Effects of oat β-glucan consumption at breakfast on *ad libitum* eating, appetite, glycemia, insulinemia and GLP-1 concentrations in healthy subjects

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Author Contribution:

All authors conceived and designed the study; SMMZ collected the data; SMMZ and RES analysed and interpreted the data, drafted and revised the manuscript; and all authors read and approved the final version of the manuscript.

Abstract

There is evidence that oat β-glucan lowers appetite and *ad libitum* eating; however, not all studies are consistent, and the underpinning mechanisms are not entirely understood. We investigated the effects of 4 g high molecular weight (MW) oat β-glucan on *ad libitum* eating, subjective appetite, glycemia, insulinemia and plasma GLP-1 responses in 33 normal-weight subjects (22 female/11 male, mean age (y): 26.9 ± 1.0, BMI (kg/m^2): 23.5 ± 0.4). The study followed a randomised double-blind, cross-over design with subjects fed two test breakfasts with and without oat β-glucan followed by an *ad libitum* test meal on two different days. Blood samples and ratings for subjective appetite were collected postprandially at regular time intervals. Oat β-glucan increased feelings of fullness (p=0.048) and satiety (p=0.034), but did not affect energy and amount eaten at the *ad libitum* test meal. There was a treatment by time interaction for plasma GLP-1, plasma insulin and blood glucose. GLP-1 was significantly reduced at 90 min (p=0.021), blood glucose at 30 min (p=0.008) and plasma insulin at 30 and 60 min (p=0.002 and 0.017, respectively) following the oat β-glucan breakfast when compared with the control breakfast. Four grams of high MW oat β-glucan lowers appetite but not *ad libitum* eating and beneficially modulates postprandial glycaemia, it does however, not increase plasma GLP-1 secretion

Keywords: oat β-glucan, energy intake, satiety, GLP-1, glucose, insulin

Introduction

Obesity is a worldwide epidemic. For example, the proportion of adults in the United Kingdom who are either overweight or obese is around 65%, according to the most recent findings (NCD Risk Factor Collaboration 2017). Not only does obesity significantly increase the risk of Type 2 Diabetes Mellitus, it also poses challenges to the management of diabetes after diagnosis (Lin et al., 2015). To combat obesity and its comorbidities from a nutrition perspective, research has focussed on increasing the satiating power of the diet so that individuals feel full with fewer calories consumed (Astrup, 2005).

A number of studies suggest that high fibre consumption is associated with increased satiation and/or satiety (Wanders et al., 2011; Poutanen et al. 2017), lower body weight (Slavin, 2005), and improved postprandial glycemia (Yuan et al., 2014). There is evidence that increased fibre consumption not only reduces energy density of ingested food (Heaton et al., 1973; Rolls et al., 1999) but exerts a direct inhibitory effect on eating (Wanders et al., 2011; Pereira & Ludwig, 2001; Ibarra et al., 2014). The effect appears to depend on the chemical structure and the physicochemical properties of the fibre type, i.e. fibre viscosity, water-holding capacity and fermentability, rather than on total fibre intake (Wanders et al., 2011). Although the inhibitory effect varies depending on the study population, type, dose and mode of fibre administered as well as the timing of food intake assessment relative to treatment (Zaremba et al., 2017), several studies suggest that fibre viscosity is the dominant characteristic that determines the satiating effect (Clark and Slavin, 2013; Wanders et al., 2011).

Cereal oat and barley β-glucan consists of high molecular weight polysaccharides that exhibit high viscosity at low concentrations, consumption of which has been shown to effectively blunt glycaemic responses by increasing the viscosity of the contents of the upper gastrointestinal

(GI) tract (Wanders et al., 2011), hence, slowing gastric emptying and glucose absorption (Marciani et al. 2001). There is a positive non-linear relationship between molecular weight and viscosity, with the molecular weight of beta-glucan being subject to cultivar variety, growing conditions, processing and storage. The molecular weight of purified oat beta glucan is in the range of 50 – 3000 kDa (Ajithkumar et al., 2005) but is decreased by food preparation such as bread-making or further extrusion that impacts bioactivity of cereal β-glucan (Tosh et al., 2008; Tosh et al., 2010; Wang and Ellis, 2014). The glucose lowering characteristics of β-glucan from oat and barley have been approved by the European Food Safety Authority (EFSA) with a condition of use health claim that 4 g of β-glucan for each 30 g of available carbohydrate be consumed per meal to obtain the claimed effect (EFSA, 2011).

The evidence that cereal β-glucan lowers appetite and *ad libitum* eating is less conclusive, and the underpinning mechanisms are not entirely understood. While increased oral exposure time, stomach distention and colonic fermentation with increased production of short chain fatty acids (SCFA) may contribute to the satiating effect (Byrne et al., 2015; Wanders et al., 2013, Kristensen and Jensen, 2011), the role of GI hormones with hypothesized roles in appetite (Steinert et a., 2017) remains controversial. Some studies report postprandial reductions in ghrelin (Vitaglione et al., 2009) and increases in cholecystokinin (CCK) and peptide YY (PYY) (Vitaglione et al., 2009; Beck et al., 2009a; Beck et al., 2009b), while others found no effects on PYY (Weickert et al., 2006) and glucagon-like peptide-1 (GLP-1) (Ames et al., 2015a). Moreover, although one study suggested that PYY secretion was increased by more viscous foods (Beck et al., 2009b), another study found that PYY, CCK and GLP-1 responses were lower after a highly viscous oat bran drink compared with an identical test drink with reduced natural viscosity due to β-glucanase treatment (Juvonen et al., 2009).

In order to better understand the satiating capacity of oat β-glucan and its underpinning mechanisms, we aimed to investigate the effect of 4 g of high MW oat β-glucan incorporated into a breakfast meal on *ad libitum* eating following a 150 min intermeal interval as well as on subjective feelings of appetite, postprandial glycemia, insulinemia and plasma GLP-1, the latter because of its central role in both appetite and glycaemic control. We hypothesised that the oat β-glucan containing breakfast would increase fullness and satiety and decrease *ad libitum* eating more than the isocaloric control breakfast, and that this would be accompanied by increases in plasma GLP-1 and reductions in blood glucose and plasma insulin.

Materials and methods

Subjects

A sample size calculation was conducted for the primary outcome measure of energy intake. Comparable cross-over trials showed a decrease in energy intake at *ad libitum* lunches of between 85 to 170kcal, which varied depending on a number of factors, such as dose of ingested β-glucan, inter-meal intervals, subject characteristics, and test-meal compositions. For example, in a study by Vitaglione *et al*. (2009) a 3 g β-glucan intervention at breakfast reduced *ad libitum* lunch energy intake after 3 hours by 170 kcal; whereas Rebello *et al*. (2016a) reported a reduction of 85 kcal at an *ad libitum* lunch following 2.68 g of oat β-glucan consumption. Using an average standard deviation of 200 kcal and assuming a conservative decrease in energy intake of 100 kcal, the resulting expected effect size was 0.5. The resulting minimum sample size was estimated to be n=32-34 (one sample t-test, $\alpha = 5\%$, power of 80%: nQueryAdvisor 7.0).

Of the 43 subjects enrolled in the study, there were seven withdrawals due to participant time constraints, and these were not included in the analysis. Of the 36 subjects who completed the study, a further three subjects did not adhere to the study protocol, and therefore, were excluded from data analysis (two subjects did not consume all of the test breakfasts and one subject arrived at both study mornings with elevated fasted blood glucose). Of the remaining 33 subjects, 22 were female and 11 were male (age 26.9 ± 1.0 years; weight 68.1 ± 2.0kg; BMI 23.5 ± 0.4 kg/m^2; waist circumference 78.0 ± 1.5cm). Before inclusion in the study, potential subjects were briefed and given the opportunity to ask questions. This was followed by a health assessment, including anthropometric measurements, vital signs, and a general health questionnaire which gave details of food allergies, metabolic disease, weight changes and smoking habits. Eating behaviour was determined using the Dutch Eating Behaviour

Questionnaire (van Strein et al., 1986). Restrained eaters were not eligible for participation. Those also excluded were breakfast skippers, postmenopausal, pregnant or lactating females, smokers, dieters or those taking medications which may affect appetite. Prior to enrolment, fasted glucose and haemoglobin measurements were checked to exclude subjects with glucose impairment (>5.6 mmol/L) and/or anaemia (<120 g/L for females and <130 g/L for males). Subjects were required to be willing to allow blood collections and not have food allergies to test meal ingredients (gluten, lactose). Ethical clearance was granted by Queen Margaret University Research Ethics Committee, Edinburgh, where the research was conducted. Participants were recruited from Musselburgh, East Lothian and surrounding areas. Written informed consent was obtained from all subjects. The trial was registered on ClinicaTrials.gov with registration number NCT02637388.

Experimental design

The study followed a randomised double-blind, cross-over design with subjects fed two test breakfasts with and without oat β-glucan followed by an *ad libitum* test meal on two different days. There was at least one week between individual study sessions and subjects were required to complete both sessions within 4 weeks. Each subject was scheduled to arrive at the same time and on the same day of the week for each treatment and instructed to abstain from strenuous exercise, alcohol and coffee consumption 24 h prior to treatments. Food diaries completed 24 h before each treatment showed no differences in energy intakes the day before study sessions (1845 ± 95 kcal and 1851 ± 115 kcal prior to control and oat β-glucan breakfast, p=0.94 respectively). Each participant arrived fasted (for 10 hours) at the laboratory between 8:30am and 10:00am during weekdays.

On each occasion, an antecubital vein catheter was inserted for blood collection (for plasma insulin and GLP-1) while blood glucose was quantified using a finger-prick blood test. Only subjects with complete data sets/blood samples were included in analysis for GLP-1 and insulin. After taking a fasted blood sample, subjects consumed the test breakfast within 10 min.

The breakfast consisted of Kellogg's Rice Krispies cereal (Kellogg Company, Manchester, UK), with semi skimmed milk (1.8 % fat) and Greek-style yoghurt (Tesco Groceries, Edinburgh, UK). Four grams of high MW oat β-glucan (from 14.6 g of OatWell Original Powder, DSM Nutritional Products Ltd., Kaiseraugst, Switzerland) was split between the cereal and Greek-style yoghurt to improve palatability of the breakfast. For this, 7.3g OatWell powder was mixed with Greek-style yoghurt and 7.3g OatWell powder was mixed with dry Rice Krispies in a bowl before semi-skimmed milk (150 mL) was poured over the Rice Krispies by the subject immediately before commencing the meal. Tosh *et al.* (2010) previously determined the MW of OatWell™ oat β-glucan to be 2.213 x10^6 g mol^{-1}.

A researcher who was not involved in the study was responsible for assigning the order of the two breakfasts (with and without oat β-glucan) using a random number generator (Microsoft Excel) and supervised the subjects whilst eating. Subjects were required to finish the breakfast within 10 minutes and afterwards to rate the palatability of both breakfasts using a VAS. The breakfasts were matched for their protein, fat and carbohydrate contents: 1) in order accommodate for the energy content of the oat bran powder, the Greek-style yoghurt was reduced by 10 g in the intervention breakfast. In order to adequately match protein and CHO contents of both breakfasts, 28 mL of PROmilk50 (ready-to-drink vanilla protein milk, MyProtein, Cheshire, UK) was added to the control breakfast (**Table 1).**

After the breakfast, additional blood samples for measurement of plasma insulin and GLP-1 and blood glucose were collected at intervals of 30 min (t=0-90) and VAS were completed at intervals of 15 min (t = 0-150 min). At t = 150 min, each subject was then offered an *ad libitum* test meal and allowed to consume as much food and water as desired until reaching comfortable fullness, for a maximum of 30 min (t = 150–180 min).

The meal consisted of ham sandwiches, made from white sliced bread (approximately 40 g per slice, Hovis medium soft white, High Wycombe, UK), butter (10 g per slice, Countrylife, Surrey, UK) and sliced cooked ham (approximately 45 g per sandwich, Tesco Groceries, Edinburgh, UK). Nutritional composition of the ham sandwiches was 10.3 g of protein, 11.7 g of fat, 23.9 g of carbohydrate with 243 kcal, all per 100 g. The sandwiches were cut into four equal-sized pieces and served in excess to the subject along with water. Subjects were told to eat until they felt 'comfortably full' and to complete a food diary for the remainder of the day (i.e. from when they left the laboratory until when they stopped eating at night). Plate waste and water left over were weighed after the subject left the laboratory.

Measurements:

Appetite and food intake: Perceptions of hunger, fullness, desire to eat, satiety and prospective food consumption were measured using validated VAS (Blundell et al., 2010). Each VAS was composed of lines 100 mm in length anchored by the descriptors *not at all* to *extremely*. Food intake at the test meal was calculated from the amount of food (g) eaten at the *ad libitum* meal. Energy intake (kcal) and macronutrient composition (expressed as g and % of energy) were then calculated using Nutritics dietary assessment software (version 4.0, Nutritics Ltd., Dublin, Ireland).

Blood glucose and plasma insulin and glucagon-like peptide (GLP-1): Finger-prick blood glucose measurements were taken using a sterile lancet device (Accu-Chek Safe T Pro Plus, Roche Diagnostics, UK) and quantified by an Accu-Check Aviva glucometer (Roche Diagnostics, UK). Blood samples for total GLP-1 and insulin measurements were collected in lavender capped BD Vacutainer® plastic K2EDTA tubes (BD Diagnostics, US). The tubes were placed on ice and centrifuged at 3,000 x rpm (Thermo Scientific Heraeus Biofuge Primo R) for 15 min at 4 °C. Plasma samples were aliquoted into cryogenic eppendorf tubes and stored at −85 °C until analysis. Plasma concentrations of total GLP-1 (intra-coefficient of variation (CV): <5 %; inter-CV: <12 %; 1.5 pM sensitivity as per Millipore, CAT# EZGLP1T-36K) and insulin (intra-CV: 4.6–7.0 %; inter-CV: 9.1–11.4 %; 1 µU/mL sensitivity as per Millipore, CAT# EZHI-14K) were measured using ELISA kits (Merck, Germany). A quantitative curve fitting program for immunoassays (MasterPlex 2010), which used a 5 Parameter Logistic model equation, was used to compute standard curves and determine insulin and total GLP-1 concentrations.

Test food viscosity: A constant shear rheometer, Bohin Rheometer C-VOR 150 (Malvern Bohin Instruments), fitted with a 4^{o} /40 mm diameter cone and plate geometry, was used for all viscosity measurements. Measurements were carried out at 37^{o}C to mimic stomach temperature and at shear rates ranging from $0.5x10^{-1}$ to $1.0x10^{2}$ s^{-1}.

Data and statistical analysis

Statistical analysis was performed using SPSS software (version 23.0; Chicago, IL, USA). Normality of all data were tested using Shapiro–Wilk statistic. Differences in energy intake between the two treatments were assessed using Students paired samples t-test. Total area under the curves (AUC) for subjective appetite ratings, blood glucose and hormones were

calculated using the trapezoidal method. Subjective appetite ratings were analysed using ANCOVA with baseline values used as co-variate (Blundell et al., 2010). Time x treatment effects for blood glucose and hormones were identified using a two-factor analysis of variance (2 factor-ANOVA) with time and treatment (breakfast) as factors. *Post hoc* comparisons, adjusted for multiple comparisons by Bonferroni's correction, were performed where ANOVAs revealed significant effects to identify differences between treatments across timepoints. All tests were two tailed and significance was set at $p<0.05$. All values are presented as means ± standard error of the mean (SEM).

Results

There was no effect of treatment on *ad libitum* eating. Total intakes at the test meal were 681 ± 46 kcal and 267 ± 18 g with the breakfast containing oat β-glucan and 704 ± 51 kcal and 275 ± 20 g with the control breakfast (t(32)=0.875, p=0.388 and t(32)=0.846, p=0.404, respectively). The oat β-glucan breakfast also did not detectably affect subjects' energy intake for the remainder of the study day when compared with the control breakfast (t(31)=-1.70, p=0.099, **Table 2**). There was also no difference in water intake at the *ad libitum* meal (t(32)=-0.32, p=0.751, **Table 2**).

There was a significant effect of oat β-glucan breakfast on total AUC of satiety ratings after controlling for baseline AUC, (F[1,60]=3.07, p=0.034. Total AUC for satiety following oat β-glucan and control breakfast were 7604 ± 459 mm x min and 6516 ± 427 mm x min, respectively. There was also a significant effect on total AUC of fullness ratings after controlling for baseline AUC, (F[1,60]=2.98, p=0.048. Total AUC for fullness following oat β-glucan and control breakfast were 7563 ± 428 mm x min and 6505 ± 453 mm x min, respectively (**Figure 1**). There was no effect of oat β-glucan on hunger (p=0.133), desire to eat (p=0.098) or prospective food consumption (p=0.213).

There were no differences in baseline (fasting) values between study days for total GLP-1 (t(20)=-1.76, p=0.09, blood glucose (t(32)=0.29, p=0.771), or plasma insulin (t(20)=-1.40, p=0.176, **Figure 2**).

For plasma GLP-1, Mauchly's Test of Sphericity indicated that the assumption of sphericity had been violated, $X^2(5) = 9.59$, p=0.03, and therefore, a Greenhouse Geisser correction was used. There was a treatment x time interaction (F[2.3,45.3]=6.62, p=0.002) for GLP-1. Plasma

GLP-1 concentrations were significantly reduced at 90 min after the oat β-glucan breakfast when compared with the control breakfast (22 ± 9 pmol/L *vs*. 17 ± 9 pmol/L, t(20)=2.50, p=0.021, **Figure 2A)**. There was no significant difference for GLP-1 AUCs between treatments (t(20)=0.59, p=0.56, **Table 3**). Only subjects with complete data sets were included in the analysis for plasma GLP-1 (full data, n=21).

For blood glucose, Mauchly's Test of Sphericity indicated that the assumption of sphericity had been violated, $X^2(5) = 16.78$, p=0.005, and therefore, a Greenhouse Geisser correction was used. There was a treatment x time interaction (F[2.3,72.3]=49.13, p<0.001) for blood glucose. Blood glucose was significantly lower at 30 min after the oat β-glucan breakfast when compared with control (6.0 ± 1.0 mmol/L *vs*. 6.5 ± 0.9 mmol/L (t(32)=2.81, p=0.008, **Figure 2B)**). There was no significant difference for blood glucose AUCs between treatments (t(32)=1.21, p=0.235, **Table 3**).

For plasma insulin, Mauchly's Test of Sphericity indicated that the assumption of sphericity had been violated, $X^2(5) = 12.2$, p=0.033, and therefore a Greenhouse Geisser correction was used. There was a treatment x time interaction (F[2.08,44.1]=56.98, p<0.001) for plasma insulin. Plasma insulin was significantly lower at 30 and 60 min after the oat β-glucan breakfast when compared with control (32.4 ± 18 µU *vs*. 50.3 ± 23.1 µU, t(20)=3.63, p=0.002 and 15.8 ± 9.2 µU *vs*. 24.4 ± 18 µU, (t(20)=2.62, p=0.017, respectively, **Figure 2C**). The AUC for insulin over the 90 min period was also significantly lower following the oat β-glucan breakfast when compared with the control breakfast (t(20)=3.99, p=0.001, **Table 3).** Only subjects with complete data sets were included in the analysis for plasma insulin (full data, n=21).

The viscosity of the different components of the breakfast containing oat β-glucan was considerably greater than that of the control breakfast, with significant differences in viscosity seen at $50s^{-1}$ ($p<0.001$), a shear rate representative of gastric conditions (**Figure 3).**

A significant reduction in palatability ratings for the β-glucan breakfast compared to control breakfast was reported (36.4±4.3 mm and 72.9±3.8 mm, respectively, $p<0.001$). There were, however, no significant differences for aftertaste (61.8 ± 4.2 mm, 64.0 ± 4.5 mm, $p=0.71$) or smell (52.3 ± 3.5 mm, 59.4 ± 3.8 mm, $p=0.07$) between breakfasts.

Discussion

The evidence for oat and barley β-glucans to lower appetite and *ad libitum* eating is contradictory and the underpinning mechanisms, particularly GI satiation peptide secretion, unclear. Here we investigated the effect of high MW oat β-glucan incorporated into a breakfast meal on *ad libitum* eating, subjective appetite, plasma GLP-1 and insulin as well as blood glucose concentrations in 33 healthy subjects. Based on previous studies, we hypothesized that oat β-glucan increases fullness and/or satiety and reduces *ad libitum* eating associated with by increases in plasma GLP-1 and reductions in blood glucose and plasma insulin. We found that subjects were more satiated and fuller after consuming the oat β-glucan breakfast when compared to the control; however, in contrast to our hypothesis, this did not translate into a reduction in food intake either during the *ad libitum* test meal or for the remainder of the day. There was also no increase in plasma GLP-1; in contrast, we found a small but significant decrease 90 min after the breakfast with oat β-glucan. In line with the literature, we observed significant reductions in postprandial blood glucose and plasma insulin (Tosh 2012).

The potency of oat and barley β-glucan to modulate appetite has been reported in several studies, although the effect seems to vary depending on the study design, subject characteristics (e.g. BMI, sex) and the dose and MW of β-glucan consumed (doses range from 2.2 to 9.4 g with varying or unreported MWs (Vitaglione et al., 2009; Beck et al., 2009a; Lyly et al., 2009; Willis et al., 2009; Clegg & Thondre, 2014)). Fullness was increased in 14 healthy overweight subjects following a 3.82 g dose of β-glucan oat bran cereal with a high MW of 1.378 x 10^6 $gmol^{-1}$ consumed at breakfast in a study conducted by Beck *et al*. (2009a), and similarly by Pentikainen *et al*. (2014) following 4 g of high MW β-glucan incorporated into biscuits and juice consumed at breakfast by normal weight female subjects. In contrast, Beck et al (2009a) reported no effect on subjective appetite ratings following a 5.65 g high MW β-glucan

containing breakfast. Our findings are in line with the majority of studies and suggest that oat β-glucan beneficially modulates appetite by increasing fullness and satiety.

The manufacturing process such as baking, cooking or extrusion (Hu et al., 2010; Ames et al. 2015b) and other test food characteristics including food matrix and formats may also affect the satiating capacity of oat β-glucan (Rebello et al., 2014; El Khoury et al., 2012). The physicochemical properties of the matrix in which the fibre is delivered in combination with the gut environment play a critical role in determining the hydration or swelling and water-retention capacity of the fibre (Rebello et al., 2016b). In our study, β-glucan was consumed in a semi-solid food matrix, with yoghurt and cereal with milk used as the vehicle to deliver β-glucan. Other studies that have used a semi-solid food matrix also reported increased satiety and fullness with test meals containing 1.6 to 4 g oat β-glucan (Rebello et al., 2014; Rebello et al., 2016a; Geliebter et al., 2015). Juvonen and colleagues (2011), however, found no effect on subjective appetite following a semi-solid semolina-based pudding that contained 5.1 g oat β-glucan, suggesting that the food matrix alone does not determine oat β-glucan´s satiating capacity. More research is, thus, warranted to better understand how a fibre's satiating capacity depends on experimental paradigms, population characteristics and fibre/food format features.

The beneficial effect on subjective appetite did not translate into a decrease in food intake at the *ad libitum* test meal, which is in line with a number of studies that have reported similar dissociations following oat β-glucan consumption (Beck et al., 2009a; Clark and Slavin, 2013). It is important to note that although appetite VAS are generally sensitive to experimental manipulations and are reproducible, they have failed to predict meal size under a number of conditions (Beck et al., 2009a; Stubbs et al., 2007; Flint et al., 2000). The magnitude of differences in self-reported VAS which precede *ad libitum* eating were investigated recently

by Sadoul *et al*. (2014) based on a large number of studies that used a wide range of nutrient preloads. They found that a significant difference in energy intake at lunch was likely to be achieved if the difference in satiety VAS (intervention *vs.* control) immediately before the *ad libitum* meal was at least 15–25 mm on a 100 mm scale. In our study, differences in satiety VAS at meal onset 150 min after the preload was only about 10 mm which may possibly explain the lack of effect on *ad libitum* eating. Whether a different inter-meal interval or a higher dose of oat β-glucan or another food matrix may have resulted in significant eating effects should be investigated more comprehensively, for example, by using varying time intervals, doses and formats in the same study. Because satiation depends on both gastric and intestinal nutrient stimulation, and their interactions (Steinert et al., 2017), an optimal dose and timing between preload and *ad libitum* test meal is likely crucial to detect an eating-inhibitory effect. Perhaps the best method may be to have participants select the time of the next meal, this approach has been scarcely explored.

Several lines of evidence support the hypothesis that increased gastric volume contributes to satiation (Steinert et al., 2017). Viscous fibres absorb large quantities of water and most studies link ingestion of viscous dietary fibres to delayed gastric emptying (Benini et al., 1995; Bergmann et al., 1992; Marciani et al., 2000, de Graaf et al., 2004), which will increase gastric-volume signals. Bergmann *et al* (1992), for example, found sensations of satiety and hunger highly correlated with gastric emptying rates following consumption of viscous psyllium fibre ($r=0.989$, $p=0.0001$). We did not measure gastric emptying in the current study; however, there are a few studies that report a slowing of gastric emptying with cereal β-glucans under similar conditions (Juntunen et al., 2002; Geliebter et al., 2015; Yu et al. 2014).

376 It has been speculated that because viscous dietary fibres increase the viscosity of digesta in
377 the small intestine, they prolong small intestinal transit time and absorption rate of nutrients,
378 which increases contact time with enteroendocrine cells and, thus, peptide release. In addition,
379 high viscous fibres may disrupt proper mixing of food particles and digestive enzymes,
380 resulting in an increased delivery of unabsorbed nutrients into distal parts of the small intestine
381 where the density of GLP-1 and PYY secreting L-cells is highest (Kristensen & Jensen, 2011;
382 Rebello et al. 2016b). Indeed, Beck and colleagues reported that CCK and PYY increases
383 linearly with increasing amounts of oat β-glucan (Beck et al., 2009a; Beck et al., 2009b). For
384 PYY, there was a significant dose response relationship between grams consumed and PYY
385 AUC ($r^2 = 0.994$, $P = 0.003$). The effect was most pronounced with doses of 4 to 6 grams at a
386 late postprandial phase, as a function of both viscosity and concentration (Beck et al., 2009b).
387 Juntunen et al., (2002) demonstrated an increase in post-prandial GLP-1 at 120 and 150 minutes
388 following 5.4 g β-glucan-containing rye bread. In contrast, a study by Ames et al., (2015)
389 reported no effect of barley fibre enriched tortillas on post-prandial GLP-1 secretion, doses of
390 which ranged from 4.5 g to 11.6 g β-glucan. Moreover, when the natural viscosity of a 300 mL
391 beverage containing 30 g oat bran concentrate (including 5.1 g soluble fibre) was reduced by
392 β-glucanase treatment, CCK, PYY and GLP-1 were increased rather than decreased when
393 compared with the high viscous isocaloric control drink (Juvonen et al., 2009). The high-
394 viscosity beverage was still rated as more filling than control, and although there was no
395 difference in *ad libitum* eating this finding suggests that increased viscosity does not favour
396 CCK, PYY and GLP-1 secretion and that oat bran affects appetite independent of GI peptide
397 secretion. Our findings are in line with the latter study. We found a small but significant
398 decrease in plasma GLP-1 at 90 min with oat β-glucan suggesting that (i) high viscous oat β-
399 glucan does not favor activation of enteroendocrine cells and, thus, GLP-1 secretion and (ii)
400 oat β-glucan beneficially modulates appetite independent of increased plasma GLP-1, at least

401 under our conditions. Assuming that oat β-glucan slowed gastric emptying 0-90 min after meal
402 onset, an explanation for the decrease in plasma GLP-1 is provided by studies showing that the
403 secretion of GLP-1 and other GI peptides is dependent on intestinal caloric load, with higher
404 loads resulting in larger responses (Pilichiewicz et al., 2007). We can, however, not exclude an
405 effect on later phase GLP-1 secretion due to increased delivery of unabsorbed nutrients into
406 distal intestinal parts.
407
408 As expected, consumption of β-glucan at breakfast significantly blunted the post-prandial
409 blood glucose and insulin responses in line with a large number of previous studies (Tosh
410 2012). This is most likely due to a delay in gastric emptying and subsequent glucose absorption,
411 although we did not directly assess this. However, we found that the test meal rich in oat β-
412 glucan showed substantially higher viscosity than the control meal, which supports this
413 hypothesis.
414
415 There are a number of limitations that require consideration. We did not measure plasma GLP-
416 1 (or other satiation peptide) concentrations >90 min after the breakfast meal and thus can only
417 speculate about hormone level at *ad libitum* test meal onset. Caution should also be taken when
418 interpreting data for GLP-1 and insulin due to missing data and, thus, smaller sample size.
419 Moreover, variability in energy intakes may have resulted due to menstrual cycle status
420 (Asarian and Geary, 2013), which was not monitored or controlled in female subjects in our
421 study. Finally, palatability ratings of the β-glucan breakfast were lower than the control
422 breakfast, thus, memory or other cognitive effects may have influenced subsequent eating as
423 suggested by some studies (Johnson and Vickers, 1992; Yeomans et al., 2001).
424
425

In conclusion, 4 g of high MW oat β-glucan beneficially modulated appetite with increased feelings of fullness and satiety but with no effect on *ad libitum* eating. This is associated with reduced plasma GLP-1 at 90 min, and a significant reduction in blood glucose and plasma insulin.

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615

Table 1. Ingredients, energy and macronutrient composition of the breakfasts

	Control	**β-glucan**
Ingredients		
Kellogg's Rice Krispies (g)	30	30
Semi-skimmed milk (1.8% fat) (mL)	150	150
Greek-style yoghurt (g)	90	80
Protein milk (mL)	28	-
OatWell oat bran (g)	-	14.6
Nutrient content		
Total energy (kcal)	319.8	329.1
Fat g (% of total energy)	11.6 (33)	11.3 (31)
Carbohydrate g (% of total energy)	39.7 (50)	39 (47)
Protein g (% of total energy)	13.8 (17)	13.9 (17)
Fibre (g)	0.3	7.9
β-glucan (g)	0	4
Weight (g)	298	274.5

Nutritional information was taken from nutrient declarations present on product food labels

623 **Table 2.** Food consumed at the *ad libitum* test meal and for the remainder of the study day

624 following control and β-glucan breakfasts

	Control	**β-glucan**	**p-value**
Energy intake at *ad libitum* test meal (kcal)	704 ± 51	681 ± 46	0.388
Food quantity at *ad libitum* test meal (g)	275 ± 20	267 ± 18	0.404
Water intake at *ad libitum* test meal (mL)	213 ± 11	218 ± 15	0.751
Energy intake for the remainder of study day (subsequent 12 h) (kcal)	886 ± 91	1094 ± 120	0.099

625 Data are from n=33 subjects except for food intake for the remainder of study days, where

626 one subject failed to return their food record (n=32). Data are means ± SEM

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636 **Table 3.** Area under the curves (AUC) for blood glucose and plasma insulin and total GLP-1

637 concentrations following control and β-glucan breakfasts

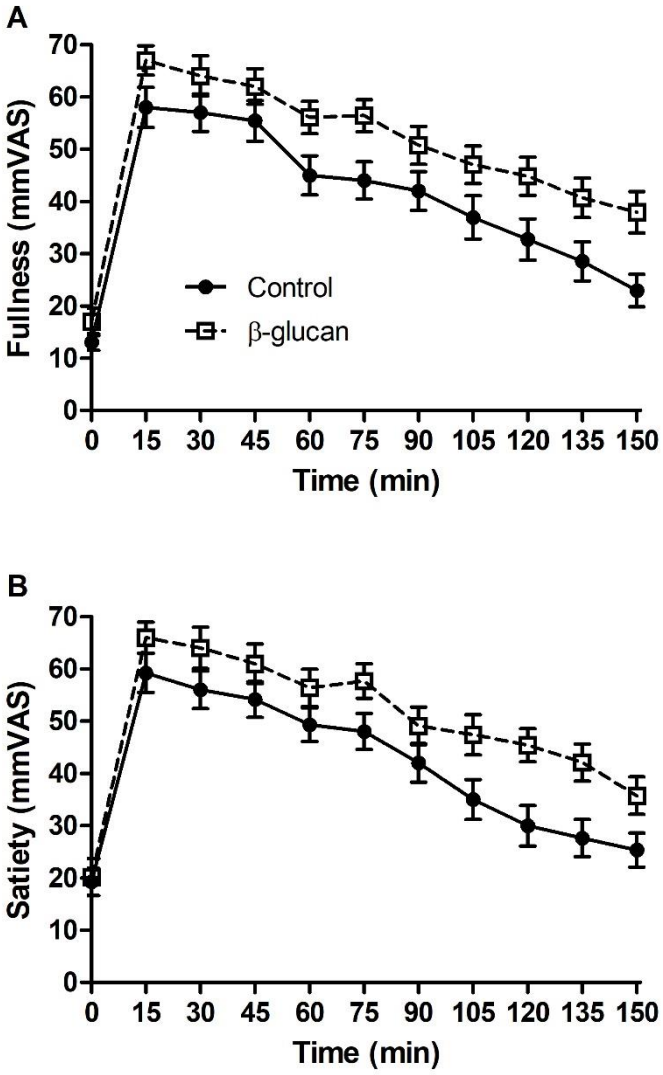
	Control	**β-glucan**	**p-value**
Glucose *AUC (mmol x L^{-1} x min^{-1})*	498.2 ± 47.3	483.0 ± 49.5	0.235
Insulin *AUC (μU x mL^{-1} x min^{-1})*	2491.0 ± 1211	1682.2 ± 902.4	0.001
Total GLP-1 *AUC (pmol x L^{-1} x min^{-1})*	1732.7 ± 713	1654.7 ± 706.8	0.560

638 Data are from n=33 subjects for blood glucose and n=21 for plasma insulin and total GLP-1.

639 AUC based on 0-90 min data. Data are means ± SEM

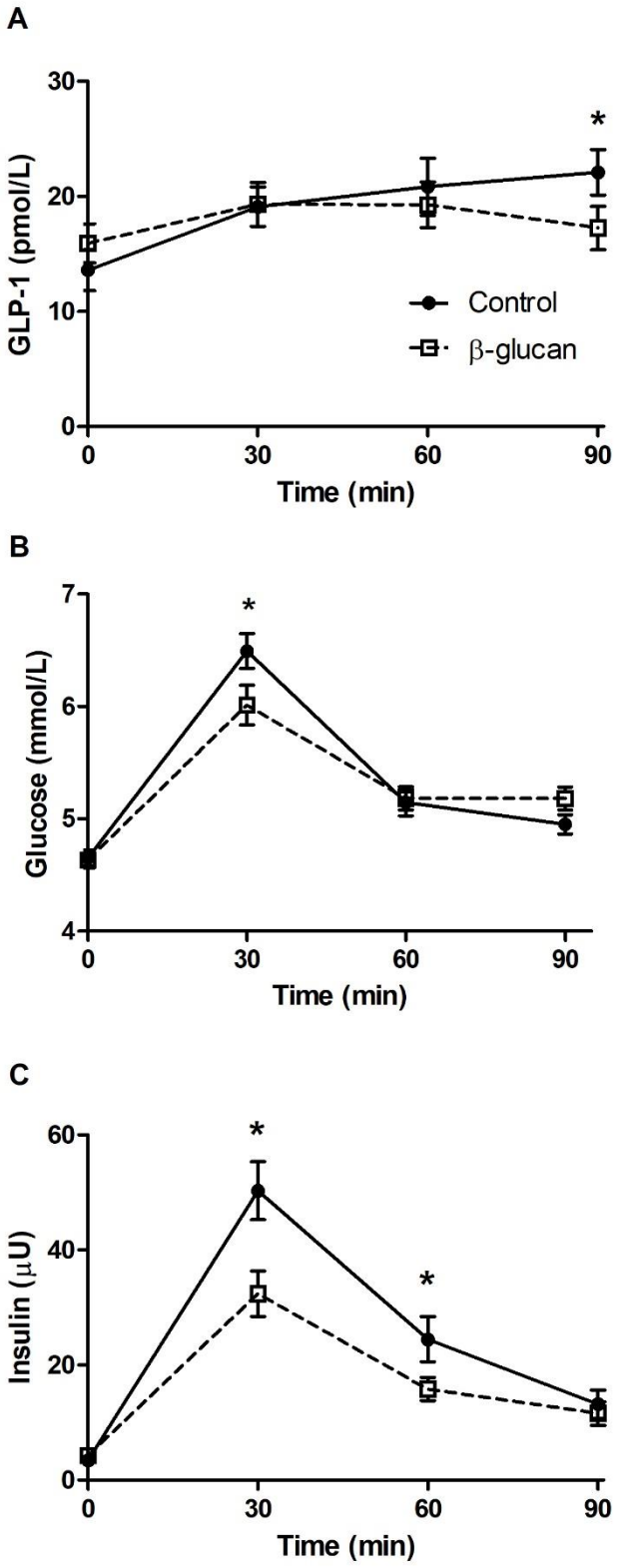
640

641 **Figure 1**



642

643 **Figure 2**



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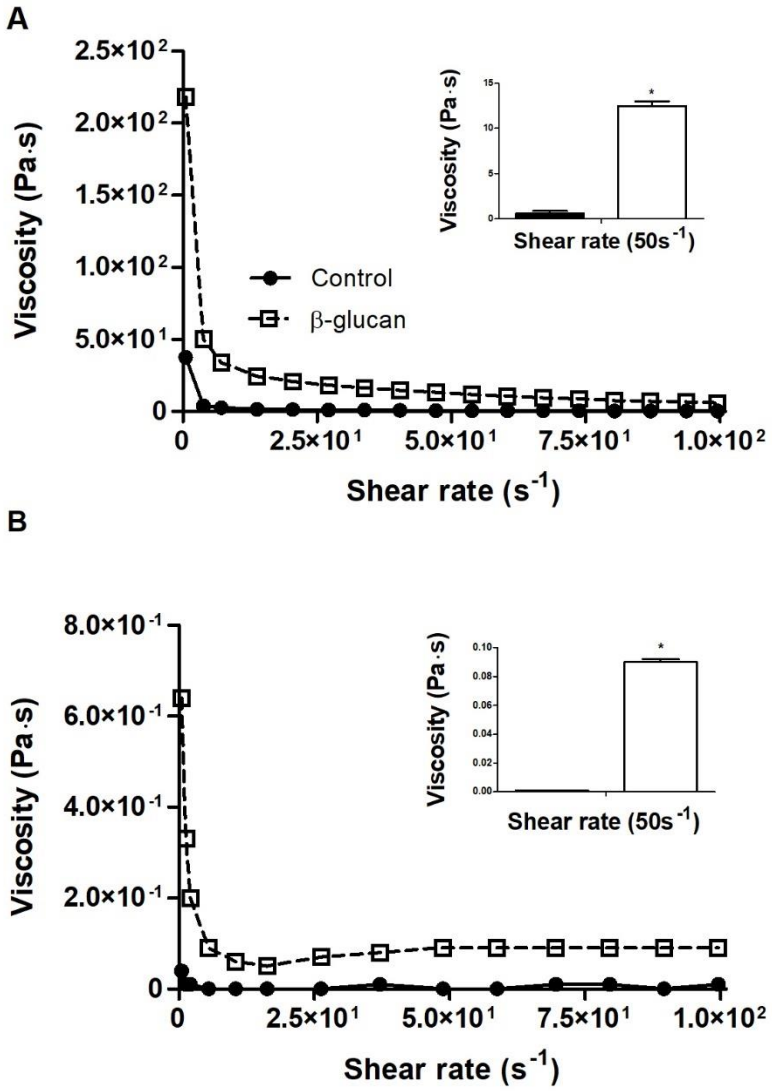
647 **Figure 3**



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651 **Figure legends:**

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653 **Figure 1** Visual analogue scales (VAS) for subjective ratings of fullness (A) and satiety (B)
654 during the 150-min postprandial period following control (●) and β-glucan (□) breakfast
655 consumption. Data were analysed by ANCOVA using baseline value as co-variate. Data are
656 means ±SEMs (n=31, two subjects were excluded from data analysis as they misunderstood
657 the VAS questionnaires).

658

659 **Figure 2** Plasma glucagon-like peptide-1 (A), blood glucose (B), and plasma insulin (C)
660 concentrations during the 90-min postprandial period following control and β-glucan breakfast
661 consumption. Data were analysed with two-factor ANOVA, with treatment and time as factors.
662 In case of significant differences, post hoc comparisons, adjusted for multiple comparisons by
663 Bonferroni correction, were performed to determine significant differences between the control
664 (●) and β-glucan (□) breakfasts. *p<0.05. Data are means ±SEMs (A, n=33; B and C, n=21, 12
665 subjects were excluded from the analysis due to incomplete data sets).

666

667 **Figure 3** Viscosity of both, the yoghurt (A) and milk (B) component of the test meal containing
668 high-molecular weight oat β-glucan (□) or control (●) across different shear rates ranging from
669 $0.5x10^{-1}$ to $1.0x10^{2}$ s^{-1}. Inserts depict a shear rate of $50s^{-1}$, representative of gastric conditions.
670 *p<0.05