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

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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

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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.

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