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ORIGINAL ARTICLE

Muesli with 4 g oat β-glucans lowers glucose and insulin responses after a bread meal in healthy subjects

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Objective: To evaluate the impact of an extruded muesli product based on β-glucan-rich oat bran on postprandial glycaemia and insulinaemia.
Subject/Design: The study is divided in two series. Blood glucose and serum insulin responses were studied after subjects consuming test meals including a serving of muesli with 3 g (series 1) and 4 g (series 2) of β-glucans, respectively. The muesli was a component in a single serving packet with muesli and yoghurt. This was served together with white wheat bread in the morning after an overnight fast. The compositions were standardized to contain 50 g available carbohydrates. As a reference meal a serving packet without β-glucans was included. The study was performed at Applied Nutrition and Food Chemistry, Lund University, Sweden. Nineteen and thirteen healthy volunteers with normal body mass index were recruited for series 1 and 2, respectively.
Results: Muesli with 3 g of β-glucans, included in a mixed bread meal, gave no significant differences in glycaemic response compared to a reference meal without muesli and β-glucans. In contrast, muesli with 4 g of β-glucans significantly ($P<0.05$) lowered the glucose and insulin responses compared to the reference meal.
Conclusions: Muesli enriched with 4 g of β-glucans reduces postprandial glucose and insulin levels to a breakfast based on high glycaemic index products. A total of 4 g of β-glucans from oats seems to be a critical level for a significant decrease in glucose and insulin responses in healthy people.
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Keywords: β-glucan; glucose response; insulin response; functional foods; oat bran; glucose tolerance

Introduction

A major challenge of nutrition science is in the combat of diet-related disorders. In particular, the global pandemic of type 2 diabetes and pre-diabetic states goes hand in hand with a global pandemic of obesity, with the majority of the type II diabetics also suffering from obesity. Dietary measures that could facilitate weight maintenance, and improve insulin sensitivity are thus of interest in the prevention of the insulin resistance syndrome.

The postprandial glucose level after carbohydrate consumption is known to induce hormonal and metabolic responses with potential influence on health. In this respect food, characterized by a low glycaemic response (low glycaemic index (GI) foods), has been found to induce benefits on certain risk factors for chronic diseases, such as type II diabetes, cardiovascular disease and obesity. A low-GI diet may improve management of diabetes by lowering early postprandial hyperglycaemi and decreasing risk for post-absorptive hypoglycaemia (Brand *et al.*, 1991; Wolever *et al.*, 1992; Järvi *et al.*, 1995; Gilbertson *et al.*, 2001). By inducing low insulin levels (Björck *et al.*, 2000) and increased insulin sensitivity (Wolever *et al.*, 1992; Frost *et al.*, 1996, 1998; Järvi *et al.*, 1999) a low-GI diet also affects the risk for other

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metabolic diseases associated with the insulin resistance syndrome, for example, cardiovascular disease (Wannamethee *et al*., 2005). A recent examination of the Framingham Offspring Study showed that diets with lower GI are associated with lower insulin resistance, and a reduced risk of developing the insulin resistance syndrome (McKeown *et al*., 2004). Epidemiological evidence are also at hand indicating a preventive role of a low glycaemic response (Salmerón *et al*., 1997a, b) and of grains and cereal fibre (Meyer *et al*., 2000) against development of type 2 diabetes. Further, a negative correlation was found between serum high-density lipoprotein cholesterol, another predictor of cardiovascular disease, and GI of the diet (Frost *et al*., 1999). Epidemiological studies have also found a preventive role of low-GI diet in relation to cardiovascular disease (Liu *et al*., 2000, 2001), whereas van Dam *et al*. (2000) did not find such a correlation.

With respect to obesity that has reached to epidemic proportions, the efficacy of low-fat diets has been questioned in recent years and instead the hypothesis has been focused on the intake of high-GI foods (Ludwig, 2000; Brand-Miller *et al*., 2002; Pawlak *et al*., 2002). Various studies have demonstrated positive acute effect of low-GI food, for example, increased satiety in healthy subjects (Granfeldt *et al*., 1994; Liljeberg and Björck, 1998; Östman *et al*., 2005) and decreased voluntary food intake in obese subjects (Ludwig, 1999). Further medium- to long-term studies in obese subjects are at hand, for example, the findings that a low-GI diet reduced fasting insulin levels in parallel to a weight loss in obese women in a 12-week study (Slabber *et al*., 1994), or that the body weight of obese children decreased more after a low-GI diet compared to a standard reduced fat diet in a 15-month study (Spieth *et al*., 2000) In the EURODIAB Complications Study of nearly 3000 adults with type I diabetes, consumption of lower GI diet was found to be related to lower measures of intra-abdominal fat mass and total body fat (waist-to-hip ratio and waist circumference) independently of carbohydrate, fat or fibre intake (Toeller *et al*., 2001). In the most recent WHO report, 'Diet nutrition and the prevention of chronic diseases' (FAO/WHO, 2003), the preventive potential of low-GI diets in relation to obesity and diabetes was graded as 'possible'.

The glycaemic response of a starchy food is influenced by its rate of intestinal absorption, which in turn is influenced by its gross matrix structure (Granfeldt *et al*., 1991; Liljeberg *et al*., 1992; Tovar *et al*., 1992), the starch crystallinity (Granfeldt *et al*., 1995a, 2000, 1995b) and food components such as certain organic acids (Liljeberg and Björck, 1998; Liljeberg *et al*., 1995) or viscous dietary fibre (Holm and Bjorck, 1992; Fairchild *et al*., 1996; Liljeberg *et al*., 1996).

Current dietary recommendations emphasize generous amounts of carbohydrate foods and dietary fibre in the diet. The daily recommendation for dietary fibre intake in Sweden is 25–35 g. Soluble fibre has generated considerable interest because of its potential to moderate the rate of the postprandial glucose delivery to the blood (Nutall, 1993) and of its capacity to affect cholesterol metabolism (Brown *et al*., 1999). Water-soluble, gel-forming fibre in the form of guar gum, and β-glucans added to glucose solution or mixed with food reduce the expected rise in blood glucose and insulin concentration both in diabetics (Tappy *et al*., 1996; Jenkins *et al*., 2002) and healthy subjects (Jenkins *et al*., 1977; Fairchild *et al*., 1996; Liljeberg *et al*., 1996). In long-term control studies, various soluble fibres have been shown to reduce low-density lipoprotein cholesterol such as psyllium (Anderson *et al*., 1995), β-glucans (Önning *et al*., 1999; Kerckhoffs *et al*., 2003), guar gum (Aro *et al*., 1981) and leguminous fibre (Simpson *et al*., 1981). Further, oat bran concentrate has been found to improve long-term control of diabetes (Pick *et al*., 1996). The active component in oats is refereed to be β-glucans.

This mechanism for reducing glycaemic response and cholesterol may be utilized in the formulation of functional food. However, when used commercially, many factors, for example, cost and taste are of importance. In a recent study by Jenkins *et al*. (2002) it was concluded that β-glucans may be useful as a functional food component for reducing postprandial glycaemia, without changing the palatability of the product. In particular, there is a need to develop new low-GI food alternatives among the cereal products and many of the current bread and muesli-type products are characterized by high GI.

The purpose of the present paper was to study the effects on postprandial glycaemia and insulinaemia of an extruded muesli product based on a β-glucan–enriched oat bran as a major ingredient. Two levels of β-glucans, 3 or 4 g, were included in a breakfast based on muesli/yoghurt and white bread, and tested in healthy subjects. A similar meal without β-glucans was used as reference. The Ethics Committee of the Faculty of Medicine at Lund University approved the study.

Materials and methods

Experimental design

The present study is divided in two series: with the test meal including a serving of muesli with 3 and 4 g of β-glucans, respectively. The muesli was a component in a single serving packet with muesli and yoghurt. The effect of this serving packet with muesli/yoghurt on blood glucose and insulin responses after a bread breakfast meal was studied. As a reference meal a serving packet without β-glucans was included.

Series 1

Test meal, including 3 g β-glucans. The test meal and the reference meal were provided by (Skånemejerier, Malmö, Sweden). The test meal consisted of a serving packet with vanilla yoghurt and muesli with 3 g β-glucans besides that a sandwich with white wheat bread, cheese and butter were included. The muesli consisted of flakes made from oat bran (OatWell, Swedish Oat Fiber/Crea Nutrition, Väröbacka,

Sweden), dried fruit (3.1 g), wheat germ, corn flakes, malt extract and salt. The amount of oat bran flakes was adjusted to contain 3 g of β-glucans. The reference meal was the same as the test meal, except for the oat bran flakes in the muesli. The content of available carbohydrates in the muesli was compensated for more bread in the reference meal. The compositions were standardized to contain 50 g available carbohydrates (Table 1).

Subjects. Nineteen healthy, non-smoking volunteers, 13 women and 6 men, took part in the study. Their average age was 37.5 ± 15.2 years (mean ± s.d.) and their mean body mass index 22.4 ± 0.6 (mean ± s.d.). The night before every test breakfast, the subjects were requested to eat a standardized late-evening meal, based on 2–3 slices of white wheat bread. After 10 pm, the subjects were allowed to drink only water. The reference and test breakfast meals were served randomized after an overnight fast. The tests were performed approximately 1 week apart and commenced at the same time in the morning. All meals were consumed steadily and finished within 12–14 min. Water (150 ml) and 150 ml tea or coffee was served with each meal. The test subjects were allowed to choose between these drinks and retained the same drink through the study.

Series 2

Test meal, including 4 g β-glucans. The test meal in series 2 consisted of a serving packet with vanilla yoghurt and muesli with 4 g β-glucans besides that a sandwich with white wheat bread, cheese and butter was included. The muesli consisted of flakes made from oat bran (OatWell), dried fruit (3.1 g), wheat germ, corn flakes, malt extract and salt. The amount of oat bran flakes was adjusted to contain 4 g of β-glucans. The test meal in series 2, thus consisted of exactly the same ingredients as the test meal in series 1 with the exception that more oat fibre flakes were included in the muesli, to an amount containing 4 g β-glucans. The reference meal was the same as the test meal, except for the muesli. The content of available carbohydrates in the muesli was compensated for more bread in the reference meal. The compositions were standardized to contain 50 g available carbohydrates (Table 2).

Table 1 Composition of the test meal and the reference meal, series 1

	Muesli[a]	*Vanilla yoghurt*[a]	*White wheat bread*[b,c]	*Cheese*[c]	*Butter*[c]	*Total*
Test meal						
Weight (g)	19	200	47.6	13	3.5	
Carbohydrates (g)	7.5	24.0	18.5	—	—	50.0
Protein (g)	3.2	8.0	2.9	4.0	—	18.1
Fat (g)	1.0	1.0	0.6	2.2	2.8	7.6
Reference meal						
Weight (g)	3.1	200	60.0	13	3.5	
Carbohydrates (g)	1.2	24.0	23.3	—	—	48.5
Protein (g)	—	8.0	3.7	4.0	—	15.7
Fat (g)	—	1.0	0.8	2.2	2.8	6.8

[a]Nutrient composition according to product information.
[b]Carbohydrates analysed according to Holm *et al.* (1986).
[c]Protein and fat are values from the Swedish food composition tables (www.slv.sc).

Table 2 Composition of the test meal and the reference meal, series 2

	Muesli[a]	*Vanilla yoghurt*[a]	*White wheat bread*[b,c]	*Cheese*[c]	*Butter*[c]	*Total*
Test meal						
Weight (g)	27	200	39.6	13	3.5	
Carbohydrates (g)	10.6	24.0	15.4	—	—	50.0
Protein (g)	4.6	8.0	2.4	4.0	—	19.0
Fat (g)	1.5	1.0	0.6	2.2	2.8	8.1
Reference meal						
Weight (g)	—	200	66.9	13	3.5	
Carbohydrates (g)	—	24.0	26	—	—	50.0
Protein (g)	—	8.0	4.1	4.0	—	16.1
Fat (g)	—	1.0	0.9	2.2	2.8	6.9

[a]Nutrient composition according to product information.
[b]Carbohydrates analysed according to Holm *et al.*, 1986).
[c]Protein and fat are values from the Swedish food composition tables (www.slv.sc).

Subjects. Thirteen healthy, non-smoking volunteers, eight women and five men, took part in the study. Their average age was 37.5±3.6 years (mean±s.d.) and their mean body mass index 22.4±0.6 (mean±s.d.). The night before every test breakfast, the subjects were requested to eat a standardized late evening meal, based on 2–3 slices of white wheat bread. After 10 pm, the subjects were allowed to drink only water. The reference and test breakfast meals were served randomized after an overnight fast. The tests were performed approximately 1 week apart and commenced at the same time in the morning. All meals were consumed steadily and finished within 12–14 min. Water (150 ml) and 150 ml tea or coffee was served with each meal. The test subjects were allowed to choose between these drinks and retained the same drink through the study.

Sampling and analysis

The subjects arrived in the laboratory in the morning after an overnight fast. A fasting blood sample was taken before the meal was served. After the breakfast, blood samples were taken at 15, 30, 45, 70, 95 and 120 min for analysis of glucose and at 15, 30, 45, 95 and 120 min for analysis of insulin.

Blood glucose concentrations were determined with a glucose oxidase peroxidase reagent and serum insulin concentrations were determined with an enzyme immunoassay kit (Mercodia AB, Uppsala, Sweden).

The Ethics Committee of the Faculty of Medicine at Lund University approved the study.

Statistical analysis

The areas under the curves (AUCs) (0–95 and 0–120 min) were determined for blood glucose and serum insulin (GraphPad Prism ver. 3.0; GraphPad Software, San Diego, CA, USA). GI and II (insulinaemic index) were calculated from the AUCs with each subject being their own reference. All areas below the baseline were excluded from the calculations. Values are presented as mean±s.e.m. All statistical calculations were performed in Minitab Statistical Software (release 13 for Windows; Minitab Inc., State College, PA, USA). Significances were evaluated with the general linear model (analysis of variance) followed by Tukey's multiple comparisons test. Values of $P<0.05$ were considered significant.

Results

Series 1

When the muesli/yoghurt with 3 g β-glucans was included in the mixed bread-based meal no significant differences were seen in the glycaemic response (Figure 1), except for at one time point, 15 min, where the blood glucose level was significantly lower after the test meal with the oat bran flakes, comparing to the reference meal ($P<0.05$). A reduction of the incremental area under curve (0–95 min) by 17.6% was obtained compared to the reference meal, although it was not significant (Table 3).

The postprandial insulin response was significantly lower at two time points, 15 and 30 min, after the test meal, compared to the reference meal ($P<0.05$, Figure 2). No significant difference was seen between the insulin areas

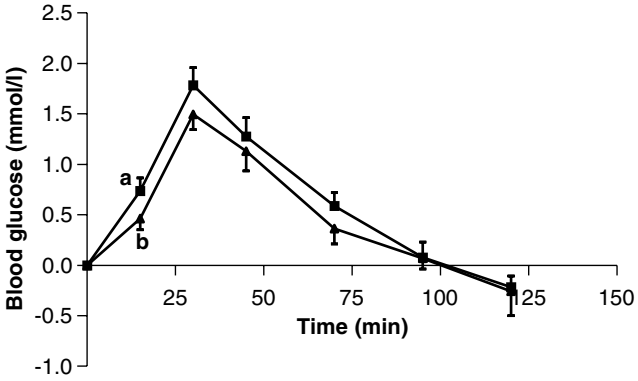

Figure 1 Mean incremental blood glucose responses in healthy subjects following ingestion of breakfast meals; a test meal with wheat bread, yoghurt and muesli with oat bran flakes containing 3 g of β-glucans (▲) and a reference meal with white wheat bread, yoghurt and muesli without oat bran flakes (■). Values with different letters are significantly different ($P<0.05$).

Table 3 Fasting values, postprandial glucose and insulin areas, series 1[1]

	Test meal	*Reference meal*	*Reduction in postprandial area, % of reference meal*
Blood glucose			
Fasting value (mmol/l)	4.8±0.1[b]	4.7±0.1[a]	
Incremental area under curve (0–95 min) (mmol min/l)	69.6±7.5[a]	84.5±7.7[a]	17.6
Incremental area under curve (0–120 min) (mmol min/l)	79.7±7.7[a]	95.7±8.6[a]	16.7
Serum insulin			
Fasting value (pmol/l)	62±5.5[a]	62±10.7[a]	
Incremental area under curve (0–95 min) (nmol min/l)	19.5±1.1[a]	22.3±2.0[a]	12.6
Incremental area under curve (0–120 min) (nmol min/l)	20.9±2.1[a]	22.3±2.1[a]	6.3

[1]Mean values±s.e.m., $n=19$. Mean values with different letters in each row are significantly different (ANOVA followed by Turkey's test), $P<0.05$.

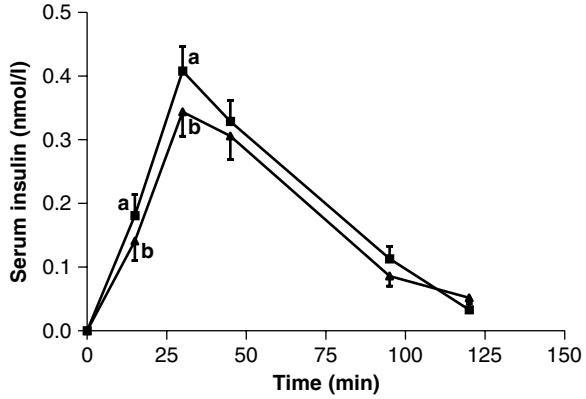

Figure 2 Mean incremental serum insulin responses in healthy subjects following ingestion of breakfast meals; a test meal with wheat bread, yoghurt and muesli with oat bran flakes containing 3 g of β-glucans (▲) and a reference meal with white wheat bread, yoghurt and muesli without oat bran flakes (■). Values with different letters are significantly different ($P<0.05$).

after the two meals, although a reduction of 12.6% could be observed for the first 95 min (Table 3).

Series 2

When muesli with 4 g β-glucans was served with the white bread the glycaemic responses were significantly lower during the first 70 min period, compared to the reference meal without muesli ($P<0.05$, Figure 3). In the late-postprandial phase, the glucose response after the reference meal decreased faster than after the test meal with muesli; and at 95 min the glucose response was significantly higher after the test meal, compared to the reference meal ($P<0.05$, Figure 3). The area under the glucose curve after the test meal was significantly lower than after the reference ($P<0.05$, Table 4).

The early postprandial insulin response ($\leqslant$45 min) was significantly lower after the test meal with muesli compared to the reference meal ($P<0.05$, Figure 4). The area under the

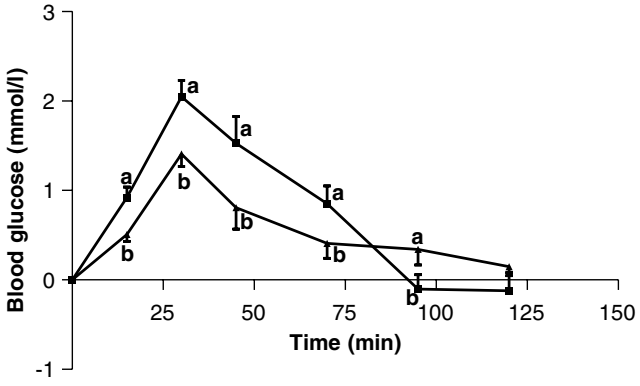

Figure 3 Mean incremental blood glucose responses in healthy subjects following ingestion of breakfast meals; a test meal with wheat bread, yoghurt and muesli with oat bran flakes containing 4 g of β-glucans (▲) and a reference meal with white wheat bread and yoghurt without muesli (■). Values with different letters are significantly different ($P<0.05$).

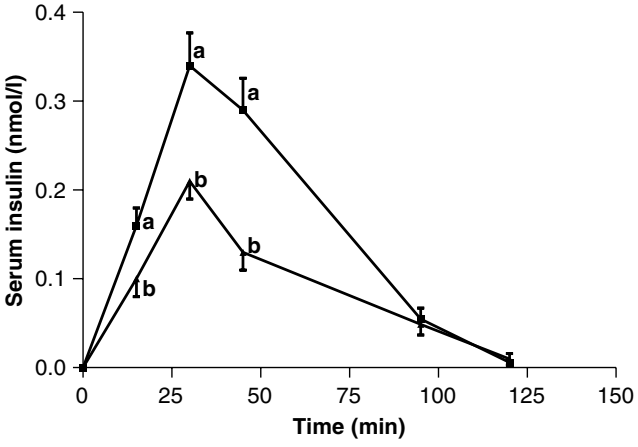

Figure 4 Mean incremental serum insulin responses in healthy subjects following ingestion of breakfast meals; a test meal with wheat bread, yoghurt and muesli with oat bran flakes containing 4 g of β-glucans (▲) and a reference meal with white wheat bread and yoghurt without muesli (■). Values with different letters are significantly different ($P<0.05$).

Table 4 Fasting values, postprandial glucose and insulin areas, series 2[1]

	Test meal	*Reference meal*	*Reduction in postprandial area, % of reference meal*
Blood glucose:			
Fasting value (mmol/l)	4.5±0.1[a]	4.6±0.1[a]	
Incremental area under curve (0–95 min) (mmol min/l)	63.0±9.5[b,**]	97.7±13.3[a,**]	35.5
Incremental area under curve (0–120 min) (mmol min/l)	71.2±11.1[b,*]	100.7±13.5[a*]	29.3
Serum insulin:			
Fasting value (pmol/l)	48±7[a]	55±12[a]	
Incremental area under curve (0–95 min) (nmol min/l)	10.3±1.1[a,***]	18.3±1.7[b,***]	43.7
Incremental area under curve (0–120 min) (nmol min/l)	11.2±1.2[a,**]	19.3±1.9[b,**]	42.0

[1]Mean values±s.e.m., $n=13$. Mean values with different letters in each row are significantly different (ANOVA followed by Turkey's test) *$P<0.05$, **$P<0.01$, ***$P<0.001$.

insulin curve was significantly lower than after the reference meal ($P<0.05$, Table 4).

Discussion

The muesli/yoghurt product containing 4 g of β-glucans from oat bran in a serving significantly reduced glycaemic and insulinaemic areas as well as levels at specific time points after a bread meal, whereas 3 g of β-glucans did not reach statistical significance regarding effects on metabolic responses although a tendency was seen to lower responses even with this lower β-glucans level. The oat barn used in series 1 and 2 was manufactured in the same way. The reduction in postprandial area (0–90 min) (% of reference meal) was doubled when 1 g more of β-glucans was present in the muesli/yoghurt portion (17.6 and 35.5%, respectively with 3 and 4 g β-glucans). Few previous studies have been carried out with β-glucan–rich oat fractions on high-GI food in healthy subjects. With oat gum (80% β-glucans) the glucose response has been shown to decrease almost 60% when 14.4 g was added to a glucose drink (50 g glucose) (Braaten *et al*., 1991) or 40% when 11 g was added to a wheat porridge (60 g of available starch) (Wood *et al*., 1990). Compared to the present study, higher amounts of β-glucans were used (12 and 9 g, respectively). In the present study we seem to have found a critical level, 4 g of β-glucans, for a significant decrease in glucose and insulin responses after a 50 g carbohydrate portion, in healthy people. We have previously shown that the naturally occurring levels in commercially available oat breakfast products like porridge or flaked cereals do not affect glucose and insulin responses (Granfeldt *et al*., 1994, 1995b). Thus, both a portion of oat flakes, and of oat porridge (50 g available carbohydrates) containing approximately 2.5 g β-glucans gave equally high glucose and insulin responses as white wheat bread. Whereas a portion of barley porridge containing 6.8 g β-glucans made from barley genotypes with elevated contents of β-glucans (17.5 g/100 g) significantly decreased postprandial glycaemia (Liljeberg *et al*., 1996) compared to white wheat bread. Reductions in postprandial glucose have also been seen in diabetics. Tappy *et al*. (1996) showed a decrease of 60% in glycaemic response after 35 g carbohydrate load with 6 g β-glucans, and in a more recent study by Jenkins *et al*. (2002) a reduction in glycaemic response by 12, 27 and 31%, respectively were seen with 3.7, 6.2 and 7.3 g β-glucans. Thus, increasing the dose of β-glucans successively reduced the glucose response, which also was seen in the current study.

The mechanism for a lowering postprandial glycaemia with β-glucans is probably related to an increased luminal viscosity, leading to a prolongation of carbohydrate digestion and absorption (Battilana *et al*., 2001). β-glucan is a source of viscous dietary fibre that preferably is found in oat and barley. However, in a recent study Biörklund *et al*. (2005) found a decrease in postprandial glucose and insulin response in hypercholesterolaemic subjects, after 5 g of β-glucans from oats but not from the same amount of β-glucans from barley. The authors hypothesized that both the lower molecular weight and the lower solubility of the barley β-glucans compared to oat β-glucans resulted in a lower viscosity in the barley product, which probably would influence the difference in glucose response between the food products containing oat and barley β-glucans. Not only is the origin of importance for the viscosity, a degradation of β-glucans may occur during food processing. Åman *et al*. (2004) showed an enzymatic degradation of β-glucan during baking, although the mean molecular weight of β-glucan in oats was retained in, for example, oat flakes and oat porridge. Variation in raw materials, processing conditions or ingredients may modify the physicochemical properties of β-glucan-like viscosity, molecular weight and solubility (Beer *et al*., 1997) and in this way influences the physiological properties. To maintain the functional nutritional attributes of β-glucan, it is important that the processing of oat kernels to oat bran, with an elevated concentration of β-glucan, does not damage the polymeric β-glucan structure. These factors require careful attention during processing, if β-glucan properties are to be maintained or improved in the final food application until consumption. Therefore, *in vitro* models should be used, for the assessment of the physiological properties of viscous dietary fibres.

Also long-term effects of β-glucans may improve glucose metabolism. Thus, in a crossover study with moderately hypercholesterolaemic men and women, a modest amount of β-glucans (5–7.5 g) during 5 weeks was shown to have beneficial effects on glucose and insulin responses after a glucose challenge (Hallfrisch *et al*., 1995), suggesting benefits on glucose tolerance. β-glucans are indigestible in the small intestine but are fermented by bacteria in the colon. With respect to glucose tolerance there is evidence in support of mechanism involving colonic fermentation of indigestible carbohydrates. Accordingly, in a recent overnight study (Granfeldt *et al*., 2004) we showed that an evening meal containing high levels of indigestible carbohydrates (resistant starch and soluble dietary fibre) substantially reduced GI and II of white bread determined at a subsequent breakfast meal compared to an evening meal of white wheat bread. Further, in a follow up study (Nilsson *et al*., 2006) the same evening meal, boiled barley kernels, gave significantly lower blood glucose response to a white wheat bread breakfast, compared to evening meals with white wheat bread or spaghetti with added wheat bran. A high fermentative activity in the colon may increase the colonic production of short-chain fatty acid including propionic acid, which has been implemented as a moderator of hepatic glucose metabolism (Venter *et al*., 1990).

The results of the present study show that a muesli/yoghurt product containing 4 g of β-glucans reduces postprandial glucose and insulin levels to a mixed bread-based breakfast.

The present work further demonstrates that 4 g of β-glucans from oats, with retained physicochemical properties, seems to be a critical level for a significant decrease in glucose and insulin responses, in healthy people. In general, oat products available on the market do not contain sufficient quantities of β-glucan to achieve appreciable health effects of this type, which opens for tailoring and production of new functional food products. However, as the origin processing parameters and food matrix may influence the physiological effects of the β-glucans, new low-GI products need to be carefully evaluated for their physiological effects before entering the market. This may have been evaluated in this study with an *in vitro* approach considering the physiological viscosity.

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