Include oats, barley and soluble fibre in your diet: an achievable goal to improve cardiometabolic health

Sally D. Poppitt[1,2,3]^, Jennifer L. Miles-Chan[1,2,4]^

[1]Human Nutrition Unit, School of Biological Sciences, University of Auckland, Auckland, New Zealand; [2]High Value Nutrition, National Science Challenge, Auckland, New Zealand; [3]Department of Medicine, University of Auckland, Auckland, New Zealand; [4]Riddet Institute, Palmerston North, New Zealand

Correspondence to: Professor Sally D. Poppitt, PhD. Human Nutrition Unit, School of Biological Sciences, University of Auckland, Waipapa Taumata Rau, Private Bag 92019, Auckland 1024, New Zealand; High Value Nutrition, National Science Challenge, Auckland, New Zealand; Department of Medicine, University of Auckland, Auckland, New Zealand. Email: s.poppitt@auckland.ac.nz.

Comment on: Reiners S, Hebestreit S, Wedekind L, *et al*. Effect of a regular consumption of traditional and roasted oat and barley flakes on blood lipids and glucose metabolism-A randomized crossover trial. Front Nutr 2023;10:1095245.

Keywords: Oats; barley; high-temperature-processing; lipids; glucose

Submitted Jul 20, 2023. Accepted for publication Aug 09, 2023. Published online Sep 04, 2023.
doi: 10.21037/atm-23-1780
View this article at: https://dx.doi.org/10.21037/atm-23-1780

Dietary fibre (DF) has long been identified as an important contributor to cardiometabolic protection. Numerous randomised controlled trials (RCTs), reporting improvements in cardiovascular (CV) risk markers including bodyweight, lipids, and glycaemic endpoints were underpinned by large observational studies, such as the US Nurses' Health Study and the Health Professional's Follow-Up Study (1,2), with protective associations between whole grains and heart disease outcomes. These early studies have now been strengthened by a wealth of evidence, summarised in a 2019 meta-analysis comprising 185 observational studies and 58 RCTs (3), which confirmed these positive outcomes. Observational data showed 15–30% decrease in CV-related mortality, incidence of coronary heart disease and stroke, and type 2 diabetes (T2D) in high *vs.* low consumers of DF. The clinical trials showed significantly lower bodyweight, blood pressure and total cholesterol (TChol). The authors reported greatest risk reduction when DF intake was between 25–29 g/day, but notably proposed that higher intakes may confer even greater benefits for CV disease (CVD) and T2D prevention.

These cardiometabolic effects are attributed at least in part to lipid- and glucose-lowering properties of soluble fibre, present in high amounts in cereals such as oats and barley (4). In grains such as wheat and rice the majority component of DF is insoluble. Other plant-based components also high in viscous soluble fibre include legumes such as beans, peas and lentils, fruits including apples, pears, nectarines, apricots and various berries, as well as dietary supplements such as psyllium and inulin fibres. It is soluble fibre β-glucan, a glucose polymer comprising a β (1→3)-linked-D-glucopyranosyl backbone with β (1→6) linked side chains, that is the major contributor to commonly reported lipid and glucose-lowering properties. β-glucan occurs naturally in the grain husk or 'bran' of plants in the grass family, with high concentrations in oats (~5%) and barley (~7%).

Importantly, however, common food manufacturing processes can significantly alter the properties of β-glucans in both oats (5) and barley (6), likely in turn to affect cardiometabolic efficacy. Our laboratory previously reported a further level of difficulty in predicting cholesterol-lowering effects of food products industrially enriched with extracts of oat and barley cereals (7,8). Solubility, viscosity and molecular weight of β-glucans may all have important roles to play in this variability (5), with varied methods

^ ORCID: Sally D. Poppitt, 0000-0002-2214-8378; Jennifer L. Miles-Chan, 0000-0003-0313-7048.

used for extraction of soluble β-glucan from oat and barley fractions (9). Depolymerisation of the linear structure plus other structural alterations during industrial purification can decrease both molecular weight and viscosity of a β-glucan extract (10). Also, extraction conditions may not always deactivate endogenous β-glucanase enzymes present, which in turn can further increase depolymerisation. In addition, processes associated with application of heat during cooking of β-glucan-containing foods and modified-products can also alter molecular weight. Further processing, including freezing at –18 ℃ as well as long-term product storage, may alter *in vivo* digestibility with evidence of decreased uptake of β-glucan from the gastrointestinal tract following these common commercial processes (11).

Recently, in *Frontiers in Nutrition*, Reiners and colleagues (12) reported the effects on blood lipids and glucose metabolism of daily consumption of oat and barley flakes, in a 5 treatment RCT. In addition to quantifying cardioprotective effects of these 2 cereals, they aimed to determine whether a commercial high temperature roasting process significantly altered efficacy. Using a cross-over design the researchers manipulated a daily breakfast meal for intervals of 3 weeks. Thirty-two participants (women, 68%) with moderately raised low-density lipoprotein (LDL)-cholesterol (LDL-C ≥2.5 mmol/L), but otherwise self-reported healthy, were recruited from the general population of Thuringia state, central Germany.

Participants consumed 80 g of traditional 'raw' or roasted oat flakes [8.8 g total DF, β-glucan 4% (3.2 g) w/w; roasted at 150 ℃ for 20 min], and traditional or roasted barley flakes [10 g total DF, special cultivar, low amylose, high β-glucan 5% (4.1 g) w/w; roasted at 160 ℃ for 20 min] in 4 of the study arms. Both cereal diets achieved European Food Safety Authority (EFSA) recommended dose of 3 g β-glucans for hypercholesterolaemic individuals. The control arm comprised 100 g of white toast bread, matched with treatments for total energy (1.1–1.2 MJ) and total carbohydrate (CHO; 45–50 g), but containing low level of total DF (2.8 g). Notably, participants were not restricted to treatment and control products only, but were allowed to supplement breakfast with 'side' dishes, with total intake at the meal estimated from self-reported food records. This resulted in significantly higher total energy, CHO and fat intake on the control arm. Fasted and postprandial risk markers were assessed at week 0/baseline and 3 week/end of intervention for each dietary arm. To evaluate 3-h postprandial response the breakfast test meal comprising the treatment or control product was consumed with 200 mL whole milk (3.5% total fat), over 20 min. The postprandial meal was fully diet controlled.

The study protocol was well adhered to, 32 participants completing each of 5 study arms. No dropouts and 100% compliance to dietary treatment were reported. Based on self-reported diet records, the authors noted that addition of cereal flakes at breakfast significantly increased the intake of DF by 3.6 g/day to a total of 6.5 g/day compared to pre-intervention baseline.

To report the intervention outcomes, the authors used a mixed model with random intercept approach. Difference between baseline and end of intervention was calculated and the delta value used to assess between-treatment main effect. When significant, *post hoc* within-treatment pairwise comparisons over the 3-week intervention, i.e., pre- *vs.* post-treatment, were then also reported. Importantly, *post hoc* between-treatment comparisons with the low fibre control diet were also conducted, not reported within text but provided in *Tab. 6* of their article. No time dependent trajectory was investigated between treatments, hence no interaction terms (diet*time) were reported.

Significant protective effects of cereal consumption were observed for TChol and LDL-C only. Unexpectedly, there were no significant between-treatment effects of high cereal intake on fasting or postprandial glycaemic endpoints, or the inflammatory acute-phase-protein C-reactive protein (CRP). To summarise, relative to low-fibre control there was a significant decrease in TChol and LDL-C for all 4 traditional 'raw' and heat processed roasted oats and barley diets. Over 3 weeks the cereal diets decreased fasting levels of each by an average of 4.8% (0.27–0.33 mmol/L) and 6.5% (0.21–0.30 mmol/L) relative to baseline, respectively. This may be considered as approaching a clinically significant decrease over 3 weeks, with evidence from prior trials that the trajectory of cholesterol-lowering may be predicted to continue if the duration of the diet was extended. Clearly in order to achieve maximal risk reduction, based for example on the meta-analysis from Reynolds and colleagues (3), a similar weight of fibre to that in the breakfast meal must also be consumed at lunch and dinner, in order to achieve a daily intake of between 25 to 29 g. Notably this would be a difficult target to achieve based on a typical Western diet. Data from pharmaceutical trials also inform these predictions, where for every 1.0 mmol/L LDL-C-lowering achieved through drug treatments such as statins [3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors] there is a predicted relative risk reduction (RRR) >20% [relative risk

(RR) =0.79; 95% confidence interval (CI): 0.77–0.81] even in low risk individuals (13). Achieving such a big decrease in LDL-C through dietary means however remains a considerable challenge.

Since there is growing evidence that CVD risk is strongly determined by cumulative lifetime exposure to LDL-C, even in those for whom CVD risk is low, 'lower is better, for longer' (14). Small decreases of LDL-C sustained over a long duration are predicted to generate substantial CVD benefits. Hence diet-driven decreases in TChol, LDL-C or non-high-density lipoprotein-cholesterol (non-HDL-C) are worthy aims. Data from the recent Reynolds low *vs.* high fibre meta-analysis of 36 RCTs reported TChol mean difference (MD) to be –0.15 mmol/L (3). Two recent meta-analyses of (I) 13 oat β-glucan interventions reported TChol standardised MD (SMD) of –0.24 mmol/L and LDL-C SMD of –0.27 mmol/L (15), and (II) 59 oat supplementation interventions (oats, oat-derived β-glucan and/or oat-derived phytochemical avenanthramides) reported TChol SMD of –0.42 mmol/L and LDL-C SMD of –0.29 mmol/L (16). Reiners and colleagues (12) do not state whether participants returned to healthy or 'ideal' TChol levels and/or LDL-C levels of 3.0 mmol/L which represents the target for individuals at low risk of CVD in many countries (17). Making the assumption that the LDL-C data were normally distributed and hence median values can be interpreted as (or close to) group mean, LDL-C concentrations were 3.51, 3.59, 3.59 and 3.52 mmol/L following the 3-week intervention with each of the 4 cereal diets.

Importantly, with respect to the primary aims of the trial, the authors found no difference in lipid outcomes between traditional or roasted flakes over the 3-week intervention, suggesting that commercial roasting for 20 minutes did not adversely affect lipid-lowering properties. Unexpectedly however, fasted HDL-C decreased by ~5% on the 4 cereal treatments, an adverse diet-induced change which was significantly different to the low fibre control for all but traditional oat flakes. The clinically informative TChol:HDL-C ratio (18) was not reported, but the adverse change in HDL-C may have at least in part ameliorated the protective effects of TChol-lowering. Nor were the informative 'non-HDL' (19) lipid components comprising very low-density lipoprotein (VLDL) or lipoprotein(a) measured. Non-HDL-C has recently replaced LDL-C as the primary target for CV risk reduction in a number of jurisdictions, including the UK (20). None of the oat or barley diets altered fasting triglyceride or high sensitivity CRP (hsCRP) over 3 weeks, nor any of fasting blood glucose, insulin, HbA_{1c} or homeostasis model assessment of insulin resistance (HOMA-IR).

A body of evidence from high fibre RCTs supports improvement in glycaemia, notably including fasting glucose, insulin, HbA_{1c} and HOMA-IR, in populations with dysglycaemia such as T2D (3). Reiners comments on earlier meta-analyses where oat β-glucan was more efficacious in T2D (12), proposing that the absence of glycaemic improvement in fasted endpoints may be due to their self-reported 'healthy' cohort (12). However, interrogation of median and interquartile range (IQR) fasting glucose levels (5.60, 0.85 mmol/L) shows ~50% to have moderate dysglycaemia within the prediabetic range based on American Diabetes Association (ADA) impaired fasting glucose criteria (IFG; 5.6–6.9 mmol/L). Alternately, since the primary mechanism attributed to improved glycaemia is the viscosity-promoting effect of β-glucans within the gastrointestinal tract, this is expected to primarily alter post-meal glycaemic response rather than fasted endpoints.

Despite known viscosity effects, Reiners found no detectable postprandial glycaemic response to either high fibre 'raw' cereal *vs.* the low-fibre control. Conversely, area under the curve (AUC) measured over 3 hours post-breakfast, for TChol and LDL-C was significantly decreased for all 4 high fibre diets relative to the low-fibre control, decreasing between 3.4–6.7% and 3.4–9.6% from baseline to 3 weeks for the 4 treatments arms. Lack of effect of cereal intake on postprandial glycaemic endpoints was unexpected, based on prior meta-analysis evidence (21) including in healthy normoglycaemic cohorts (22). Reiners *et al.*, did report a *post hoc* within-treatment decrease over time in postprandial AUC glucose following the traditional 'raw' oats treatment, proposing an unfavourable effect of roasting on glucose metabolism since this protective decrease was absent with processed flakes. The authors note (12) that viscosity significantly decreased with roasting, reported in prior *in vitro* assessments by their collaborator Schlormann and colleagues. More importantly, however, no effect was observed in any cereal treatment *vs.* the low-fibre control.

Four g β-glucan per 30 g available carbohydrate (CHO) (avCHO) is the EFSA approved dose for oats or barley to obtain a significant decrease in postprandial glucose (23). This was likely achieved by Reiners *et al.*, based on the 3.2 g β-glucan/44.8 g total CHO (estimated as 3.2 g/35 g avCHO) in oat flakes and 4.1 g β-glucan/50.6 g total CHO (estimated as 4.1 g/40 g avCHO) for barley flakes. A recent

Table 1 Strategic action points—protective role of dietary change

Areas of focus	Achievable actions for cardiometabolic health
Lifestyle, diet	Even small changes made to the diet, such as regular inclusion of whole-grain high-fibre cereals, are important. Aim to maintain changes long-term as (I) prevention for those at risk and (II) treatment (in combination with pharmaceutical strategies) for established CV disease
Intermediary risk markers	Even small diet-driven changes made to lipid and glycaemic endpoints are important to achieve, since prolonged lowering of LDL-C (non-HDL-C) and glucose-related biomarkers can result in significant CV benefits
Early intervention	Intervene with diet for CV risk protection at an early age, consider young adults since even mild biomarker elevation can result in poor disease outcomes long term
Low/moderate CV risk	Intervene with diet at early (low/moderate) CV risk stages where significant benefits can be achieved

CV, cardiovascular; LDL-C, low-density lipoprotein-cholesterol; HDL-C, high-density lipoprotein-cholesterol.

systematic review from the Wolever lab (24), comprising 57 trials in >300 healthy participants, investigating minimum dose of oat β-glucan required to decrease postprandial glycaemic response showed molecular weight to be a key determinant. Minimum dose for high (>1,000 kg/mol), medium (300–1,000 kg/mol) and low (<300 kg/mol) molecular weight products increased from 0.2, 2.2 up to 3.2 g/30 g avCHO respectively. In their current study molecular weight data was not provided by Reiners and colleagues for oat or barley products, which was an oversight.

In summary, the Reiners intervention has contributed to a body of RCTs showing moderate-dose high-β-glucans cereals, even when consumed over a short 3-week duration, can improve cardiometabolic lipid markers even in low-risk individuals. Whilst there was no evidence that roasting at 150–160 ℃ dry heat ameliorated the positive effects, prior studies clearly show loss of efficacy under varied processing conditions and hence it remains important that novel methods and products are carefully assessed. Perhaps unexpectedly, Reiners and colleagues did not observe positive effects of traditional 'raw' oats or barley on postprandial glycaemic endpoints, despite approximately half of the cohort having a level of IFG characteristic of pre-diabetes. Other trials however have consistently shown improved postprandial glycaemic profile.

Clearly, a healthy diet maintained throughout a lifetime is critical for all individuals, yet there is particular relevance for those with increased risk of cardiometabolic disease and where cereals such as oats and barley can make a significant contribution. Dietary intervention, alongside bodyweight loss, has long been first line treatment for metabolic conditions such as T2D, and in turn diet must be considered as first line prevention for those at risk of later disease. Strategic points that should be acted upon in the public health system and where the protective role of diet is key (see *Table 1*), include: (I) make small changes in the diet, and aim to sustain them long-term—even small dietary changes are important if maintained over a prolonged timeframe. Both as prevention for those at risk and, in combination with pharmaceutical strategies, as treatment for those with established disease and elevated lifetime risk; (II) make small changes in lipid (and glucose) profiles, and aim to sustain them long-term—even ~5% lowering achieved by Reiners *et al.*, is important. Small decreases in LDL-C if maintained over a long duration can result in important CVD benefits; (III) intervene with CV risk at an early age—consider young adults. A recent meta-analysis of RCTs (25) showed even mild LDL-C elevation in younger adults increased CVD risk and the likelihood of worse disease outcomes *vs.* similar elevations in older adults; (IV) intervene with cohorts at low or moderate CV risk—do not ignore these groups until risk elevation. In their RCT, Reiners and colleagues showed significant benefits of dietary intervention in a low risk cohort. 'Lower is better for longer' for intermediary CV markers such as LDL-C (14).

Overall, prioritise younger adults alongside their older peers even when at low risk, to achieve and maintain lifestyle (dietary) changes such as regular incorporation of whole grain cereals, to promote small improvements in lipid and glycaemic profile. It is likely that even these small changes will drive clinically significant benefits from a lower life-long exposure to cardiometabolic risk.

Acknowledgments

Funding: This work was supported by funding from the New Zealand National Science Challenge-High Value Nutrition (NSC-HVN) Programme (MBIE grant No.

3710040, to S.D.P., J.L.M.C.) and the New Zealand Health Research Council (HRC grant No. 17/009, to J.L.M.C.).

Footnote

Provenance and Peer Review: This article was commissioned by the editorial office, *Annals of Translational Medicine*. The article did not undergo external peer review.

Conflicts of Interest: Both authors have completed the ICMJE uniform disclosure form (available at https://atm.amegroups.com/article/view/10.21037/atm-23-1780/coif). S.D.P. reports funding from the New Zealand National Science Challenge-High Value Nutrition (NSC-HVN) Programme (MBIE grant No. 3710040). J.L.M.C. reports funding from the New Zealand National Science Challenge-High Value Nutrition (NSC-HVN) Programme (MBIE grant No. 3710040), and a Health Research Council New Zealand Fellowship (HRC grant No. 17/009). She also serves as Associate Editor of the *European Journal of Clinical Nutrition*. The authors have no other conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

References

1. Jensen MK, Koh-Banerjee P, Hu FB, et al. Intakes of whole grains, bran, and germ and the risk of coronary heart disease in men. Am J Clin Nutr 2004;80:1492-9.
2. Liu S, Stampfer MJ, Hu FB, et al. Whole-grain consumption and risk of coronary heart disease: results from the Nurses' Health Study. Am J Clin Nutr 1999;70:412-9.
3. Reynolds A, Mann J, Cummings J, et al. Carbohydrate quality and human health: a series of systematic reviews and meta-analyses. Lancet 2019;393:434-45.
4. Brown L, Rosner B, Willett WW, et al. Cholesterol-lowering effects of dietary fiber: a meta-analysis. Am J Clin Nutr 1999;69:30-42.
5. Makela N, Brinck O, Sontag-Strom T. Viscosity of β-glucan from oat products at the intestinal phase of the gastrointestinal model. Food Hydrocolloids 2020;100:105422.
6. Lukinac J, Jukić M. Barley in the Production of Cereal-Based Products. Plants (Basel) 2022;11:3519.
7. Poppitt SD. Soluble fibre oat and barley beta-glucan enriched products: can we predict cholesterol-lowering effects? Br J Nutr 2007;97:1049-50.
8. Keogh GF, Cooper GJ, Mulvey TB, et al. Randomized controlled crossover study of the effect of a highly beta-glucan-enriched barley on cardiovascular disease risk factors in mildly hypercholesterolemic men. Am J Clin Nutr 2003;78:711-8.
9. Maheshwari G, Sowrirajan S, Joseph B. Extraction and Isolation of β-Glucan from Grain Sources-A Review. J Food Sci 2017;82:1535-45.
10. Würsch P, Pi-Sunyer FX. The role of viscous soluble fiber in the metabolic control of diabetes. A review with special emphasis on cereals rich in beta-glucan. Diabetes Care 1997;20:1774-80.
11. Beer MU, Wood PJ, Weisz J, et al. Effect of Cooking and Storage on the Amount and Molecular Weight of (1→3)(1→4)-β-d-Glucan Extracted from Oat Products by an In Vitro Digestion System. Cereal Chemistry 1997;74:705-9.
12. Reiners S, Hebestreit S, Wedekind L, et al. Effect of a regular consumption of traditional and roasted oat and barley flakes on blood lipids and glucose metabolism-A randomized crossover trial. Front Nutr 2023;10:1095245.
13. Cholesterol Treatment Trialists' (CTT) Collaborators; Mihaylova B, Emberson J, et al. The effects of lowering LDL cholesterol with statin therapy in people at low risk of vascular disease: meta-analysis of individual data from 27 randomised trials. Lancet 2012;380:581-90.
14. Penson PE, Pirro M, Banach M. LDL-C: lower is better for longer-even at low risk. BMC Med 2020;18:320.
15. Yu J, Xia J, Yang C, et al. Effects of Oat Beta-Glucan Intake on Lipid Profiles in Hypercholesterolemic Adults: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. Nutrients 2022;14:2043.
16. Llanaj E, Dejanovic GM, Valido E, et al. Effect of oat supplementation interventions on cardiovascular disease risk markers: a systematic review and meta-analysis of

randomized controlled trials. Eur J Nutr 2022;61:1749-78.
17. Mach F, Baigent C, Catapano AL, et al. 2019 ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk. Eur Heart J 2020;41:111-88.
18. National Institute for Health and Care Excellence: NICE. Cardiovascular disease: risk assessment and reduction, including lipid modification. 2023. Available online: https://www.nice.org.uk/guidance/cg181
19. Grundy SM, Stone NJ, Bailey AL, et al. 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation 2019;139:e1046-81. Erratum in: Circulation 2019;139:e1178-81.
20. National Institute for Health and Care Excellence: NICE. Lipid modification - CVD prevention. 2023. Available online: https://www.cks.nice.org.uk/topics/lipid-modification-cvd-prevention
21. Musa-Veloso K, Noori D, Venditti C, et al. A Systematic Review and Meta-Analysis of Randomized Controlled Trials on the Effects of Oats and Oat Processing on Postprandial Blood Glucose and Insulin Responses. J Nutr 2021;151:341-51.
22. Wolever TMS, Johnson J, Jenkins AL, et al. Impact of oat processing on glycaemic and insulinaemic responses in healthy humans: a randomised clinical trial. Br J Nutr 2019;121:1264-70.
23. EFSA Panel on Dietetic Products, Nutrition and Allergies (NDA). Scientific opinion on the substantiation of health claims related to betaglucans from oats and barley and maintenance of normal blood LDL-cholesterol concentrations (ID 1236, 1299), increase in satiety leading to a reduction in energy intake (ID 851, 852), reduction of post-prandial glycaemic responses (ID 821, 824), and "digestive function" (ID 850) pursuant to article 13 of regulation (EC) No 1924/2006. EFSA J 2011;9:2207.
24. Noronha JC, Zurbau A, Wolever TMS. The importance of molecular weight in determining the minimum dose of oat β-glucan required to reduce the glycaemic response in healthy subjects without diabetes: a systematic review and meta-regression analysis. Eur J Clin Nutr 2023;77:308-15.
25. Wang N, Fulcher J, Abeysuriya N, et al. Intensive LDL cholesterol-lowering treatment beyond current recommendations for the prevention of major vascular events: a systematic review and meta-analysis of randomised trials including 327 037 participants. Lancet Diabetes Endocrinol 2020;8:36-49.

Cite this article as: Poppitt SD, Miles-Chan JL. Include oats, barley and soluble fibre in your diet: an achievable goal to improve cardiometabolic health. Ann Transl Med 2024;12(1):17. doi: 10.21037/atm-23-1780