogeneity was assessed using the Cochrane Q statistic and quantified using the I^2 statistic, where $p < 0.100$ and $I^2 \geq 50.00\%$ were used as evidence of significant substantial heterogeneity.

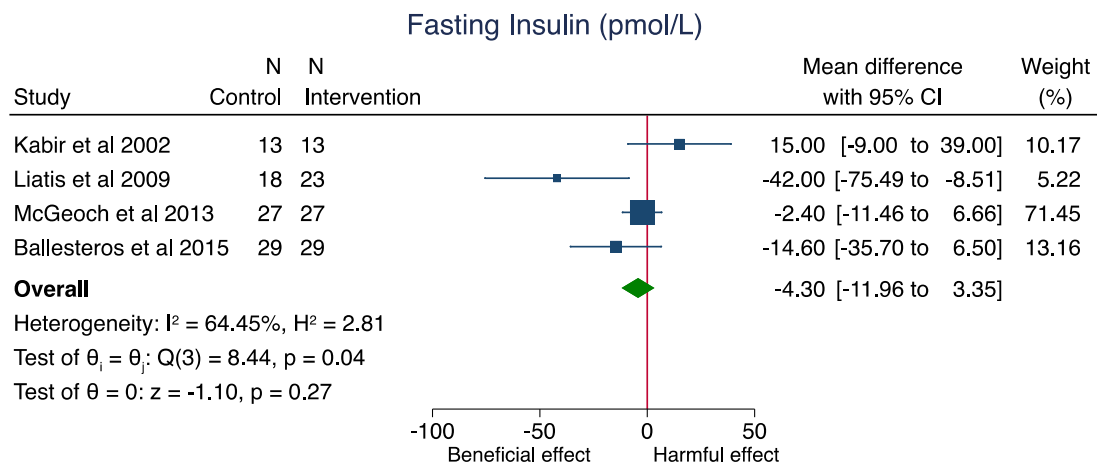
Supplementary Fig. 13. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β -glucan on 2h-PG

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

Supplementary Fig. 14. Pooled linear dose-response relationship between oat β -glucan and 2h-PG



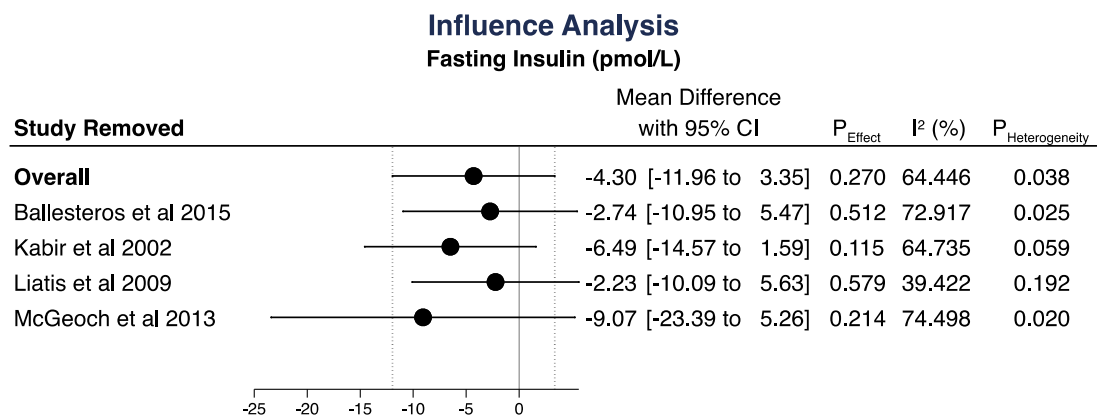
Individual trial comparisons are represented by the circles, with the weight of the comparison in the analysis represented by the size of the circle. The solid line represents the linear dose response modelled by random effect with restricted maximum likelihood methods. The dashed lines represent the 95% CIs.

Supplementary Fig. 15. Pooled effect estimates of oat β -glucan on fasting insulin

Fixed-effects inverse-variance model

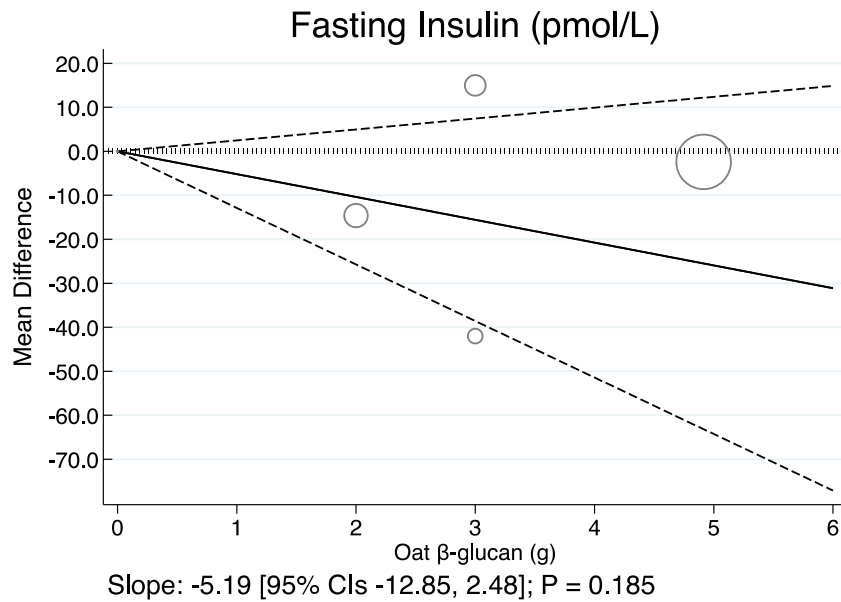
The total pooled effect estimate is represented by the green diamond. Data are expressed as MDs with 95% CIs using the generic inverse variance method modelled by fixed effects.

Heterogeneity was assessed using the Cochrane Q statistic and quantified using the I^2 statistic, where $p < 0.100$ and $I^2 \geq 50.00\%$ were used as evidence of significant substantial heterogeneity.

Supplementary Fig. 16. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β -glucan on fasting insulin

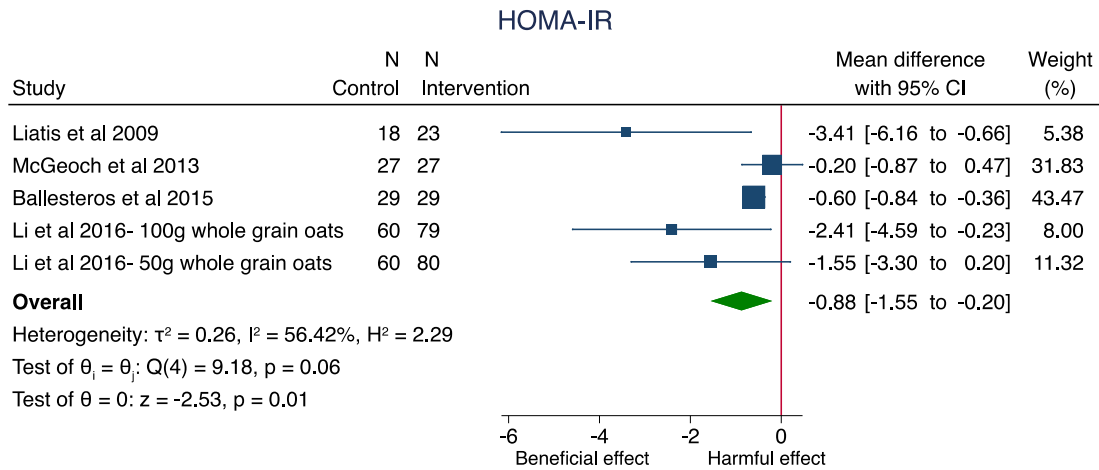
Influence analysis: Removal of each study, one at a time and recalculation of the overall effect and heterogeneity

Supplementary Fig. 17. Pooled linear dose-response relationship between oat β -glucan and fasting insulin



Individual trial comparisons are represented by the circles, with the weight of the comparison in the analysis represented by the size of the circle. The solid line represents the linear dose response modelled by random effect with restricted maximum likelihood methods. The dashed line represents the 95% CIs.

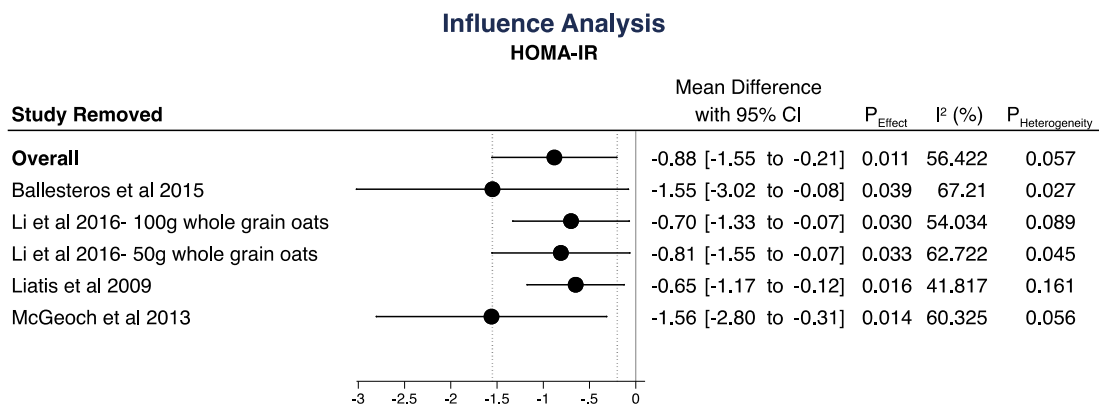
Supplementary Fig. 18. Pooled effect estimates of oat β -glucan on HOMA-IR



Random-effects DerSimonian-Laird model

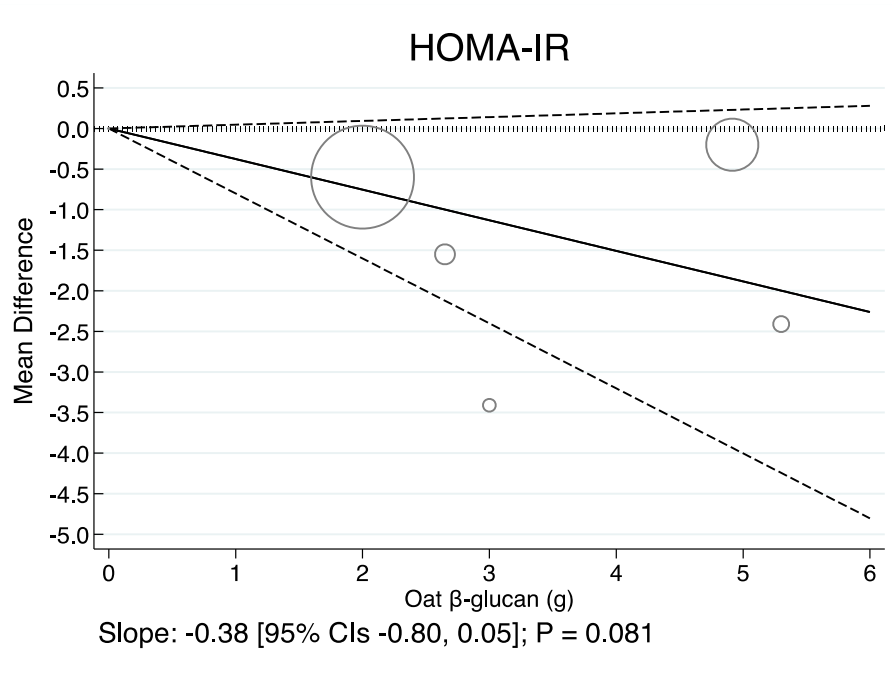
The total pooled effect estimate is represented by the green diamond. Data are expressed as MDs with 95% CIs using the generic inverse variance method modelled by random effects (DerSimonian Laird). Heterogeneity was assessed using the Cochrane Q statistic and quantified using the I^2 statistic, where $p < 0.100$ and $I^2 \geq 50.00\%$ were used as evidence of significant substantial heterogeneity.

Supplementary Fig. 19. Sensitivity analysis of the systematic removal of individual trials for the effect of oat β -glucan on HOMA-IR



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

Supplementary Fig. 20. Pooled linear dose-response relationship between oat β -glucan and HOMA-IR



Individual trial comparisons are represented by the circles, with the weight of the comparison in the analysis represented by the size of the circle. The solid line represents the linear dose response modelled by random effect with restricted maximum likelihood methods. The dashed line represents the 95% CIs.