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Oat beta-glucans consumed at breakfast improve glucose tolerance acutely and after a subsequent lunch – a randomized dose–response study in healthy young adults†

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Oat beta-glucans (OBGs) lower postprandial blood glucose by increasing gastrointestinal viscosity, delaying gastric emptying, and slowing glucose absorption. While the European Food Safety Authority (EFSA) recommends a minimum intake of 4 g of OBGs per 30 g of available carbohydrates (avCHO) for a significant reduction in glycaemic response, this poses formulation challenges. This study investigated the effects of a commercially available OBG ingredient on postprandial glycemia and appetite sensations immediately after ingestion and following a standardized lunch 3.5 hours later, also exploring whether doses below 4 g of OBGs per 30 g of avCHO could be effective. Nineteen healthy subjects consumed test drinks containing 0 g (Ref), 2 g (BG2), 3 g (BG3), or 4 g (BG4) of OBGs, each providing 30 g of avCHO, in a crossover study. BG2 and BG4 reduced the incremental glucose peak (iPeak) compared to Ref ($P < 0.05$), with BG3 showing a trend ($P = 0.09$). BG4 reduced an early glucose incremental area under the curve (iAUC 0–60 min) and improved the post-lunch glycaemic response compared to Ref ($P < 0.05$). Insulin iPeaks and iAUC (0–120 min) were lower for BG3 and BG4 ($P < 0.05$). BG4 enhanced satiety and reduced hunger throughout the experimental period ($P < 0.05$). Doses below 4 g of OBGs per 30 g of avCHO improved postprandial glycemia and appetite, and OBG intake at breakfast enhanced post-lunch glycaemic regulation, suggesting that a lower threshold may be effective in blood glucose management and appetite control.

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Introduction

Oats (*Avena sativa* L.) are rich in health-promoting components, such as soluble dietary fibers (beta-glucans (BGs) and arabinoxylans). Beta-glucans from oats and barley (BBGs) have been shown to reduce acute postprandial blood glucose concentrations.[1–3] Maintaining strictly regulated blood glucose concentrations is crucial, resulting in a potentially reduced risk of obesity, type 2 diabetes and cardiovascular diseases.[4,5] The postprandial glycemia-modulating effect of BGs is believed to relate to its ability to increase the viscosity of the content of the gastrointestinal tract, which delays the gastric emptying rate, restricts the interaction of digestive enzymes with their substrate, and leads to slower carbohydrate digestion and absorption of glucose.[6,7] The effectivity of BGs in producing this viscous layer depends on their concentration, solubility/extractability, and molecular weight.

In the European Union (EU), the EFSA concluded, following a review of the scientific evidence, that the postprandial glycaemic response is reduced with the intake of at least 4 g of OBGs or BBGs per 30 g of avCHO.[8] However, due to the markedly high viscosity of BGs, the incorporation of 4 g of OBGs or BBGs per 30 g of avCHO is technologically challenging when formulating a commercial product. To our knowledge, there is no commercial product in the European market that complies with that specific ratio of BGs to avCHO. Based on recent studies, the exact ratio of BGs to avCHO required for postprandial glycaemic response lowering may, indeed, be lower than the minimum threshold established by the EFSA, particularly when the molecular weight of BGs is considered.[9]

Barley kernels, which are rich in BGs, have been shown to reduce the postprandial glycaemic response long after consumption; indeed, when consumed as part of a standardised

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† Electronic supplementary information (ESI) available: (1) Table S1: macronutrient composition of a standardised lunch meal, (2) Fig. S1: the consort flow diagram and study progress. See DOI: https://doi.org/10.1039/d5fo00353a

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dinner, the postprandial glycaemic response was reduced the following morning after the consumption of a standardised breakfast.[10] Likewise, when consumed as part of a breakfast test meal, barley kernels reduced the postprandial glycaemic responses after the consumption of the subsequent standardised lunch and dinner meals.[10] This phenomenon, known as the 'second meal effect' on blood glucose regulation, plays a crucial role in the context of maintaining glycaemic control over an extended period of time (*e.g.* breakfast to lunch). Similarly, it has been reported that BBGs improve second meal blood glucose responses in mice; specifically, Mio *et al.* reported that mice receiving a single dose of BGs, followed by a standard glucose tolerance test conducted 6 h later, showed an improved glycaemic response to the standard glucose load.[11] To our knowledge there is no information available regarding the effects of isolated OBG preparations on second-meal blood glucose tolerance in humans.

The aim of this study was to investigate the impact of a commercially available OBG ingredient on postprandial glycemia and subjective appetite sensations, immediately after ingestion, and, after a standardised lunch consumed 3.5 h following the intake of OBGs. The OBG ingredient was added to a glucose-based drink. We also investigated the effects of doses lower than 4 g of OBGs per 30 g of avCHO on the postprandial glycemic responses. For this purpose, a randomized crossover study was performed in healthy young adults.

Materials and methods

Study subjects

Participants were recruited through public advertisements on **https://www.accindi.se** and Lund University notice boards. Recruitments of test subjects took place between December 2022 and February 2023, and the clinical phase took place between March and May 2023. The inclusion criteria were individuals aged between 20 and 40 years with BMIs between 19 and 28 kg m^{-2}. The exclusion criteria included: fasting blood glucose concentration $\geq$ 6.1 mmol L^{-1}, any diagnosed metabolic diseases, regular use of oral hypoglycaemic drugs or corticosteroids, current smoking, changes in diet or physical activity within the past three months, regular oat or oat-based product consumption, and any diagnosed eating disorders (*e.g.*, binge eating) that could compromise satiety assessments. Each subject received a full explanation (written and oral) of the purpose and protocol of the study, and written informed consent was obtained. Test subjects were aware of the ability to withdraw from the study at any time. Anthropometric measurements, including body weight and height, were recorded during the screening visit using a calibrated digital scale (TANITA MC-780U). BMI was calculated by dividing weight (kg) by the square of height (m^2). Nineteen healthy volunteers, 7 men and 12 women, aged (mean ± SEM) 23.51 ± 0.21 years, and with a BMI (mean ± SEM) of 23.39 ± 0.46 kg m^{-2} were enrolled to participate in the study. The consort flow diagram and study progress are shown in ESI Fig. S1.†

Breakfast test drinks

The OBGs were kindly provided by Lantmännen Functional Foods AB (PromOat® Instant, Lantmännen Functional Foods AB, Box 30192, 104 25 Stockholm, Sweden). All breakfast test drinks contained 30 g of avCHO and one of four different doses of OBGs, as follows: (i) 4 g of BGs (BG4); (ii) 3 g of BGs (BG3); (iii) 2 g of BGs (BG2); or (iv) 0 g of BGs, considering the reference (Ref). The breakfast test drinks were prepared by adding OBGs in 150 mL of hot water (100° C) with added strawberry flavour (wild strawberry, Aarke AB, Östgötagatan 100, Stockholm, Sweden) and pink food colours (Dr Oetker Sverige AB, Åvägen 40, 412 51 Göteborg, Sweden). The composition of PromOat® Instant is shown in Table 1, and the composition of each breakfast test drink is shown in Table 2. The average molecular weight of PromOat® Instant was 800 kDa.

Standardised lunch meal

A standardised lunch meal was served 3.5 h (210 min) after the breakfast test drink to investigate second-meal effects. The standardised lunch meal consisted of 50 g of avCHO from 121 g of white wheat bread and 8 g of avCHO from 100 g of Swedish meatballs (Scan AB, Halmstad, Sweden), for a total of 58 g of avCHO for the entire lunch meal. According to the nutritional information given by the manufacturers, the lunch meal contained a total caloric value of 485 kcal (ESI Table S1†).

Study design

The study was conducted using a single-blind randomized crossover design. Each breakfast test drink was consumed by all participants in a random sequence. The blinding was applied solely to the participants. Prior to each study visit, participants were instructed to refrain from engaging in vigorous physical activity, consuming alcoholic beverages, and consuming foods containing oats or high in dietary fiber (such as beans, whole grain bread, fiber-enriched pasta, and whole cereal kernels). The participants were instructed to establish a standardised food routine before each study day. In order to ensure consistency, participants were instructed to provide a record of their dietary intake from the day preceding each study visit. Additionally, participants were instructed to consume standardized meals in the evening prior to each

Table 1 Composition of PromOat® Instant[a]

Nutritional information	Nutrients per 100 (g)
Moisture	3.00
Available carbohydrates	47.00
Sugars	2.00
Total fat	6.50
Protein	3.50
Total fibre	40.00
Beta glucans	32.20
Other fibre (AX)	7.80

[a] In accordance with the manufacturer's provided information.

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Table 2 Composition of breakfast test drinks[a]

Drink	PromOat (g)	Beta-glucans (g)	AvCHO (g)			Protein (g)	Fat (g)	Total fibre (g)	Total energy (kcal)
			From PromOat	From glucose	Total				
BG4	13.15	4.00	5.99	24.00	30.00	0.45	0.83	5.10	135
BG3	9.86	3.00	4.49	25.50	30.00	0.34	0.62	3.83	131
BG2	6.50	2.00	2.96	27.04	30.00	0.22	0.41	2.52	127
Ref	0.00	0.00	0.00	30.00	30.00	0.00	0.00	0.00	120

[a] All drinks were prepared using 150 mL of hot water (100 °C).

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study visit. Specifically, they were asked to have a standardized dinner at 18:00 and an evening snack at 21:00. The standardized dinner was selected by each participant and remained consistent across all pre-visit evenings. The standardized evening snack consisted of commercially available white wheat bread with a topping of the participant's choice, ensuring the same topping was used in all pre-experimental evenings to maintain consistency.

The participant arrived at the study centre at 07:30 after 10 hours of fasting during the night. Capillary blood samples were collected, followed by the consumption of a breakfast test drink at 08:00 (time zero (0 min)), with a designated consumption time of 10–12 minutes. Subsequent capillary blood samples were collected at specific time intervals of 15, 30, 45, 60, 90, 120, 150, 180, and 210 minutes after the start of consumption of the breakfast test drink. Following the blood test conducted at the 210-minute mark, a standardised lunch was provided which was consumed within 10–12 min, and further blood samples were collected at intervals of 225, 240, 255, 270, 300, and 330 minutes following the start of consumption of the breakfast test drink. Throughout the duration of the trial, the participants were confined to the clinical facility and were strictly prohibited from consuming any food or beverages, with the exception of the provided breakfast test drink and standardised lunch meal. Participants were instructed to minimize their engagement in physical activities to the greatest extent feasible. Participants were monitored for any adverse effects, including gastrointestinal discomfort, dizziness, or nausea. They were instructed to report any symptoms experienced throughout the study period. Each test day was separated by a minimum 5-day wash-out period. An overview of the schedule of procedures and evaluations on each test day is provided in Fig. 1.

Test variables

The measurement of all variables was carried out using capillary blood samples. Plasma glucose concentrations were measured in whole blood at the time intervals specified above, using a HemoCue Glucose 201+ analyzer manufactured by HemoCue AB in Ängelholm, Sweden. Samples for the analysis of serum insulin were obtained using BD Microtainer SST tubes. Serum insulin samples were assessed at the same time points as the glucose determinations, except for 15 and 150 minutes. The sampling tubes were centrifuged for 10 min (2000 G) at a temperature of 25 °C, using an Eppendorf centrifuge model 5425. Subsequently, the serum was separated and kept at a temperature of −40 °C until it was subjected to analysis. The measurement of insulin concentrations was conducted using a solid phase two-site enzyme immunoassay kit (Insulin ELISA 10-1113-01, Mercordia AB, Uppsala, Sweden).

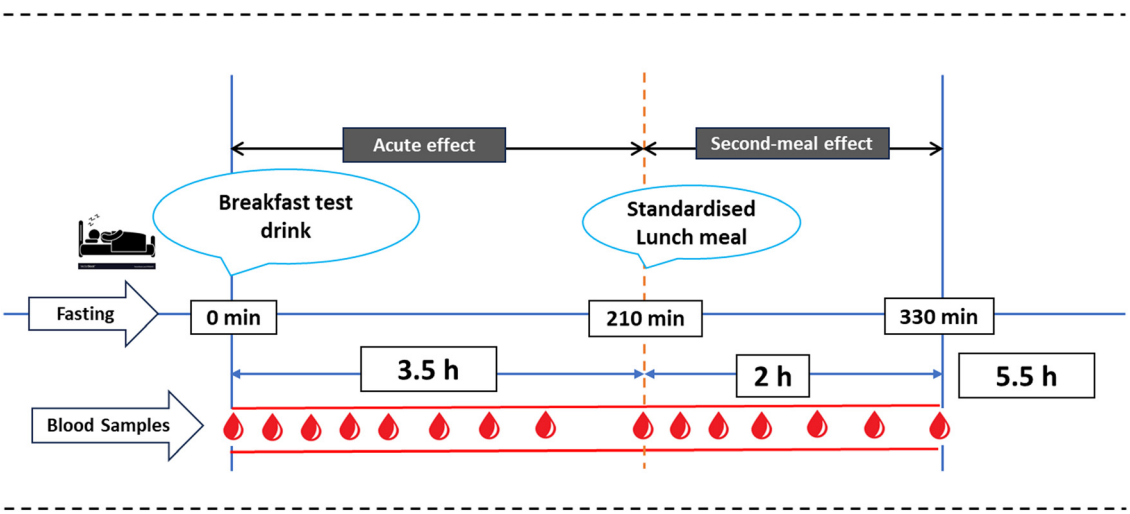

Fig. 1 Schedule of procedures and evaluations on each test day.

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A 100 mm visual analogue scale (VAS) was used to rank the subjective appetite variables namely satiety, hunger, and desire to eat. The left end of the VAS scale represented not at all (*e.g.*, hungry), and the right end of the scale represented extremely. The participants were told to complete the VAS at every blood sampling point.

Statistical analysis

Data are expressed as means ± SEM. A trapezoid model was used to calculate the incremental areas and areas under the curves (iAUCs and AUCs, respectively) for each subject and test drink. The iAUCs were used for statistical evaluations of blood glucose and insulin concentrations. Results presenting iAUCs of postprandial glucose and insulin responses after the standardised lunch (210–330 min) were calculated by considering the glucose concentrations prior to lunch (time = 210 min) as the baseline. AUCs were used to present the results of appetite sensations. The plotting of graphs and calculations of areas were performed using GraphPad Prism (version 10.0, GraphPad software, San Diego, CA, USA). Randomisation of the consumption order of the test drinks was performed by using random tools of Microsoft Excel (Washington, DC, USA). The effects of the test drink on physiological responses were evaluated using ANOVA (general linear model), followed by Tukey's pairwise multiple comparison in MINITAB statistical software (version 21.4, Minitab, Minitab Inc., State College, PA, USA). If the value from a test subject was missing for one of the products, the test subject was excluded from the statistical evaluation of that specific test variable. Data from all 19 participants were included in the statistical evaluations of blood glucose and insulin concentrations. Five subjects failed to follow the instructions in ranking appetite sensations, and these subjects were therefore excluded from the statistical analysis for appetite sensations, resulting in evaluable data for 14 subjects. The significance level was set at a p-value <0.05, using a two-sided test.

Power calculation

The primary outcome measure of the study was incremental blood glucose concentration changes, iAUC, 0–120 min after the breakfast drink. The number of participants required for the study was determined based on a previous study with an oat-based beverage.[12] Assuming a difference of 22 mmol min L^{-1} (15%) between test drinks and a SD of 27 mmol min L^{-1}, with $\alpha = 0.05$ and $1 - \beta = 0.8$,[12] 13 test subjects were required. We decided to increase the number of test subjects to 19 to account for a 30% rate, as has been used in sample size calculations in similar crossover studies with multiple testing arms.

Results

Parameters prior to the consumption of the breakfast test drinks and standardised lunch

The demographic characteristics of the participants are summarized in ESI Table S2.† At the fasting time point (*i.e.*, before consuming the test drinks), there were no statistically significant differences in glucose concentrations, insulin concentrations, or appetite (hunger, satiety and desire to eat) ratings. Likewise, prior to the consumption of the standardised lunch meal, there were no significant differences between treatments in blood glucose or serum insulin. Data can be found in Table 3. Additionally, no adverse effects were reported by participants, indicating that the OBG test drinks were well tolerated.

Glucose and insulin responses

Glucose. Table 4 and Fig. 2, 3 show the glucose response following the consumption of each breakfast test drink and the subsequent standardised lunch meal. The glucose iPeak was significantly reduced following the consumption of the BG4 breakfast test drink *versus* the Ref breakfast drink (−28%, $P < 0.01$; Table 4) and following the consumption of the BG2 breakfast drink *versus* the Ref breakfast drink (−17%, $P < 0.01$; Table 4). There was a 28% reduction in iAUC (0–60 min) following BG4 breakfast drink consumption compared to consumption of the Ref breakfast drink ($P < 0.01$). There was no statistically significant difference in the 0–120 min iAUC between the Ref breakfast drink and any of the OBG breakfast test drinks ($P > 0.05$, Table 4). The iAUC after the standardised lunch with the BG4 breakfast test drink was 24% lower compared to the Ref breakfast test drink ($P < 0.05$, Table 4).

Serum insulin. The insulin concentrations following the consumption of the breakfast test drinks and the subsequent

Table 3 Parameters prior to the consumption of the breakfast test drinks and standardised lunch

Test variable	Ref	BG2	BG3	BG4
Fasting blood glucose (mmol L^{-1})[a]	5.19 ± 0.06[a]	5.13 ± 0.08[a]	5.06 ± 0.09[a]	5.12 ± 0.09[a]
Blood glucose prior to Std. lunch (nmol L^{-1})[a]	4.76 ± 0.11[a]	4.72 ± 0.09[a]	4.72 ± 0.13[a]	4.82 ± 0.11[a]
Fasting serum insulin (nmol L^{-1})[a]	0.033 ± 0.004[a]	0.038 ± 0.005[a]	0.037 ± 0.004[a]	0.035 ± 0.003[a]
Serum insulin prior to Std. lunch (nmol L^{-1})[a]	0.033 ± 0.004[a]	0.036 ± 0.005[a]	0.037 ± 0.005[a]	0.034 ± 0.003[a]
Satiety, fasting (mm)[b]	17.29 ± 2.99[a]	19.36 ± 3.69[a]	19.57 ± 3.97[a]	20.36 ± 3.86[a]
Hunger, fasting (mm)[b]	75.57 ± 3.84[a]	70.14 ± 5.29[a]	65.14 ± 5.72[a]	69.93 ± 5.53[a]
Desire to eat, fasting (mm)[b]	77.43 ± 4.25[a]	67.71 ± 5.8[a]	69.79 ± 4.52[a]	71.79 ± 4.59[a]

Different superscript letters indicate statistically significant differences between values in the same row, $p < 0.05$ (ANOVA, followed by Tukey's test). BG2, 30 g of glucose plus 2 g of beta-glucan; BG3, 30 g of glucose plus 3 g of beta-glucan; BG4, 30 g of glucose plus 4 g of beta-glucan; and Ref, 30 g of glucose. [a] Values are reported as means ± SEM, with a sample size of $n = 19$. [b] Values are reported as means ± SEM, with a sample size of $n = 14$.

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Table 4 Blood glucose and serum insulin

Test variable	Ref	BG2	%Δ	BG3	%Δ	BG4	%Δ
Blood glucose							
iPeak (mmol L^{-1})	3.24 ± 0.21^{a}	2.69 ± 0.201^{b}	−16.91	2.79 ± 0.21^{ab}	−13.66	2.32 ± 0.16^{b}	−28.29
iAUC 0–60 min (mmol min L^{-1})	109.2 ± 10.1^{a}	89.2 ± 9.7^{ab}	−18.32	93.9 ± 10.1^{ab}	−14.01	78.1 ± 6.2^{b}	−28.48
iAUC 0–120 min (mmol min L^{-1})	139.3 ± 18.1^{a}	126.1 ± 19.2^{a}	−9.48	144 ± 14.9^{a}	3.37	116.6 ± 11.7^{a}	−16.29
iAUC 210–330 min (mmol min L^{-1})	192.4 ± 20.6^{a}	185.3 ± 20.0^{ab}	−3.69	158.8 ± 15.4^{ab}	−17.46	145.6 ± 17.1^{b}	−24.32
iAUC 0–330 min (mmol min L^{-1})	290.4 ± 25.9^{a}	278.6 ± 33.8^{a}	−4.06	282.7 ± 31.5^{a}	−2.65	244.1 ± 24.4^{a}	−15.94
Serum insulin							
iPeak (nmol L^{-1})	0.21 ± 0.031^{a}	0.16 ± 0.027^{ab}	−24.36	0.15 ± 0.019^{ab}	−31.17	0.11 ± 0.012^{b}	−46.97
iAUC 0–60 (nmol min L^{-1})	7.46 ± 1.19^{a}	5.82 ± 0.99^{ab}	−22.05	5.32 ± 0.75^{bc}	−28.65	3.92 ± 0.49^{c}	−47.40
iAUC 0–120 (nmol min L^{-1})	9.86 ± 1.64^{a}	7.29 ± 1.15^{b}	−26.06	7.61 ± 1.24^{ab}	−22.82	5.45 ± 0.78^{b}	−44.77
iAUC 210–330 (nmol min L^{-1})	20.48 ± 2.55^{a}	18.3 ± 1.88^{ab}	−10.64	16.28 ± 1.49^{b}	−20.51	15.62 ± 1.51^{b}	−23.74
iAUC 0–330 (nmol min L^{-1})	29.94 ± 3.72^{a}	24.19 ± 2.72^{b}	−19.20	23.45 ± 2.69^{b}	−21.68	20.09 ± 2.15^{b}	−32.90

Values are reported as means ± SEM, with a sample size of n = 19. Different superscript letters indicate statistically significant differences between values in the same row, $p < 0.05$ (ANOVA, followed by Tukey's test). BG2, 30 g of glucose plus 2 g of beta-glucan; BG3, 30 g of glucose plus 3 g of beta-glucan; BG4, 30 g of glucose plus 4 g of beta-glucan; and Ref, 30 g of glucose.

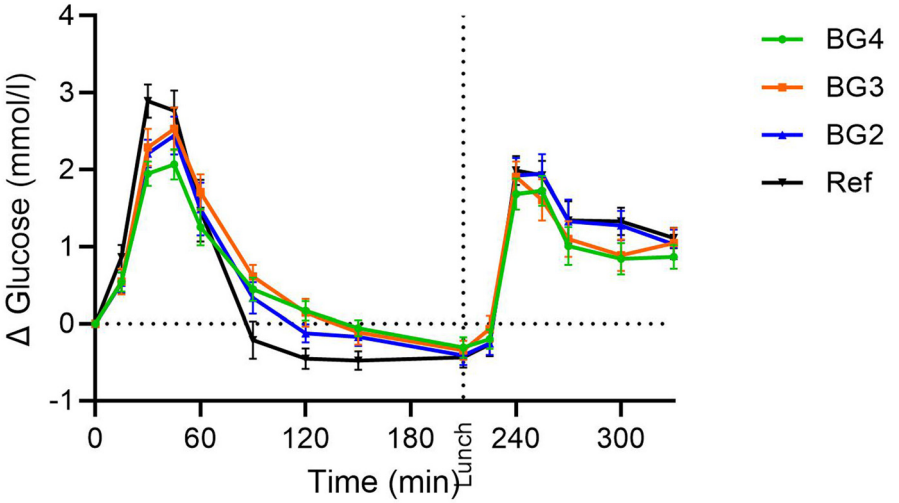

Fig. 2 Incremental changes in blood glucose concentrations after consuming the breakfast test drinks and the subsequent standardised lunch meal. Values are means ± SEM and n = 19 healthy subjects. BG2, 30 g of glucose plus 2 g of beta-glucan; BG3, 30 g of glucose plus 3 g of beta-glucan; BG4, 30 g of glucose plus 4 g of beta-glucan; and Ref, 30 g of glucose.

standardised lunch are shown in Fig. 4 and 5 and summarized in Table 4. The insulin iPeak was significantly reduced following the consumption of the BG4 breakfast drinks compared to the Ref breakfast drink. The iAUC 0–60 min following the consumption of the BG3 and BG4 breakfast drinks was significantly lower compared to the following consumption of the Ref breakfast drink ($P < 0.01$). The iAUC 0–120 min following the consumption of the BG2 and BG4 breakfast drinks was significantly reduced compared to that following the consumption of the Ref breakfast drink. After consumption of the standardised lunch (between 210 and 330 min), the iAUC was significantly reduced with the BG3 and BG4 breakfast drinks compared to the Ref breakfast drink ($P < 0.05$, Table 4 and Fig. 4). The iAUC during the course of the entire investigation (*i.e.*, iAUC 0–330 min) was significantly lower after consuming the BG2, BG3, and BG4 breakfast drinks compared to after consuming the Ref breakfast drink ($P < 0.05$).

Subjective appetite rating

Hunger. Fig. 6 shows the subjective scores for hunger following the consumption of the breakfast test drinks and the subsequent standardised lunch. During the entire period of investigation (0–330 min), the hunger AUC was significantly lower for BG4 compared to Ref ($P < 0.05$). In addition, the 0–210 min hunger AUC was significantly lower following the intake of each BG containing test drink compared to the Ref breakfast drink ($P < 0.01$). No significant differences were observed between the test conditions in hunger in the 210–330 min period following the consumption of the standardised lunch meal.

Satiety. Fig. 7 shows the subjective scores for satiety following consumption of the breakfast test drinks and the subsequent standardised lunch. The BG4 breakfast drink resulted in a greater subjective feeling of satiety compared to the Ref

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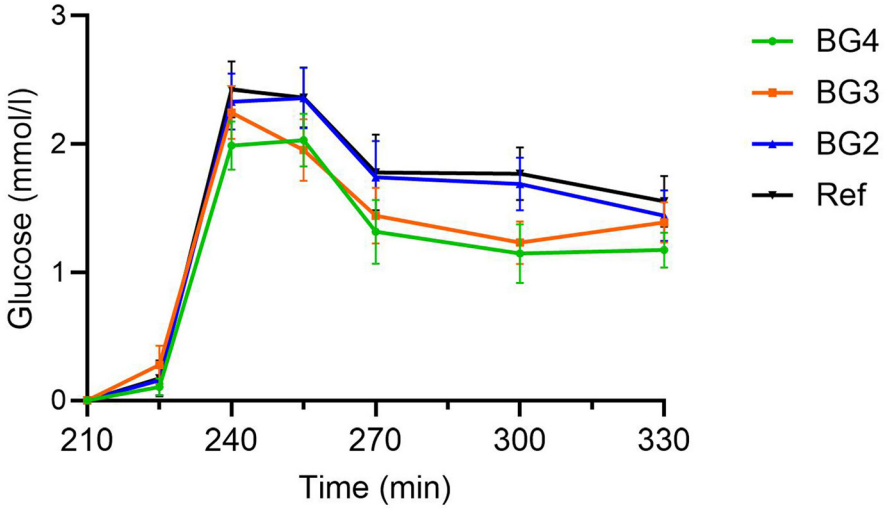

Fig. 3 Incremental changes in blood glucose concentrations after consuming the standardised lunch meal. Values are means ± SEM and n = 19 healthy subjects. BG2, 30 g of glucose plus 2 g of beta-glucan; BG3, 30 g of glucose plus 3 g of beta-glucan; BG4, 30 g of glucose plus 4 g of beta-glucan; and Ref, 30 g of glucose.

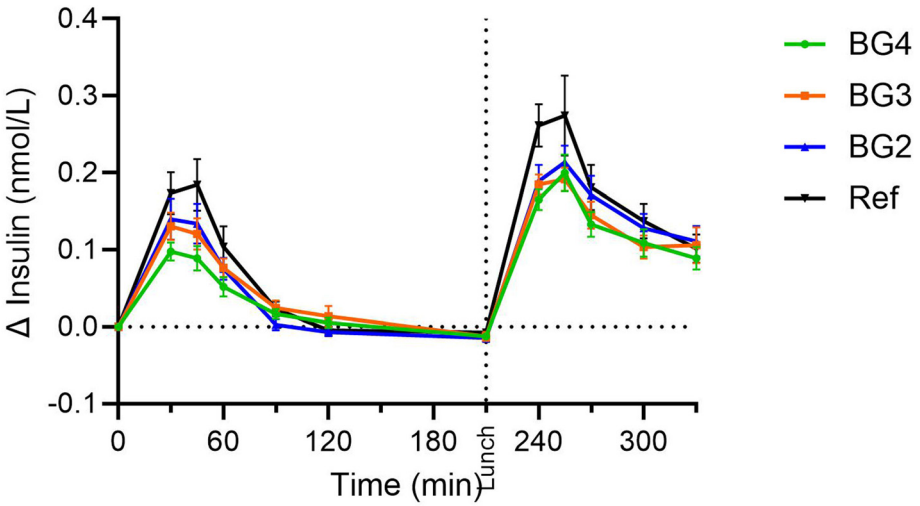

Fig. 4 Incremental changes in serum insulin concentrations after consuming the breakfast test drinks and the subsequent standardised lunch meal. Values are means ± SEM and n = 19 healthy subjects. BG2, glucose plus 2 g of beta-glucan; BG3, glucose plus 3 g of beta-glucan; BG4, glucose plus 4 g of beta-glucan; and Ref, glucose solution.

breakfast drink during the entire period of investigation (0–330 min, $P < 0.01$). The 0–210 min AUC for satiety was significantly greater with the BG3 ($P < 0.01$) and BG4 ($P < 0.001$) breakfast drinks compared to the Ref breakfast drink (Fig. 7 and Table 5). No significant differences were observed between the test conditions in the satiety AUC in the 210–330 min period following the consumption of the standardised lunch meal.

Desire to eat

Fig. 8 shows the subjective score for desire to eat following consumption of the breakfast test drinks and the subsequent standardised lunch. During the entire period of investigation (0–330 min) and during 0–210 min, the desire to eat AUC after consumption of each OBG breakfast drink was significantly lower compared to that after consumption of the Ref breakfast drink ($P < 0.05$). No significant differences were observed between the test conditions in the desire to eat AUC in the 210–330 min following the consumption of the standardised lunch meal ($P > 0.05$).

Discussion

The present study investigated the impact of OBGs on postprandial glycaemic responses acutely and following a standar-

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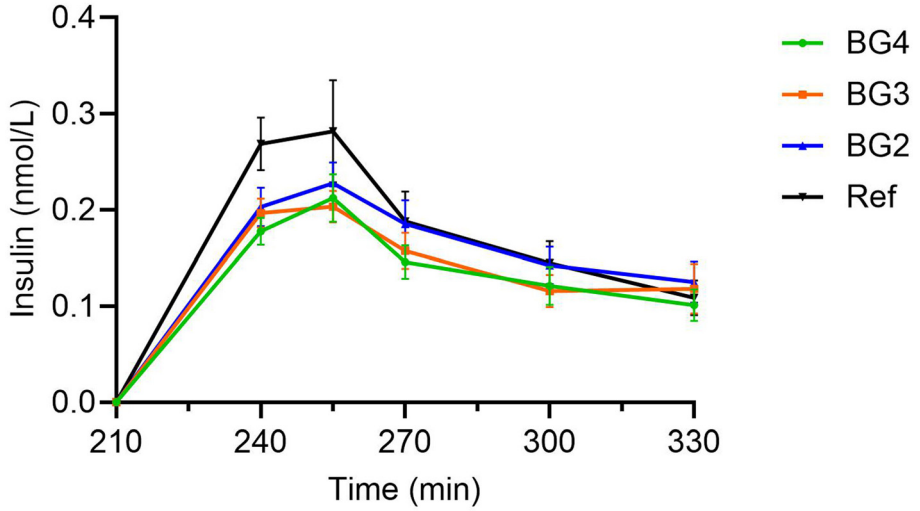

Fig. 5 Incremental changes in serum insulin concentrations after consuming the standardised lunch meal. Values are means ± SEM and *n* = 19 healthy subjects. BG2, 30 g of glucose plus 2 g of beta-glucan; BG3, 30 g of glucose plus 3 g of beta-glucan; BG4, 30 g of glucose plus 4 g of beta-glucan; and Ref, 30 g of glucose.

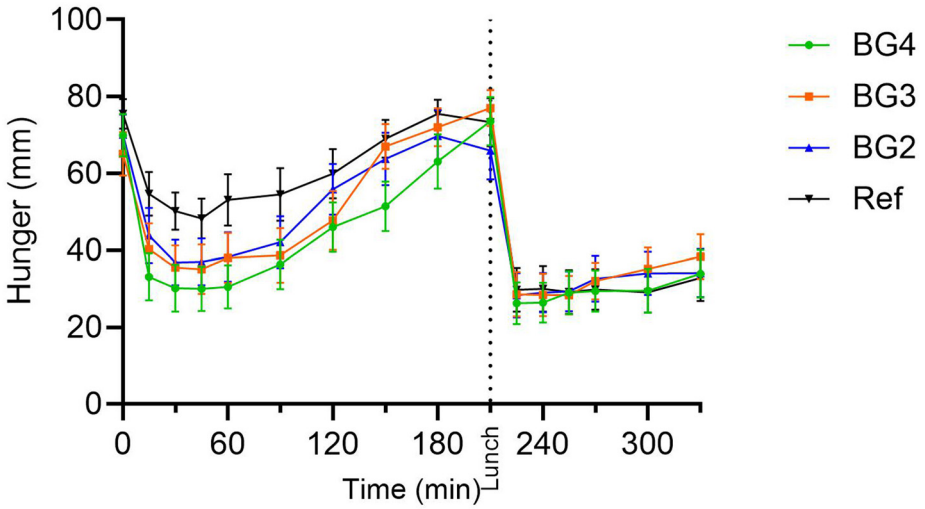

Fig. 6 Changes in the hunger rating score after consuming the breakfast test drinks and the subsequent standardised lunch meal. Values are means ± SEM and *n* = 14 healthy subjects. BG2, 30 g of glucose plus 2 g of beta-glucan; BG3, 30 g of glucose plus 3 g of beta-glucan; BG4, 30 g of glucose plus 4 g of beta-glucan; and Ref, glucose.

dised lunch meal in healthy young adults. One of the objectives was to evaluate whether an OBG dose of less than 4 g of OBGs per 30 g of avCHO improves postprandial glucose responses. Following the intake of 4 g of OBGs per 30 g of avCHO, there were several significant improvements relative to the 30 g of glucose Ref, including in the blood glucose peak, 0–60 min iAUC, and 210–330 min iAUC as well as in the insulin peak and 0–60 min, 0–120 min, 210–330 min, and 0–330 min insulin iAUCs. With doses of OBGs less than 4 g per 30 g of avCHO, the results were also promising. Indeed, with 3 g of OBGs per 30 g of avCHO, there were significant reductions relative to the 30 g of glucose Ref in insulin iAUC 0–60 min, 210–330 min, and 0–330 min, with a near-significant reduction in the glucose iPeak (p = 0.09). With 2 g of OBGs per 30 g of avCHO, there were significant reductions relative to the 30 g of glucose Ref in glucose iPeak as well as insulin iAUC 0–120 min and 0–330 min. These results thus indicate the potential postprandial blood glucose reducing effect of a dose of OBGs less than the 4 g per 30 g of avCHO stipulated by the EFSA in their evaluation of the health claim.[8]

The acute postprandial effect of OBGs on the glucose response was most significant in the early postprandial period (iAUC 0–60 min). Thereafter, no significant effects on blood glucose responses were detected following the consumption of 4 g of OBGs when the iAUC was calculated based on a 2 h postprandial period. The modulated postprandial blood glucose

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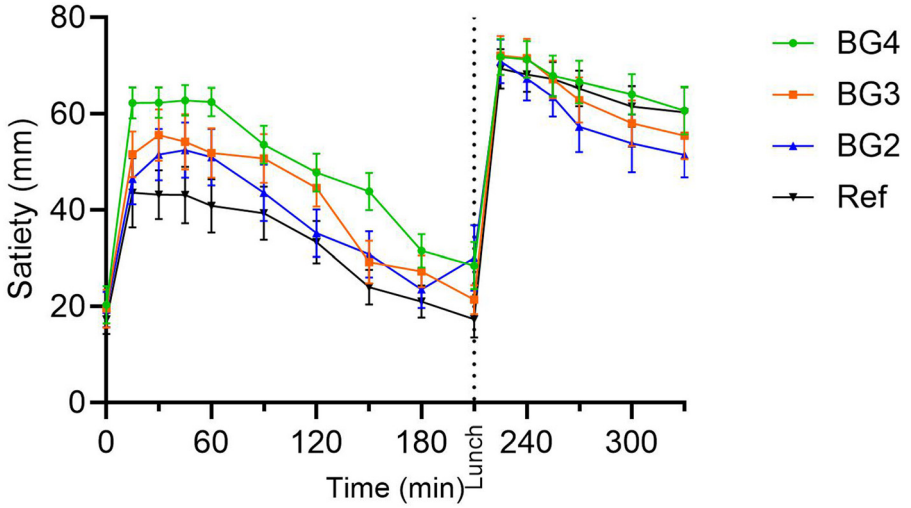

Fig. 7 Changes in the satiety rating score after consuming the breakfast test drinks and the subsequent standardised lunch meal. Values are means ± SEM and n = 14 healthy subjects. BG2, 30 g of glucose plus 2 g of beta-glucan; BG3, 30 g of glucose plus 3 g of beta-glucan; BG4, 30 g of glucose plus 4 g of beta-glucan; and Ref, 30 g of glucose.

Table 5 Appetite rating score after breakfast and standardised lunch

Test variable	Ref	BG2	%Δ	BG3	%Δ	BG4	%Δ
Satiety, AUC 0–210 min (mm min)	6793 ± 834^{a}	8000 ± 905^{ab}	17.76	8617 ± 660^{bc}	26.85	10 109 ± 485^{c}	48.81
Satiety, AUC 210–330 min (mm min)	7423 ± 420^{a}	6933 ± 503^{a}	−6.60	7313 ± 456^{a}	−1.48	7712 ± 431^{a}	3.89
Satiety, AUC 0–330 min (mm min)	14 215 ± 1107^{a}	14 930 ± 1167^{a}	5.03	15 930 ± 987^{ab}	12.06	17 821 ± 713^{b}	25.37
Hunger, AUC 0–210 min (mm min)	12 933 ± 912^{a}	11 082 ± 1148^{b}	−14.31	10 931 ± 1062^{b}	−15.48	9592 ± 1140^{b}	−25.83
Hunger, AUC 210–330 min (mm min)	3923 ± 590^{a}	4062 ± 611^{a}	3.54	4213 ± 552^{a}	7.39	3838 ± 622^{a}	−2.17
Hunger, AUC 0–330 min (mm min)	16 856 ± 1347^{a}	15 144 ± 1646^{ab}	−10.16	15 144 ± 1468^{ab}	−10.16	13 429 ± 1661^{b}	−20.33
Desire to eat, AUC 0–210 min (mm min)	13 713 ± 823^{a}	11 277 ± 1157^{b}	−17.76	11 256 ± 980^{b}	−17.92	9436 ± 1065^{b}	−31.19
Desire to eat, AUC 210–330 min (mm min)	4463 ± 508^{a}	4099 ± 629^{a}	−8.16	3944 ± 572^{a}	−11.63	3880 ± 590^{a}	−13.06
Desire to eat, AUC 0–330 min (mm min)	18 175 ± 1182^{a}	15 370 ± 1667^{b}	−15.43	15 199 ± 1450^{b}	−16.37	13 316 ± 1548^{b}	−26.73

Values are reported as means ± SEM, with a sample size of n = 14. Different superscript letters indicate statistically significant differences between values in the same row, $p < 0.05$ (ANOVA, followed by Tukey's test). BG2, 30 g of glucose plus 2 g of beta-glucan; BG3, 30 g of glucose plus 3 g of beta-glucan; BG4, 30 g of glucose plus 4 g of beta-glucan; and Ref, 30 g of glucose.

excursion observed after OBG intake, *i.e.* low iPeak but prolonged net increment of blood glucose concentrations above the fasting values, is probably related to an increased viscosity of the chyme, which decreases the gastric emptying rate and the absorption of glucose in the small intestine.[13–15] Such appearance of the postprandial blood glucose profile has been related to health benefits, both with respect to the lower blood glucose peak, and also due to the prolonged net increment of glucose, which potentially may increase insulin sensitivity at the following meal.[16] It is noteworthy that the insulin response was significantly reduced after all doses of OBGs during the entire test period (iAUC 0–330 min), compared with the Ref, which also constitutes an important metabolic benefit.

Our results extend the current knowledge regarding improved glycaemic response after intake of 4 g of OBGs, by showing that this quantity of OBGs also has the potential to improve the glycaemic response after the forthcoming meal, *i.e.* OBGs may induce a so-called "second meal effect" on glucose tolerance. Maintaining tightly controlled blood glucose concentrations over a prolonged period is vital, as it can lead to a decreased risk of obesity, type 2 diabetes, and cardiovascular diseases.[17,18]

An additional important property of OBGs observed in the current study is their potential to enhance appetite regulation towards a more satiating sensation and less postprandial hunger, which may be used as a tool for the control of food intake for reducing the risk of obesity. The more pronounced and rapid decline in blood glucose following the consumption of the reference drink induced transient postprandial hypoglycemia, a condition known to stimulate the release of appetite-regulating hormones such as neuropeptide Y.[19] This mechanism may partly explain the differences observed in appetite sensations between the reference and OBG-containing test drinks. Notably, previous research has reported a dose-dependent increase in circulating PYY levels following OBG intake, suggesting the potential role of OBGs in promoting satiety.[20]

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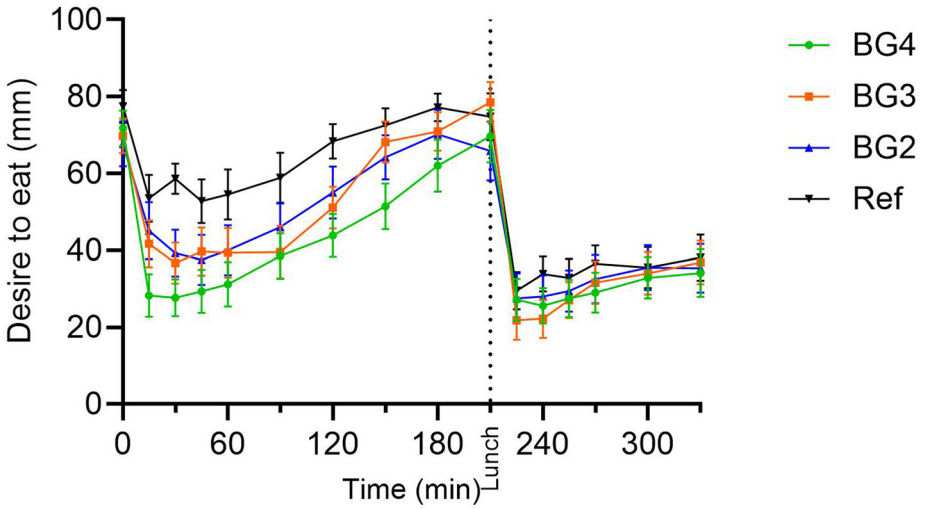

Fig. 8 Changes in the desire to eat rating score after consuming the breakfast test drinks and the subsequent standardised lunch meal. Values are means ± SEM and n = 14 healthy subjects. BG2, 30 g of glucose plus 2 g of beta-glucan; BG3, 30 g of glucose plus 3 g of beta-glucan; BG4, 30 g of glucose plus 4 g of beta-glucan; and Ref, 30 g of glucose.

The sustained glycaemic response observed with OBGs may thus contribute to appetite regulation by reducing the magnitude of glucose–insulin fluctuations that are known to drive hunger and subsequent food intake. This observation is in agreement with previous reports describing similar effects on appetite traits after OBG consumption.[21,22] Our study adds to the current knowledge by demonstrating a prolonged effect of OBGs on appetite sensations, *i.e.* OBGs have the potential to improve appetite sensations also after a forthcoming meal.

The present study aligns with the current evidence suggesting the incorporation of OBGs into carbohydrate-rich meals as a means to reduce glycaemic and insulinaemic responses in the acute postprandial period.[2] The magnitude of glycaemia reduction observed is consistent with the dose-dependent manner outlined in previous systematic reviews and meta-analyses.[2,9,23] The molecular weight (MW) of OBGs is a significant modifier of its postprandial glycemia-lowering efficacy, with MW exceeding 300 kDa being effective.[24] The MW of the OBG preparation used herein was 800 kDa. According to a recent meta-analysis,[9] a 2.2 g dose of OBGs in the 300–1000 kDa range should result in a reduced acute blood glucose response. Our results indicate that the reduction of acute glucose iPeak was possible even with a lower dose of OBGs (2 g).

The glycaemic response reduction by OBGs is not only dependent on the dose and molecular weight of the preparation tested, but it is also influenced by the type and nature of the food matrix ingested.[21,25] It has been reported that incorporation of BGs in a solid matrix modulates the glycaemic response more effectively than when it is present in a liquid matrix.[25] Perhaps the liquid matrix in the present study was one factor contributing to the lack of statistical difference between all-test drinks and Ref in glucose iAUC during 0–120 min.

Data regarding the metabolic effects of OBGs on a second meal consumed after approximately 3–4 h are scarce. It is noteworthy that the few studies looking at the impact of BGs on the second meal glycaemic tolerance have investigated the effects of BGs when they are included in a matrix of whole grain or refined barley flour. Matsuoka *et al.* found that the second meal glycaemic tolerance was significantly improved 4 h after consuming refined barley flour bread containing 2.5 g of BBGs compared to a refined wheat flour bread, in healthy Japanese adults.[26] In addition, Fukuhara *et al.* found that when white rice was combined with refined barley kernels for the first meal, the postprandial blood glucose concentration was lower after both the first and second meals in an intervention including eighteen healthy Japanese participants.[27] Nilsson *et al.* reported that choosing a specific low-glycaemic index whole-grain cereal (rye or barley kernels) meal has the potential to enhance glucose tolerance throughout subsequent meals over the span of a whole day.[10]

The mechanisms underlying the second meal effect on glucose tolerance in the 5.5 h perspective are not fully understood but, as discussed above, an improved insulin sensitivity due to a more favourable postprandial glucose excursion may be partly involved.[28] Furthermore, it is possible that parts of the OBGs have entered the colon within this time frame, providing a substrate for bacterial fermentation. As other soluble components of dietary fibre, OBGs are metabolized by the gut bacteria into short chain fatty acids, which have been shown to stimulate the secretion of gut hormones, *e.g.* peptide YY (PYY) and glucagon-like peptide-1 (GLP-1), hormones that play an important role in glycaemic and appetite regulation.[29,30]

The limitations of our study include the relatively small sample size for appetite variables and the short-term nature of the interventions. Moreover, it would be interesting to investigate additional variables that could provide deeper insights

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into the mechanisms behind the observed effects. It would thus be fruitful to examine variables such as the gut hormones GLP-1 and PYY. Further research with larger cohorts and longer intervention periods is imperative to substantiate the long-term benefits of OBGs on glycaemic control and appetite regulation, and to elucidate the underlying mechanisms driving the observed effects.

Conclusion

Our study demonstrates that the intake of 4 g of OBGs per 30 g of avCHO led to improved postprandial glucose tolerance after a second meal. Notably, we observed a significant reduction in acute postprandial glucose peaks with 2 g of OBGs, as well as several significant improvements in the insulin iAUC with 2 or 3 g of OBGs per 30 g of avCHO, suggesting the effectiveness of OBGs at lower doses than the current EFSA recommendation of 4 g per 30 g of avCHO. Additionally, OBGs showed potential to enhance satiety and reduce postprandial hunger, which may be important tools for controlling food intake, thereby contributing to reduced risks of obesity, type 2 diabetes, and cardiovascular diseases.

Institutional review board statement

The ethical approval of the study was given by the Regional Ethical Review Board in Lund, Sweden (Dnr. 2018/658) and the study was conducted according to the guidelines laid down in the Declaration of Helsinki. The study was registered at ClinicalTrials.gov (NCT05801653).

Informed consent statement

Informed consent was obtained from all subjects involved in the study.

Author contributions

Conceptualization: M. M. H., J. T., L. C. and A. N.; experiments and formal analysis: M. M. H, M. V. G, and C. V.; visualization: M. M. H.; draft preparation: M. M. H.; and finalizing the manuscript: M. M. H., J. T., L. C. and A. N. All authors have read and agreed to the published version of the manuscript.

Data availability

The datasets analyzed during this study are available from the corresponding author on reasonable request.

Conflicts of interest

The authors declare no conflicts of interest.

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References

1 J. Chen and K. Raymond, Beta-glucans in the treatment of diabetes and associated cardiovascular risks, *Vasc. Health Risk Manage.*, 2008, **4**, 1265–1272, DOI: **10.2147/vhrm.s3803**.

2 S. M. Tosh, Review of human studies investigating the post-prandial blood-glucose lowering ability of oat and barley food products, *Eur. J. Clin. Nutr.*, 2013, **67**, 310–317, DOI: **10.1038/ejcn.2013.25**.

3 L. Cloetens, M. Ulmius, A. Johansson-Persson, B. Akesson and G. Onning, Role of dietary beta-glucans in the prevention of the metabolic syndrome, *Nutr. Rev.*, 2012, **70**, 444–458, DOI: **10.1111/j.1753-4887.2012.00494.x**.

4 G. Livesey, R. Taylor, H. F. Livesey, A. E. Buyken, D. J. A. Jenkins, L. S. A. Augustin, J. L. Sievenpiper, A. W. Barclay, S. Liu, T. M. S. Wolever, *et al.*, Dietary Glycemic Index and Load and the Risk of Type 2 Diabetes: A Systematic Review and Updated Meta-Analyses of Prospective Cohort Studies, *Nutrients*, 2019, **11**, 1280, DOI: **10.3390/nu11061280**.

5 G. Livesey and H. Livesey, Coronary Heart Disease and Dietary Carbohydrate, Glycemic Index, and Glycemic Load: Dose-Response Meta-analyses of Prospective Cohort Studies, *Mayo Clin. Proc. Innov. Qual. Outcomes*, 2019, **3**, 52–69, DOI: **10.1016/j.mayocpiqo.2018.12.007**.

6 D. J. Jenkins, T. M. Wolever, A. R. Leeds, M. A. Gassull, P. Haisman, J. Dilawari, D. V. Goff, G. L. Metz and K. G. Alberti, Dietary fibres, fibre analogues, and glucose tolerance: importance of viscosity, *Br. Med. J.*, 1978, **1**, 1392, DOI: **10.1136/bmj.1.6124.1392**.

7 R. E. Steinert, D. Raederstorff and T. M. S. Wolever, Effect of Consuming Oat Bran Mixed in Water before a Meal on Glycemic Responses in Healthy Humans—A Pilot Study, *Nutrients*, 2016, **8**, 524.

8 Efsa Panel on Dietetic Products, Carlo Agostoni, Jean-Louis Bresson, Susan Fairweather-Tait, Albert Flynn, Ines Golly, Hannu Korhonen, Pagona Lagiou, Martinus Løvik, Rosangela Marchelli, Ambroise Martin, Bevan Moseley, Monika Neuhäuser-Berthold, Hildegard Przyrembel, Seppo Salminen, Yolanda Sanz, Sean (J.J.) Strain, Stephan Strobel, Inge Tetens, Daniel Tomé, Hendrik van Loveren and Hans Verhagen, Scientific Opinion on the substantiation of health claims related to beta-glucans 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

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function" (ID 850) pursuant to Article 13(1) of Regulation (EC) No 1924/2006, *EFSA J.*, 2011, **9**, 2207, DOI: 10.2903/j.efsa.2011.2207.

9 J. C. Noronha, A. Zurbau and T. M. S. Wolever, 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**, 308–315, DOI: 10.1038/s41430-022-01176-5.

10 A. C. Nilsson, E. M. Oöstman, Y. Granfeldt and I. M. E. Björck, Effect of cereal test breakfasts differing in glycemic index and content of indigestible carbohydrates on daylong glucose tolerance in healthy subjects2, *Am. J. Clin. Nutr.*, 2008, **87**, 645–654, DOI: 10.1093/ajcn/87.3.645.

11 K. Mio, M. Togo-Ohno, N. Tadenuma, R. Ogawa, C. Yamanaka and S. Aoe, A single administration of barley β-glucan and arabinoxylan extracts reduces blood glucose levels at the second meal via intestinal fermentation, *Biosci., Biotechnol., Biochem.*, 2023, **87**, 99–107, DOI: 10.1093/bbb/zbac171.

12 M. M. Hossain, J. Tovar, L. Cloetens, M. T. S. Florido, K. Petersson, F. Prothon and A. Nilsson, Oat Polar Lipids Improve Cardiometabolic-Related Markers after Breakfast and a Subsequent Standardized Lunch: A Randomized Crossover Study in Healthy Young Adults, *Nutrients*, 2021, **13**, 988.

13 C. A. Edwards, I. T. Johnson and N. W. Read, Do viscous polysaccharides slow absorption by inhibiting diffusion or convection?, *Eur. J. Clin. Nutr.*, 1988, **42**, 307–312.

14 H. D. Goff, N. Repin, H. Fabek, D. El Khoury and M. J. Gidley, Dietary fibre for glycaemia control: Towards a mechanistic understanding, *Bioact. Carbohydr. Diet. Fibre*, 2018, **14**, 39–53, DOI: 10.1016/j.bcdf.2017.07.005.

15 P. Battilana, K. Ornstein, K. Minehira, J. M. Schwarz, K. Acheson, P. Schneiter, J. Burri, E. Jéquier and L. Tappy, Mechanisms of action of β-glucan in postprandial glucose metabolism in healthy men, *Eur. J. Clin. Nutr.*, 2001, **55**, 327–333, DOI: 10.1038/sj.ejcn.1601160.

16 T. M. S. Wolever, A. Bentum-Williams and D. J. A. Jenkins, Physiological modulation of plasma free fatty acid concentrations by diet. Metabolic implications in nondiabetic subjects, *Diabetes Care*, 1995, **18**, 962–970.

17 A. V. Poznyak, L. Litvinova, P. Poggio, V. N. Sukhorukov and A. N. Orekhov, Effect of Glucose Levels on Cardiovascular Risk, *Cells*, 2022, **11**, 3034, DOI: 10.3390/cells11193034.

18 C. T. Rentsch, V. Garfield, R. Mathur, S. V. Eastwood, L. Smeeth, N. Chaturvedi and K. Bhaskaran, Sex-specific risks for cardiovascular disease across the glycaemic spectrum: a population-based cohort study using the UK Biobank, *Lancet Reg. Health Eur.*, 2023, **32**, 100693, DOI: 10.1016/j.lanepe.2023.100693.

19 K. Wynne, S. Stanley, B. McGowan and S. Bloom, Appetite control, *J. Endocrinol.*, 2005, **184**, 291–318, DOI: 10.1677/joe.1.05866.

20 E. J. Beck, L. C. Tapsell, M. J. Batterham, S. M. Tosh and X. F. Huang, Increases in peptide Y-Y levels following oat beta-glucan ingestion are dose-dependent in overweight adults, *Nutr. Res.*, 2009, **29**, 705–709, DOI: 10.1016/j.nutres.2009.09.012.

21 S. Pentikäinen, L. Karhunen, L. Flander, K. Katina, A. Meynier, P. Aymard, S. Vinoy and K. Poutanen, Enrichment of biscuits and juice with oat β-glucan enhances postprandial satiety, *Appetite*, 2014, **75**, 150–156, DOI: 10.1016/j.appet.2014.01.002.

22 M. Redondo-Puente, R. Mateos, M. A. Seguido, J. García-Cordero, S. González, R. M. Tarradas, L. Bravo-Clemente and B. Sarriá, Appetite and Satiety Effects of the Acute and Regular Consumption of Green Coffee Phenols and Green Coffee Phenol/Oat β-Glucan Nutraceuticals in Subjects with Overweight and Obesity, *Foods*, 2021, **10**, 2511.

23 T. M. S. Wolever, O. Mattila, N. Rosa-Sibakov, S. M. Tosh, A. L. Jenkins, A. Ezatagha, R. Duss and R. E. Steinert, Effect of Varying Molecular Weight of Oat β-Glucan Taken just before Eating on Postprandial Glycemic Response in Healthy Humans, *Nutrients*, 2020, **12**, 2275.

24 A. Zurbau, J. C. Noronha, T. A. Khan, J. L. Sievenpiper and T. M. S. Wolever, The effect of oat β-glucan on postprandial blood glucose and insulin responses: a systematic review and meta-analysis, *Eur. J. Clin. Nutr.*, 2021, **75**, 1540–1554, DOI: 10.1038/s41430-021-00875-9.

25 J. Paquin, A. Bédard, S. Lemieux, S. Tajchakavit and S. L. Turgeon, Effects of juices enriched with xanthan and β-glucan on the glycemic response and satiety of healthy men, *Appl. Physiol., Nutr., Metab.*, 2013, **38**, 410–414, DOI: 10.1139/apnm-2012-0207%M23713534.

26 T. Matsuoka, A. Tsuchida, A. Yamaji, C. Kurosawa, M. Shinohara, I. Takayama, H. Nakagomi, K. Izumi, Y. Ichikawa, N. Hariya, *et al.*, Consumption of a meal containing refined barley flour bread is associated with a lower postprandial blood glucose concentration after a second meal compared with one containing refined wheat flour bread in healthy Japanese: A randomized control trial, *Nutrition*, 2020, **72**, 110637, DOI: 10.1016/j.nut.2019.110637.

27 I. Fukuhara, T. Ikenaga, H. Noguchi, C. Kohashi, N. Kohda, S. Tomita, S. Konagai and K. Zembutsu, Effect of cooked rice with β-glucan enriched barley on postprandial glucose response and its second-meal effect, *Jpn. Pharmacol. Ther.*, 2013, **41**, 789–795.

28 M. O. Weickert and A. F. H. Pfeiffer, Impact of Dietary Fiber Consumption on Insulin Resistance and the Prevention of Type 2 Diabetes, *J. Nutr.*, 2018, **148**, 7–12, DOI: 10.1093/jn/nxx008.

29 C. B. Christiansen, M. B. N. Gabe, B. Svendsen, L. O. Dragsted, M. M. Rosenkilde and J. J. Holst, The impact of short-chain fatty acids on GLP-1 and PYY secretion from the isolated perfused rat colon, *Am. J. Physiol.: Gastrointest. Liver Physiol.*, 2018, **315**, G53–G65, DOI: 10.1152/ajpgi.00346.2017.

30 P. Larraufie, C. Martin-Gallausiaux, N. Lapaque, J. Dore, F. M. Gribble, F. Reimann and H. M. Blottiere, SCFAs strongly stimulate PYY production in human enteroendocrine cells, *Sci. Rep.*, 2018, **8**, 74, DOI: 10.1038/s41598-017-18259-0.