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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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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.

1. 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, Kent, Winn, Cohen, & Neumann, 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 (Poutanen et al., 2017; Wanders et al., 2011), 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, 1973; Rolls et al., 1999) but exerts a direct inhibitory effect on eating (Ibarra, Astbury, Olli, Alhoniemi, & Tiihonen, 2014; Pereira & Ludwig, 2001; Wanders et al., 2011). 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, Drummond, & Steinert, 2017), several studies suggest that fibre viscosity is the dominant characteristic that determines the satiating effect (Clark & 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

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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, Andersson, & Åman, 2005) but is decreased by food preparation such as bread-making or further extrusion that impacts bioactivity of cereal β-glucan (Tosh, Brummer, Wolever & Wood, 2008; Tosh et al., 2010; Wang & 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, Chambers, Morrison, & Frost, 2015; Kristensen & Jensen, 2011; Wanders et al., 2013), the role of GI hormones with hypothesized roles in appetite (Steinert et al., 2017) remains controversial. Some studies report postprandial reductions in ghrelin (Vitaglione, Lumaga, Stanzione, Scalfi, & Fogliano, 2009) and increases in cholecystokinin (CCK) and peptide YY (PYY) (Beck, Tapsell, Batterham, Tosh, & Huang, 2009; Beck, Tosh, Batterham, Tapsell, & Huang, 2009; Vitaglione et al., 2009), while others found no effects on PYY (Weickert et al., 2006) and glucagon-like peptide-1 (GLP-1) (Ames, Blewett, et al., 2015). Moreover, although one study suggested that PYY secretion was increased by more viscous foods (Beck, Tapsell, et al., 2009), 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 hypothesized 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.

2. Materials and methods

2.1. 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 and 170 kcal, 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 h by 170 kcal; whereas Rebello, Johnson, et al. (2016) 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, α = 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 ± 2.0 kg; BMI 23.5 ± 0.4 kg/m^2; waist circumference 78.0 ± 1.5 cm). 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, Frijters, Bergers, & Defares, 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.

2.2. 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 h) 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, 7.3 g OatWell powder was mixed with Greek-style yoghurt and 7.3 g 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 2.213×10^6 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 min and afterwards to rate the palatability of both breakfasts using a VAS. The breakfasts were matched for their protein,