

Original Article

Supplementation of a high-carbohydrate breakfast with barley β -glucan improves postprandial glycaemic response for meals but not beverages

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There is growing support for the protective role of soluble fibre in type II diabetes. Soluble fibre β -glucan found in cereal products including oats and barley may be the active component. There is evidence of postprandial blunting of blood glucose and insulin responses to dietary carbohydrates when oat soluble fibre is supplemented into the diet but few trials have been carried out using natural barley or enriched barley β -glucan products. The aim of this trial was to investigate the postprandial effect of a highly enriched barley β -glucan product on blood glucose, insulin and lipids when given with a high-carbohydrate (CHO) food and a high-CHO drink. 18 lean, healthy men completed a 4 treatment intervention trial comprising (i) high-CHO_{food control}, (ii) high-CHO_{food+fibre}, (iii) high-CHO_{drink control}, (iv) high-CHO_{drink+fibre} where a 10g dose of barley β -glucan fibre supplement (Cerogen) containing 6.31g β -glucan was added to food and drink controls. There was an increase of glucose and insulin following all 4 treatments. Addition of the β -glucan supplement significantly blunted the glycaemic and insulinaemic responses on the food ($p < 0.05$) but not drink ($p > 0.05$) treatments when compared to controls. The high-CHO breakfasts decreased total, LDL- and HDL-cholesterol from baseline to 60mins postprandially but there were no differential effects of β -glucan treatment on circulating lipids. We conclude that a high dose barley β -glucan supplement can improve glucose control when added to a high-CHO starchy food, probably due to increased gastro-intestinal viscosity, but not when added to a high-CHO beverage where rapid absorption combined with decreased β -glucan concentration and viscosity may obviate this mechanism.

Key Words: soluble fibre, barley β -glucan, carbohydrate, glucose, insulin

Introduction

The protective role of whole grain foods in the prevention of coronary heart disease is reasonably well established¹ with a body of evidence supporting the hypocholesterolaemic effects of cereal-derived soluble fibre in the diet.¹⁻⁵ Evidence that soluble fibre may also reduce the incidence of type II diabetes (T2DM) is not as robust although there is growing support from a number of epidemiological trials that both high β -glucan whole grain products and high-fibre products *per se* may be protective.⁶⁻⁹

Linear mixed-link (1 \rightarrow 3)(1 \rightarrow 4)- β -D glucan (β -glucan) is found predominantly in the endosperm wall of cereals, is present in relatively high levels in oats and barley and appears to be an active component of these cereals. The majority of β -glucan intervention studies have focused on oat rather than barley cereal since it is a more prevalent item within the diet despite the higher natural content found in barley cereal (5-10% w/w β -glucan). Trials have shown the beneficial effects of oat β -glucan on lipids to be mediated long term through bile acid binding and subsequent removal of cholesterol from circulation.¹⁰⁻¹² Whilst pro-

longed feeding appears to have little effect on fasting glucose and insulin levels, postprandial trials have shown acute glycaemia and insulinaemia to be blunted in the hours following intake of oat soluble fibre in healthy^{6,13,14} as well as diabetic¹⁵ subjects. This may be a consequence of the rapidly increased gastrointestinal (GI) viscosity that occurs following a soluble fibre meal¹³, which in turn leads to a slower rate of digestion in the intestinal lumen and slower absorption of glucose into the portal and systemic circulation and a reduced demand for insulin.¹⁶ Improved postprandial glucose control may in part explain the emerging role of soluble fibre in reduction of T2DM risk.

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In order for β -glucan enriched barley products to be successful they must fulfil the requirements for both acceptable sensory properties and improvement in risk factor profile. Previous work from our laboratory has urged caution when developing these enriched products and highlights the need to confirm efficacy.¹⁷ We supplemented a group of hyperlipidaemic men with 6g/day of a highly enriched, processed form of barley β -glucan for a period of 3 weeks but were unable to detect an improvement in lipid profile, despite the body of evidence of hypocholesterolaemic effects of β -glucan. We and other authors¹⁸ have attributed this to alteration in bioactivity effected during enrichment processing.

In our current trial we wanted to investigate the effect of a high-CHO barley β -glucan enriched meal on postprandial measures of T2DM and CVD risk. Barley β -glucan has been shown to improve glucose and insulin control in some¹⁹⁻²³ but not all²³⁻²⁵ postprandial studies of healthy subjects and also in diabetics.²⁰ In particular we were interested to determine whether the β -glucan supplement could improve glucose response to a high-CHO challenge when given within a breakfast food and a breakfast drink. To do this we compared a solid and liquid test meal supplemented with a commercial barley β -glucan product with a no-added-fibre food and drink treatment matched for available CHO in a group of healthy men.

Methods

Subjects

Twenty lean, male volunteers aged 21-34 years entered this 4 treatment intervention. Two subjects withdrew after the first treatment; a result of illness and non-compliance with the protocol respectively. Eighteen subjects completed the trial. All had normal clinical biochemistry as assessed by lipid profile, fasting blood glucose and blood pressure. None had a current or previous history of treatment for significant disease, nor were they taking medications for lipid, blood pressure or metabolic disorders. All participants provided written informed consent. Ethics approval for this protocol was obtained from the Auckland Ethics Committee, New Zealand and conformed with the 1995 Declaration of Helsinki as revised in Edinburgh 2000.

Protocol

This was a 4 treatment cross-over trial, where subjects were randomised to (i) high-CHO_{food control} (ii) high-CHO_{food+fibre} (iii) high-CHO_{drink control} and (iv) high-CHO_{drink+fibre}. Participants were randomised to treatment using a 4 x 4 Latin Square, each treatment separated by a minimum 7 day washout. A fixed 10g dose of high barley β -glucan fibre supplement (CerogenTM, Roxdale Foods Ltd, Auckland, NZ) was added to food and drink controls.

Barley β -glucan supplement

The commercial fibre supplement contained 6.31g β -glucan/10g fibre (Table 1). It was added to the food and drink treatments immediately prior to presentation to the subjects, and was hand stirred into berry jam on food and hand stirred into the cold beverage on drink treatment. *In*

vitro dynamic viscosity of β -glucan in the fibre supplement was determined in a calibrated Cannon-Manning semi-micro viscometer (Cannon Instruments, USA) at 25 °C. The fibre supplement was not frozen, heated or treated in any way during preparation of the breakfasts. Table 2 shows details of the high-CHO food and drink treatments which were balanced for energy content and available-CHO between control and high-fibre arms. Food treatment contained both sugars and starch, whilst starch was absent from the drink treatment. The food treatments comprised a standard high-energy (approximately 24% of energy requirements), high-CHO breakfast of white bread and berry jam. The drink treatments comprised a commercial sports drink (PoweradeTM, Coca Cola Amatil Ltd, Auckland, NZ) supplemented with high glucose syrup (PolycalTM, Nutricia, Zoetermeer, The Netherlands).

Table 1. High barley β -glucan fibre supplement. A fixed 10g dose was given to each subject on the food + fibre and drink + fibre treatments.

Component	g/100g fibre supplement
Total CHO	81.1
β -glucan	63.1
arabinoxylans	6.5
sucrose	2.0
maltose	9.5
Total Protein	7.9
Total Lipid	1.7
Moisture	7.2

Blood tests

The effect of the high-CHO test meals on blood glucose, insulin and lipids was assessed through collection of venous blood samples over a 6 hour postprandial period. Subjects arrived fasted at the human nutrition unit at 0730h, consumed 200ml of cold water, an indwelling venous cannula was inserted and a fasting baseline blood sample collected. At 0800h one of the 4 high-CHO test meals was given with 300ml water, and consumed within 15 minutes. A further 750 ml of water was consumed by the subjects throughout the morning. Blood samples were collected at 15, 30, 45, 60, 90, 120, 180 and 360 minutes postprandially, based on an extended FAO/WHO 7 sample protocol²⁶ as recently assessed by Wolever²⁷ and analysed for glucose and lipids. A subset of samples from all subjects was analysed for serum insulin (0, 60, 180, 360 minutes). Subjects were confined within the human nutrition facility at the University of Auckland throughout each treatment. All were sedentary and refrained from smoking during the test.

Analytical methods

Venous blood samples were analysed for glucose, insulin, total cholesterol (TC), HDL-cholesterol (HDL-C) and triacylglycerol (TAG). LDL-cholesterol (LDL-C) was calculated using the Friedwald equation²⁸. Serum TC, HDL-C and TAG were measured in duplicate by COBAS MiraTM auto-analyser (Hoffman-La Roche Ltd, Basel, Switzerland). A 3-step enzymatic colour method utilis-

ing cholesterol esterase, cholesterol oxidase and peroxidase was used to analyse TC. A 2 step precipitating reagent set (Pointe Scientific, Canton, MI, USA) followed by enzymatic cholesterol analysis was used for analyses of HDL-C. A TAG-GPO reagent set multi-step enzymatic colour reaction method (Pointe Scientific) was used to analyse total TAG. Plasma glucose concentration was measured enzymatically using the glucose hexokinase reagent set method (Pointe Scientific). Serum insulin concentration was measured by micro-particle enzyme immunoassay (MEIA) technology, a non-competitive sandwich assay, using a commercial reagent kit (Abbott Diagnostics, Illinois, US). Insulin is bound by anti-insulin antibody bonded to micro-particles which are then trapped within a matrix cell by glass fibre matrix, washed and a second anti-insulin antibody conjugated to alkaline phosphatase added. Labelled antibody not bound to insulin is removed by washing. Addition of enzyme substrate methylumbelliferyl phosphate results in fluorescence dependent on the presence of bound enzyme label.

Statistical analyses

Between treatment comparison of blood parameters over time was made by repeat measures 2-way ANOVA, performed using GraphPad Prism version 4.02 for Windows (GraphPad Software, San Diego, Ca, USA, www.graphpad.com). Calculation of the integrated area under the curve (AUC) for changes in blood glucose and insulin was also calculated using GraphPad Prism (San Diego, Ca, USA) and comparisons made by paired t-test. Screen data is presented as mean \pm SD. All metabolic outcome variables are expressed as mean \pm SE.

Results

Eighteen young, lean, healthy men completed this 4 treatment trial. Mean age was 27 (5.9, SD) years, body mass index (BMI) was 22.9 (2.1, SD) kg/m² and waist circumference was 77.8 (5.9, SD) cm. All subjects had normal glucose control when defined by fasting plasma glucose <5.5 mmol/L, (mean 4.6, 0.6 SD mmol/L), and were normotensive (mean SBP: 110, 11 SD mmHg; mean DBP: 61, 6 SD mmHg). There was no evidence of hyperlipidaemia based on fasting total-cholesterol (mean 4.5, 0.8 SD mmol/L), LDL-C (mean 2.7, 0.8 SD mmol/L), HDL-C (mean 1.3, 0.3 SD mmol/L) and TAG (mean 1.0, 0.5 SD mmol/L). Mean fasting insulin was 35 (17, SD) pmol/L. There was no significant difference between treatments at baseline for any parameter ($p > 0.05$).

In vitro viscosity of barley β -glucan supplement

The weight of β -glucan as a percentage of total water content of the test meal (104g) plus drinking water (300g) was 1.6%. The dynamic viscosity of β -glucan at a concentration of 1.6% was determined to be 3.7 mPa.s. When added to the berry jam, informal comments from the subjects indicated that there was no noticeable change in taste or texture of the test meal. The addition of fibre increased the weight of the jam component of the meal. The weight of β -glucan as a percentage of total water content of the test drink (640g) plus drinking water (300g) was 0.6%, and hence lower than on the food treatment. The dynamic viscosity of β -glucan at a concentration of

0.6% was determined to be 1.6 mPa.s. When added to the drink, informal comments from the subjects indicated that, whilst there was no significant change in taste of the test drink, there was a change in both visual appearance and texture. The fibre enriched drink had clearly visible particular suspension which, when consumed, had the texture of pithy orange juice.

Glucose & Insulin

The change in blood glucose in response to the high-CHO challenge meals is shown in Table 3. Maximum peak glucose occurred 30 minutes after the high-CHO meal in both the food control and food + fibre arms and was not different between treatments (ANOVA, $p > 0.05$). The AUC calculated from the change in glucose from baseline over 360 minutes (Fig 1, top panel) was significantly lower on the food + fibre treatment (354.5 ± 44 mmol-min/L) when compared with food control (452.6 ± 50 mmol-min/L, $p < 0.05$, paired t-test). The appearance of glucose in venous circulation was more rapid when the dietary CHO was given as a drink. Circulating glucose was 30% and 16 % higher respectively on drink control at 15 and 30 minutes post CHO load than on food control (see Table 3, $p < 0.05$). Maximum peak glucose also occurred 30 minutes after the high-CHO meal in both the drink control and drink + fibre arms and was not different between treatments (ANOVA, $p > 0.05$). AUC of the change in glucose from baseline over 360 minutes (Fig 1, bottom panel) was not significantly lower on the drink + fibre treatment (327.0 ± 53 mmol-min/L) when compared with drink control (349.4 ± 68 mmol-min/L, $p > 0.05$). The more rapid appearance and disappearance of glucose in venous circulation and return of fasting glucose to baseline concentrations by 180 minutes post meal resulted in a lower AUC on drink control compared with food control (paired t-test, $p < 0.05$). AUC calculated from the change in insulin from baseline over 360 minutes (Fig 2, top panel) was significantly lower on the food + fibre treatment ($52,703 \pm 8,076$ pmol-min/L) when compared with food control ($67,265 \pm 10,263$ pmol-min/L, $p < 0.05$, paired t-test) but was not significantly lower on the drink + fibre treatment ($83,298 \pm 19,017$ pmol-min/L) when compared with drink control ($90,987 \pm 20,907$ pmol-min/L, $p > 0.05$, Fig 2, bottom panel). There was a more rapid and greater insulin response following the high-CHO drink control when compared with food control. At 60 minutes post dose, insulin concentration was 75% higher following the high-CHO drink (850.4 pmol/L) than on food treatment (483.5 pmol/L, $p < 0.05$, Fig 2).

Lipids

There was a significant decrease in TC, LDL-C and HDL-C between baseline and 60 minutes on all treatments (Figure 3, ANOVA, time, $p < 0.001$). Circulating levels of cholesterol and fractions gradually returned to close to baseline levels between 60 and 360 minutes. There were no significant between treatment effects, hence no difference between food control and food + fibre, nor drink control and drink + fibre over any time point.

The pattern of change in circulating TAG did not mimic change in cholesterol (see Fig 3). Analysis of treatments combined showed a decrease between 60-120

Table 2. Composition of the 4 high-CHO treatments

	Energy (kJ)	Fibre supplement (g)	Barley β -glucan (g)	Total CHO (%)	Total CHO (g)	Available CHO (g)	Monosac- charide (g)	Non-starch available polysaccharide (g)	Starch (g)	Fat (g)	Protein (g)
Food control	3006	0	0	79	150.7	145.2	64	0	79	4.0	15.5
Food + fibre	3071	10	6.3	80	157.4	145.0	64	0	79	4.1	16.0
Drink control	2458	0	0	92	144.5	144.5	62	80	0	0.0	0.0
Drink + fibre	2544	10	6.3	94	152.0	145.1	62	80	0	0.2	0.8

Table 3. Glycaemic response to the high-CHO breakfasts (food control and drink control) and following supplementation with 6.3g of barley β -glucan (food + fibre and drink + fibre)

	Plasma glucose (mmol/L)									
	0m	15m	30m	45m	60m	90m	120m	180m	360m	
<i>High-CHO_{food control}</i>	4.93±0.1	6.25±0.2	8.80±0.2	8.71±0.3	7.74±0.4	6.89±0.2	6.41±0.2	6.10±0.2	4.86±0.1	
<i>High-CHO_{food+fibre}</i>	5.03±0.1	6.34±0.2	8.46±0.3	8.13±0.3	7.48±0.4	6.39±0.2	6.29±0.1	5.88±0.2	4.84±0.1	
<i>High-CHO_{drink control}</i>	4.91±0.1	7.73±0.3	9.51±0.4	8.76±0.5	7.85±0.4	6.49±0.4	6.75±0.3	4.83±0.4	4.89±0.1	
<i>High-CHO_{drink+fibre}</i>	4.96±0.1	7.61±0.3	9.35±0.3	8.55±0.4	7.63±0.4	6.44±0.2	6.43±0.2	4.97±0.3	4.91±0.1	

CHO, carbohydrate; m, minutes; Mean ± SE

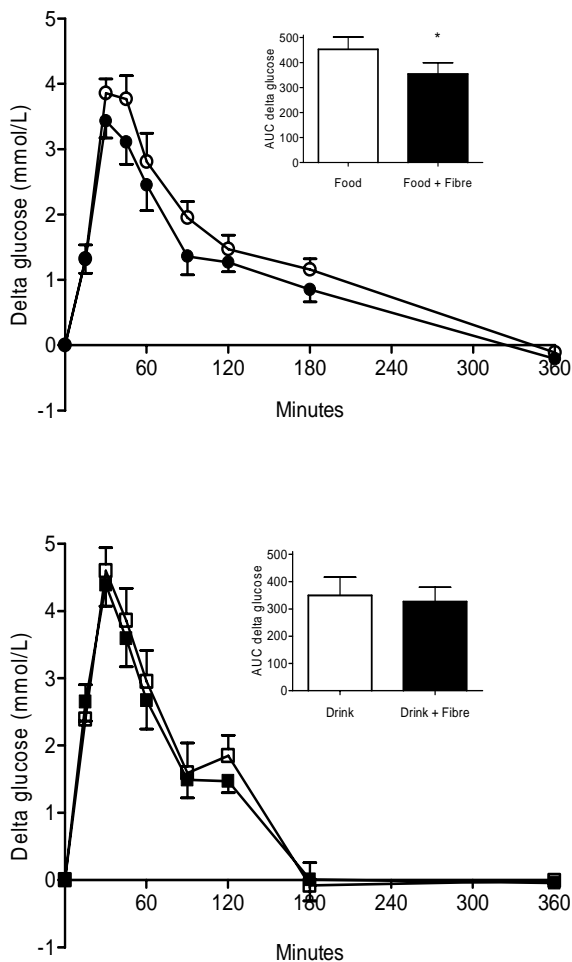


Figure 1. Postprandial changes in blood glucose following supplementation of a high-CHO breakfast meal (top panel: food \circ , food + fibre \bullet) and drink (bottom panel: drink \square , drink + fibre \blacksquare) with barley β -glucan. There was a significant decrease in the area under the delta glucose curve (AUC delta glucose) during the 360 minutes following the meal treatment (* $p < 0.05$).

minutes post CHO test load ($p < 0.001$). There was a small but significant early post meal increase in TAG over 0-60 minutes ($p < 0.05$). There were no differential effects of β -glucan supplementation on either food or drink treatments.

Discussion

Whilst there is considerable evidence to show that cereal fibres are protective against cardiovascular disease^{1, 29-31} and which underpins the US Food and Drug Administration (USFDA) ratification of health claims supporting the relationship between soluble fibre and risk of coronary heart disease³², far fewer trials have investigated the role of viscous fibres in prevention of type II diabetes.^{7, 8, 18, 33} Soluble fibre β -glucan has been proposed as an important active component of cereals and protection against heart disease may be in part due to a decrease in fasting lipids levels, specifically cholesterol-rich lipoproteins.^{3, 10, 11} Conversely, protection against T2DM may be driven through acute changes in postprandial glycaemic response to eating^{13, 15, 19, 20, 22, 23} rather than a suppression of fasted basal glucose concentration. Relatively few trials have investigated the action of barley derived

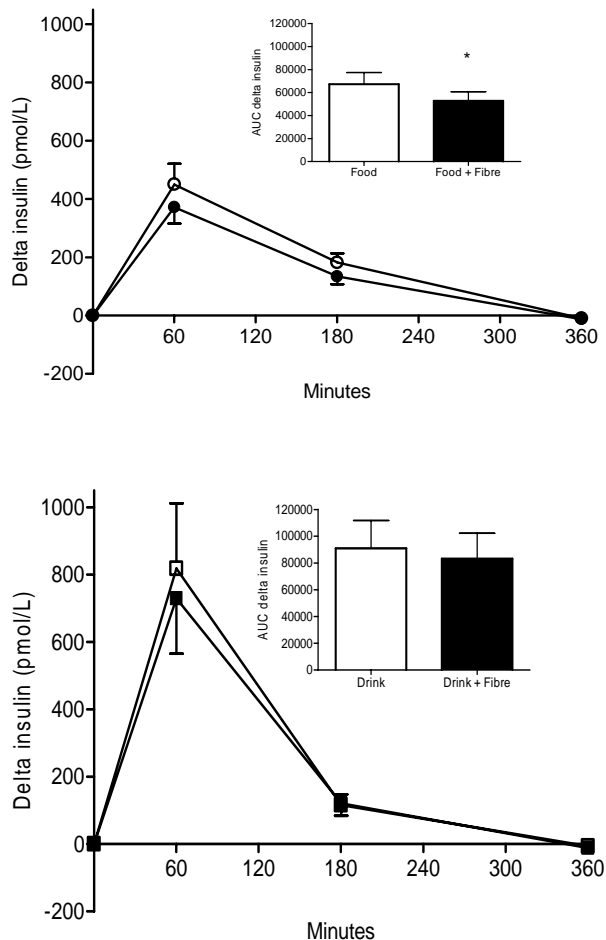


Figure 2. Postprandial changes in serum insulin following supplementation of a high-CHO breakfast meal (top panel: food \circ , food + fibre \bullet) and drink (bottom panel: drink \square , drink + fibre \blacksquare) with barley β -glucan. There was a significant decrease in the area under the delta insulin curve (AUC delta insulin) during the 360 minutes following the meal treatment (* $p < 0.05$).

β -glucan since it is a less common dietary component compared with cereals such as oats, although the natural β -glucan content of barley cereal is high.³⁴ In order to enhance glucose control and contribute to protection against development of T2DM it would be advantageous to encourage barley consumption through introduction of enriched barley β -glucan products into the diet if efficacy can be well demonstrated. The development of a naturally high β -glucan barley genotype, *Prowashonupana*, has been a reasonably successful advancement in this area.^{20, 23}

Data from our current trial showed that glycaemic response of healthy men to a high-CHO food but not to a high-CHO drink could be significantly blunted by the addition of an enriched barley β -glucan viscous fibre. Whilst peak glucose concentration was reached at 30 minutes postprandially on both of the fibre-free food and drink treatments, glucose appearance in venous circulation was significantly more rapid when the CHO bolus was given in liquid form. This implies that gastric emptying and/or small intestine absorption and hence transport of monosaccharide into circulation via the portal vein occurred more quickly following the CHO drink.

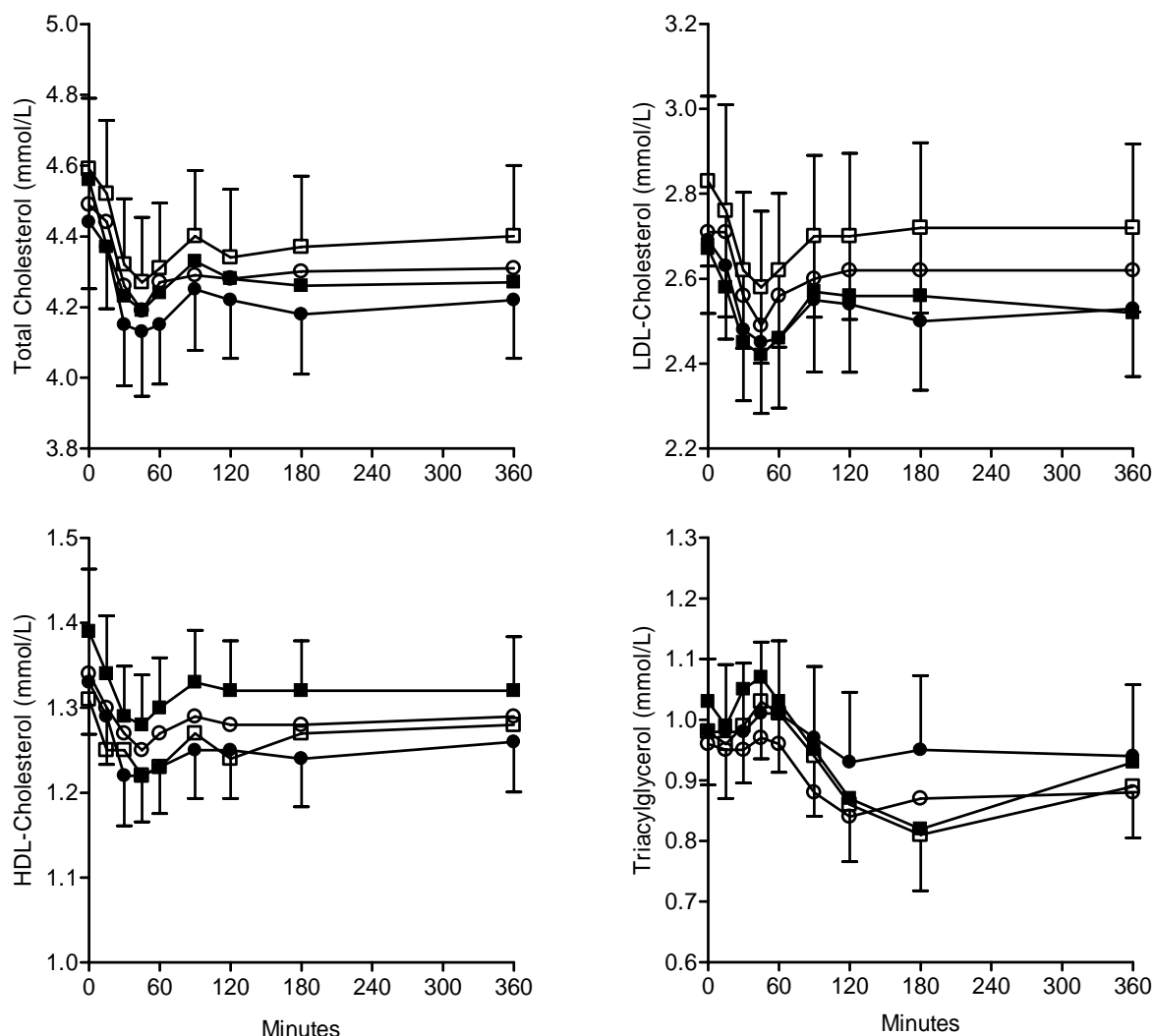


Figure 3. Postprandial changes in lipid profile following supplementation of a high-CHO breakfast meal (food \circ , food + fibre \bullet , drink \square , drink + fibre \blacksquare) with barley β -glucan. There was a significant decrease in TC, LDL-C and HDL-C between 0-60 minutes ($p < 0.001$) and TAG between 60-120 minutes ($p < 0.001$) when analysed as all treatments combined. No difference in lipid profile was found between treatments.

Certainly absence of starch in the CHO drink would be expected to increase rate of absorption relative to the food (bread) treatment where amylase action is an additional digestive step. When fibre was added to the drink it is possible that there was insufficient time for formation of a viscous environment within the gut, hence post-absorptive glycaemia proceeded unchecked. Alternately, if starch is an important component in fiber-induced development of small intestine viscosity then absence of this complex CHO in combination with the relatively lower *in vitro* viscosity of the drink treatment may be important. The observation of rapid transport of glucose across the gut wall and into venous circulation following the high-CHO liquid bolus in our trial is also supported by the rapid increase in insulin concentration and the greatly increased peak value on drink control treatment.

Lower dynamic viscosity of the β -glucan supplement when incorporated into the CHO drink may have lead to decreased GI viscosity. Earlier oat trial data has shown that glycaemic response depends on $\log(\text{viscosity})^{13}$, and

that a decrease in β -glucan content from 1.6% to 0.6% would be predicted to decrease glucose lowering by up to 2.8 times. GI glucose absorption is reliant on intestinal contractions which create turbulence, mix GI contents and bring them to the epithelial surface, and the diffusion of glucose across the layer of fluid against the epithelium.³⁵ The action of viscous fibres may include prevention of contact of nutrients with the surface of the mucosal epithelium^{36, 37}, inhibiting glucose transport across the intestinal mucosa through decreased mobility of fluid layers covering intestinal villi.³⁸ *In vitro* dialysis models have shown that the most viscous fibres produce the greatest slowing of glucose absorption.³⁹ Postprandial studies assessing intestinal absorption of glucose *in vivo* using breath hydrogen measurements following a high fibre CHO load confirm decreased rate of CHO absorption.⁴⁰ Similarly, in a study of diabetics fed meals of cooked barley, rice or wheat pasta there was a decreased rate of absorption of barley directly correlating to *in vitro* starch digestibility data.⁴¹ Intestinal perfusion methods have also demonstrated CHO gelling properties of vis-

cous apple pectin³⁸ and a glucose solution containing guar gum³⁶ both of which decreased glucose absorption. Hence it is reasonable to assume that decreased *in vitro* viscosity may result in decreased *in vivo* gut viscosity and decreased efficacy.

The inability of barley β -glucan to improve glucose control when added to a drink has also recently been observed by Biorklund et al., who supplemented beverages with 5g and 10g barley β -glucan as part of a low-energy breakfast in hypercholesterolaemic subjects but observed no improvement in postprandial glycaemia.²⁴ Conversely oat gum which contains approximately 80% β -glucan has been shown to improve glucose control when added to a 50g liquid glucose load.^{13, 14} Supplementing beverages with 5g and 10g oat β -glucan also improved postprandial glucose control in the Biorklund study²⁴. Hence there may be mechanistic differences between β -glucan of different cereal origins. There are examples of better glucose control using barley rather than oat-derived β -glucan but only in studies of the high β -glucan barley genotype *Prowashonupana*.^{20, 23}

Whilst addition of barley β -glucan in our trial did not blunt maximal glucose response on either treatment, over 360 minutes there was a significant decrease of 21% in the area under the delta glucose curve when the fibre was added to food treatment. A change in postprandial glucose of this magnitude would represent an improvement in clinical risk for diabetic patients in whom raised fasting and raised post-meal glucose contributes to protein glycation and ensuing vascular complications. Whilst basal levels of intermediary metabolism are commonly chosen as markers of risk, there is growing evidence that postprandial changes are also important since much of each 24h is spent in the postprandial state. A well established marker of CVD risk is postprandial TAG, with independent relationships between post meal levels of chylomicron-TAG and CVD.⁴² By eating three meals a day we are constantly exposed to increased glycaemia. Blunting the postprandial rise will ensure less glycation of susceptible protein moieties.

We conclude that postprandial glycaemic response to supplementation of dietary CHO with an enriched source of barley β -glucan may depend upon the form of the CHO consumed, the water volume of the meal and hence the final viscosity of the supplement. In this trial of healthy men, plasma glucose increased more rapidly when the CHO load was in liquid rather than solid form, there was no evidence of improvement in glucose control when barley soluble fibre was added to a cold beverage and it is possible that rapid absorption of monosaccharide from the small gut or high water content of the drink prevented an increase in GI viscosity. Addition of soluble fibre barley β -glucan to a high CHO food significantly improved postprandial glycaemia, probably due to action of the viscous fibre within the gut. This improvement in postprandial glucose control would be expected to also result in an improvement in diabetic risk if barley β -glucan is included as a habitual dietary constituent.

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Original Article

Supplementation of a high-carbohydrate breakfast with barley β -glucan improves postprandial glycaemic response for meals but not beverages

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補充含有大麥 β -glucan 的高糖早餐而非飲料可以改善飯後血糖反應

有愈來愈多證據支持水溶性纖維素對第二型糖尿病的保護角色。包含燕麥及大麥等穀類產品中所含的水溶性纖維 β -glucan 可能是有效成分。有證據指出在飲食中補充燕麥的水溶性纖維，可以減弱飯後血糖及胰島素對膳食醣類的反應。但是只有少數的試驗使用天然大麥或是富含大麥 β -glucan 的產品，本試驗的目的為研究給予高糖(CHO)食物及高 CHO 的飲料同時給大量富化大麥 β -glucan 的產品對於飯後血糖、胰島素及脂質的影響。18 名瘦的健康男性完成四種治療介入試驗，包含(i) high-CHO_{food control}, (ii) high-CHO_{food+fibre}, (iii) high-CHO_{drink control}, (iv) high-CHO_{drink+fibre}，其中劑量 10 公克的大麥 β -glucan 纖維補充品 (Cerogen) 含 6.31 克的 β -glucan，添加到食物及飲料控制組。全部四個治療組的血糖跟胰島素都上升。添加 β -glucan 補充品在食物治療組($p < 0.05$)但不是飲料治療組比起控制組($p > 0.05$)其血糖及胰島素反應均呈現顯著的緩慢。高-CHO 的早餐降低飯後基線到 60 分鐘後的總膽固醇、LDL-膽固醇及 HDL 膽固醇，但是 β -glucan 治療對於循環脂質則沒有可分辨的影響。我們總結當添加高劑量的大麥 β -glucan 補充品在高-CHO 澱粉食物中，可以改善血糖控制，可能是由於胃腸黏性增加，但是高 CHO 飲料則沒有此現象，可能是其被快速吸收，再加上 β -glucan 濃度及黏性的降低可能消除了這個機制。

關鍵字：水溶性纖維、大麥 β -glucan、醣類、葡萄糖、胰島素。