Research Article

Modulation of the postprandial phase by β-glucan in overweight subjects: Effects on glucose and insulin kinetics

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Decreasing the postprandial glucose response is potentially of *major importance to public health* when low-*glycemic* index or high-fibre content foods are associated with a decreased risk of diabetes. We investigated in overweight subjects the effect of adding β-glucan (BG) to a polenta (Pol) meal on postprandial metabolism and glucose bioavailability using stable isotopes. In this single-blind, randomized, crossover trial, 12 subjects ate two meals containing Pol with (Pol + BG) or without (Pol) 5 g BG. Concentrations of glucose, insulin, C-peptide, nonesterified fatty acids, triacylglycerol, total and exogenous glucose kinetics were assessed for 6 h postprandially. The kinetics of total and exogenous glucose importantly differed between the meals, but not the quantity of total and exogenous glucose appearing in plasma. Less total and exogenous glucose appeared during the first 120 min after the Pol + BG meal; *the phenomenon was then reversed* (both $p < 0.0001$). After 120 min, glucose and insulin responses declined, but *remained* higher after the Pol + BG meal ($p < 0.05$) in parallel to the inhibition of lipolysis. The endogenous glucose production (EGP) was significantly more inhibited after the Pol + BG meal. The addition of BG slowed the appearance of glucose *in plasma*, resulting in longer-lasting insulin secretion which exerted a prolonged inhibition of EGP and lipolysis.

Keywords: β-Glucan / Glucose stable isotope / Insulin / Postprandial kinetics / Starch

Received: January 18, 2008; revised: April 21, 2008; accepted: May 20, 2008

1 Introduction

At present in many Western countries, the recommended dietary fibre intake is far from being reached, despite the recommendations of several health organisations to *increase consumption* of foods with a high fibre content or with a low *glycemic* index. The beneficial effect of dietary fibre on postprandial metabolic parameters and *glucose* control has been the object of many studies over recent decades [1 – 7]. Several studies have shown that the risk of type 2 diabetes mellitus is inversely correlated with the intake of diets with a low *glycemic* index or with a high fibre content [8, 9]. It is also well known that a weight gain or an overweight is also risk factor for developing type 2 diabetes [10, 11]. For these reasons, it seemed important to study the mechanisms of action and the effect of β-glucan (BG) fibre when added to a meal in this particular population of overweight individuals.

BGs are fibres which are highly viscous, soluble, fermentable polysaccharides, notably found in barley and oats. BG fibre has been shown to decrease postprandial glucose

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Abbreviations: ANOVA, analysis of variance; **AUC,** area under the curve; **BG,** β-glucan; **EGP,** endogenous glucose production; **NEFA,** nonesterified fatty acid; **Pol,** polenta; **Ra,** rate of glucose appearance; **RaE,** rate of appearance of Exogenous glucose; **RaT,** rate of appearance of Total glucose; **Rd,** rate of glucose disposal;**TG,** triacylglycerol

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and insulin responses during oral glucose tests, or when added to food products (pasta, bread, soup, cereals, *etc*.) in healthy or diabetic subjects [1 – 3, 12 – 15]. Soluble fibres like BGs are presumed to increase the viscosity of the meal bolus, and thus to reduce the rate of nutrient absorption and flatten postprandial *glucose and insulin responses* [13, 14, 16]. BG enrichment of food products has been increasingly studied because it provides more BG *per* serving with minimal loss of palatability and in a smaller ingested volume. But it is difficult to study objectively the effects of BG on glucose metabolism where the fibre is added to a complex meal, as opposed to an intake of glucose only. Moreover, other intervention studies have not revealed the same lowering effect of BG on the glucose response or have even shown some discrepancies between glucose and insulin responses [4, 5].

Thus, it is meaningful to accurately *define* the kinetics of the response to the ingestion of BG in terms of total, exogenous and endogenous glucose, in parallel to measuring plasma glucose and insulin. We could then better understand the coordinated kinetics *underlying final glucose* response. In our study, the use of stable isotopes with double glucose labelling allowed to measure the rate of appearance of exogenous glucose (RaE) (^{13}C glucose) and total glucose (RaT) *and the rate of glucose disposal* (Rd) (D-[6,6-$^2H_2$]glucose) in plasma and to estimate endogenous glucose production (EGP) [17]. Thus, *the glucose coming* from a naturally ^{13}C-enriched product like corn polenta (Pol) can be traced in plasma and the influence of BG on the kinetics of total, exogenous and endogenous glucose can be clarified.

The aim of the study was to compare the bioavailability of the carbohydrates from a meal containing Pol (80 g equiv. glucose), with or without added BG fibre, in overweight subjects. The effects of the two meals on postprandial metabolites and hormone concentrations, on glucose kinetics and on total carbohydrate and lipid oxidation were studied by *analysing* postprandial responses for 6 h after meal ingestion.

2 Materials and methods

2.1 Subjects

Twelve healthy overweight men, aged 34 ± 2 years, weighing 84.0 ± 1.7 kg, with a BMI of 27.5 ± 0.3 kg/m^2, and fasting *glucose concentration* of 5.07 ± 0.09 mmol/L, fasting insulinaemia of 49.03 ± 7.10 pmol/L and fasting total cholesterol concentration of 4.86 ± 0.22 mmol/L (mean ± SEM) were included in the study. We performed a power calculation using as the study of primary endpoint; the difference in *the cumulated quantity of* exogenous glucose appearing postprandially in plasma during 360 min between the two meals. Assuming a difference of 10 g between the two meals (SD = 12) for this parameter, 12 subjects *per* group provided >80% power to detect a significant difference in the total quantity of exogenous glucose appearing in the plasma between the two groups at the $p < 0.05$ level [18].

Inclusion tests were conducted where each subject gave a blood sample, was measured for height and weighed and was interviewed regarding general health. Eating habits, especially usual fibre consumption, were also explored by a trained dietician through a dietary survey. The inclusion criteria for the study were men aged 20 – 60 years, BMI 25 – 30 kg/m^2, normal fasting *glucose concentration* < 7 mmol/L and HbA1c < 6%, stable body weight over the previous 3 months, normal results for preinclusion biological tests, sedentary or moderate physical activity.

All subjects received written and oral information about the protocol and signed an informed consent form. The study was approved by the Scientific Ethics Committee of Lyon (CCPPRB Lyon A) and accorded with both the French 'Huriet-Serusclat' law and the Second Declaration of Helsinki.

2.2 Experimental meals

The fibre-rich preparation was an oat concentrate containing predominately the soluble fraction of BG and was manufactured according to Oatly's method [19], Oatly AB (Landskrona, Sweden). Corn Pol was chosen, as corn is naturally enriched in stable isotope ^{13}C. The corn Pol (Tipiak, Pont Saint-Martin, France) was boiled for 5 min. The subjects consumed one of the meals containing either Pol alone or Pol with BG (5 g) and both were taken with 250 mL of water. The meals were similar in calorie and carbohydrate load (72 g) and both represented 80 g equiv. of glucose (adjusted on the Pol + BG meal: 75 g from Pol and 5 g from BG). The composition of the meals is given in Table 1.

D-[6,6-$^2H_2$] glucose (99 mol% excess) was obtained from Eurisotop (Gif-sur-Yvette, France); chemical and isotopic purity was confirmed by GC selected-ion-monitoring MS analysis. It was dissolved in sterile, isotonic saline (0.9% NaCl) and passed through a 0.22 μm Millipore filter (Millipore, Bedford, MA, USA) before infusion. The preparation was pyrogen-free. The actual concentration of deuterated glucose in the infusate was determined at the end of each test.

2.3 Experimental design

This was a randomised, crossover, single-blind study. A week before the tests, the subjects were asked to avoid nutrients known to be enriched in ^{13}C (corn starch and oil, cane sugar, tropical fruits and tinned foods). Twenty-four hours before the study, they were asked to limit their physical activity, not to drink alcohol-containing beverages but to eat a normal evening meal.

Each subject came to the centre on two separate days, with a wash-out of 2 wk in between, and had either the Pol meal alone (Pol) or the Pol meal with BG (Pol + BG),

selected at random. They arrived at the Centre de Recherche en Nutrition Humaine Rhône-Alpes, at Hôpital Edouard Herriot at 6.30 a.m. on the exploration day following a 12 h overnight fast.

The body weight was measured with a calibrated scale (SECA©). Intravenous catheters were inserted into deep forearm veins of both arms; for tracer infusion on one side, and blood sampling on the other. A primed-continuous infusion of D-[6,6-$^2H_2$] glucose (0.06 mg · kg^{-1} · min^{-1}) was started 120 min before the meal and was maintained during the next 360 min in order to determine the RaT. The priming dose was 80 times the infusion rate over 1 min. At time 0, subjects ate the test meal in 15 min. Blood samples were taken at the basal state and sequentially each 15 min until 60 min, then each 30 min until 360 min following ingestion of the meal. Blood samples were used to determine glucose, triacylglycerol (TG), nonesterified fatty acids (NEFA), insulin and C-peptide concentrations and D-[6,6-$^2H_2$] and ^{13}C glucose isotopic enrichments. Blood was collected in tubes maintained at 4°C and immediately centrifuged at 4500 rpm for 10 min at 4°C. Plasma was then stored at −20°C until assay. O_2 consumption and CO_2 production were monitored by indirect calorimetry (Datex Instrument, Helsinki, Finland) before (from T-60 to T0) and after test meal ingestion (from T15 to T360) in order to calculate total carbohydrate and lipid oxidation. Subjects remained in the supine position for the duration of the study except when required to void bladder (urinary nitrogen measured twice: before the meal (T0) and at the end of the experiment (T360).

2.4 Analytical procedures

2.4.1 Metabolites and hormones

Glucose, TG and NEFA concentrations were measured with an enzymatic colorimetric method on a Cary 50 Bio© spectrophotometer (Varian©) using a BioMérieux© Glucose RTU kit (Marcy l'Etoile, France), a BioMérieux TG PAP 150 kit (Marcy l'Etoile) and a WakoChemicals© NEFA-C kit (Neuss, Germany), respectively. Plasma insulin and C-peptide concentrations were determined by RIA kit (respectively Medgenix Diagnostics, Rungis, France and Immunotech, Marseille, France).

2.4.2 Indirect calorimetry

Results of respiratory exchange were integrated before and after meal ingestion and the substrate oxidation was calculated from VO_2, VCO_2 and urinary nitrogen excretion determined by chemioluminescence [20].

Total carbohydrate and lipid oxidation were calculated according to the equation developed by Ferrannini [21].

2.4.3 Isotope analysis

Plasma glucose isotopic enrichments were determined on neutral fractions of de-proteinised plasma samples, partially purified over sequential anion–cation exchange resins, as previously described [17]. Plasma [6,6-$^2H_2$] glucose was measured by organic GC-MS (Hewlett Packard 5971, Evry, France) on acetyl-bis-butane-boronyl glucose derivative, using an electron impact mode and a selective monitoring of *m*/*z* 297 and 299 [22]. Plasma ^{13}C glucose enrichment was measured by GC-combustion-isotope ratio MS (GC-C-IRMS, Isoprime, GV Instruments, Lyon, France) after derivatisation to pentacetyl glucose as previously described [23].

The ^{13}C enrichment of ingested meals (Pol and Pol + BG) was determined after enzymatic hydrolysis using the Thivend method [24] and the glucose obtained was purified by sequential anion–cation exchange chromatography before derivatisation as glucose pentacetate and analysed as previously described [25]. The ^{13}C enrichment of the derivatised glucose molecule was −34.89 $\partial^{13}C°/°°$ (1.07288 atom%^{13}C) and −35.51 $\partial^{13}C°/°°$ (1.07219 atom %^{13}C) for Pol meal and Pol + BG meal, respectively.

Table 1. Macronutrient composition of the two test meals: Pol alone and (Pol + BG)

Test meals	Pol alone (g)	(Pol + BG) (g)
Serving size	92.7	109.1
Glucose equivalent	80	80
Proteins	6.3	12.3
Lipids	0.9	4.1
Total CHO	72.3	72.3
of which fibre (without BG)	1	2.4
BG	0	5
	kcal	kcal
Energy	323	375

2.4.4 Calculations

The Rates of glucose appearance (Ra) and Rd were calculated from plasma [6,6-$^2H_2$] glucose enrichment RaT and from plasma ^{13}C glucose enrichment (RaE), using Steele's equation for nonsteady state [26, 27] as previously described [17] and the correction of Livesey *et al*. [28]. EGP was calculated as RaT – RaE.

Postprandial data were also assessed as areas under the curve (AUCs) calculated using the trapezoidal method and integrated throughout the experiment (0–360 min) and between 0 and 120 and 120 and 360 min.

2.5 Statistical analysis

The results at specific time points and AUCs were expressed as mean ± SEM.

A two-way repeated measures analysis of variance (ANOVA; meal × time) was used to determine significant main effects and interactions. When there was a significant meal × time interaction, the difference between meals at specific time points was tested using Tukey's posthoc test.

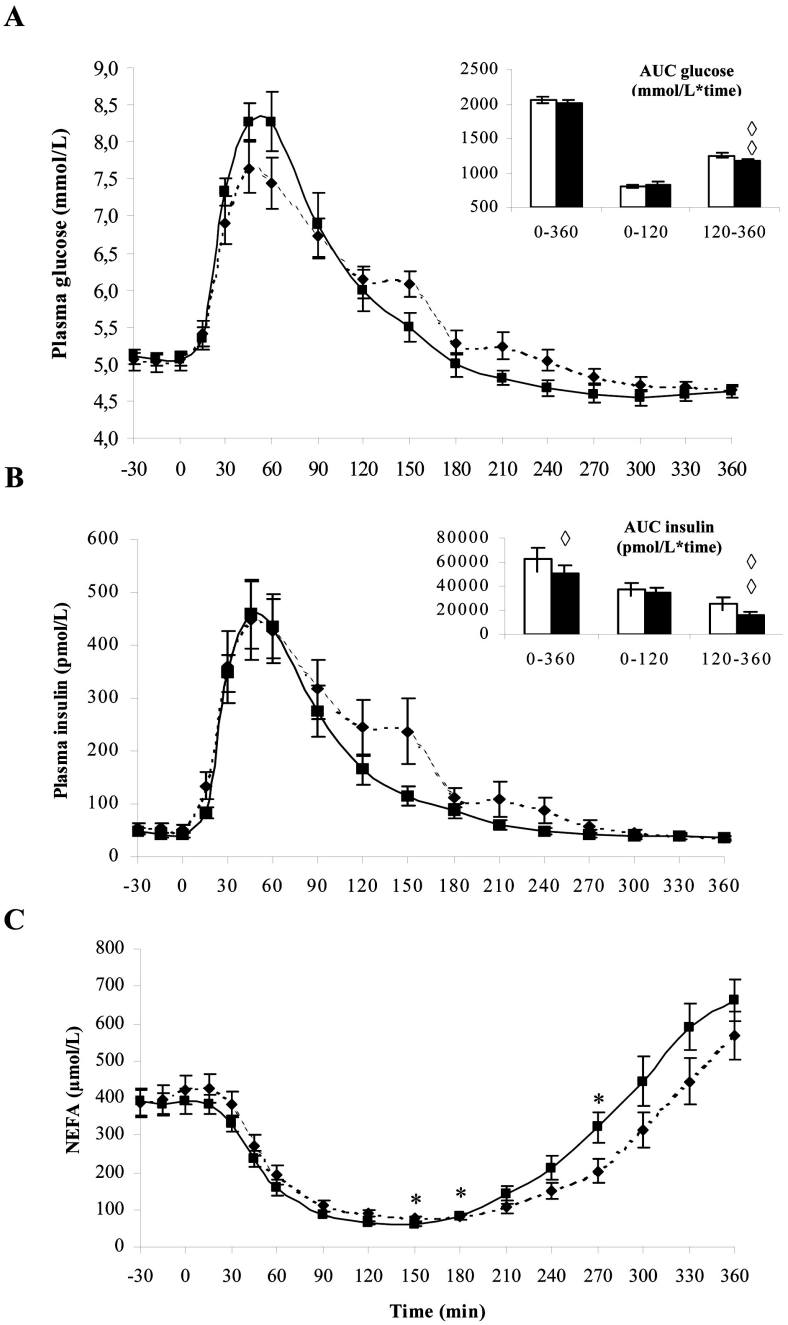

Figure 1. Plasma glucose (A), insulin (B) and NEFA (C) concentration and 0–360 min, 0–120 min, 120–360 min plasma glucose (A), insulin (B) and NEFA (C) response (AUC) after subjects ingested either the test meal with Pol alone (—, ■) or the test meal with Pol + BG (- - - ♦). Values are means ± SEM, $n = 12$. A two-way ANOVA showed a significant diet × time interaction for the glucose and NEFA responses ($p < 0.05$). At specific time points, significant difference between the 2 test meals is indicated by * ($p < 0.05$, Tukey's test). A paired *t*-test showed a significant meal effect on 120–360 min plasma glucose AUC (◇◇ $p < 0.01$) and on 120–360 min. (◇◇ $p < 0.01$) and 0–360 min plasma insulin AUC (◇ $p < 0.05$).

Significant differences between AUCs were assessed using a paired-sample *t*-test for normally distributed data.

Statistical significance was inferred at $p < 0.05$. All statistical analyses were performed using Statview v 5.0 (SAS Institute, Cary, NC) software.

3 Results

3.1 Metabolites, insulin and C-peptide responses

The response kinetics to the meals and AUC of glucose and insulin are shown in Fig. 1.

There was a significant meal × time interaction for glucose response ($p = 0.02$), which was expressed by a difference in *glucose* kinetics induced by the two meals over the 360 min after ingestion. For the first 120 min, the *glycemic* curve for the Pol + BG meal tended to be lower. But there was no significant difference in glycemic peak or in the AUC of glucose over this period. For the 120–360 min period, the positions of the curves were reversed; the *glycemic* curve following the Pol + BG meal was significantly higher. As a consequence, the area under the blood glucose curve for this 120–360 min period was also significantly different, as can be seen in Fig. 1.

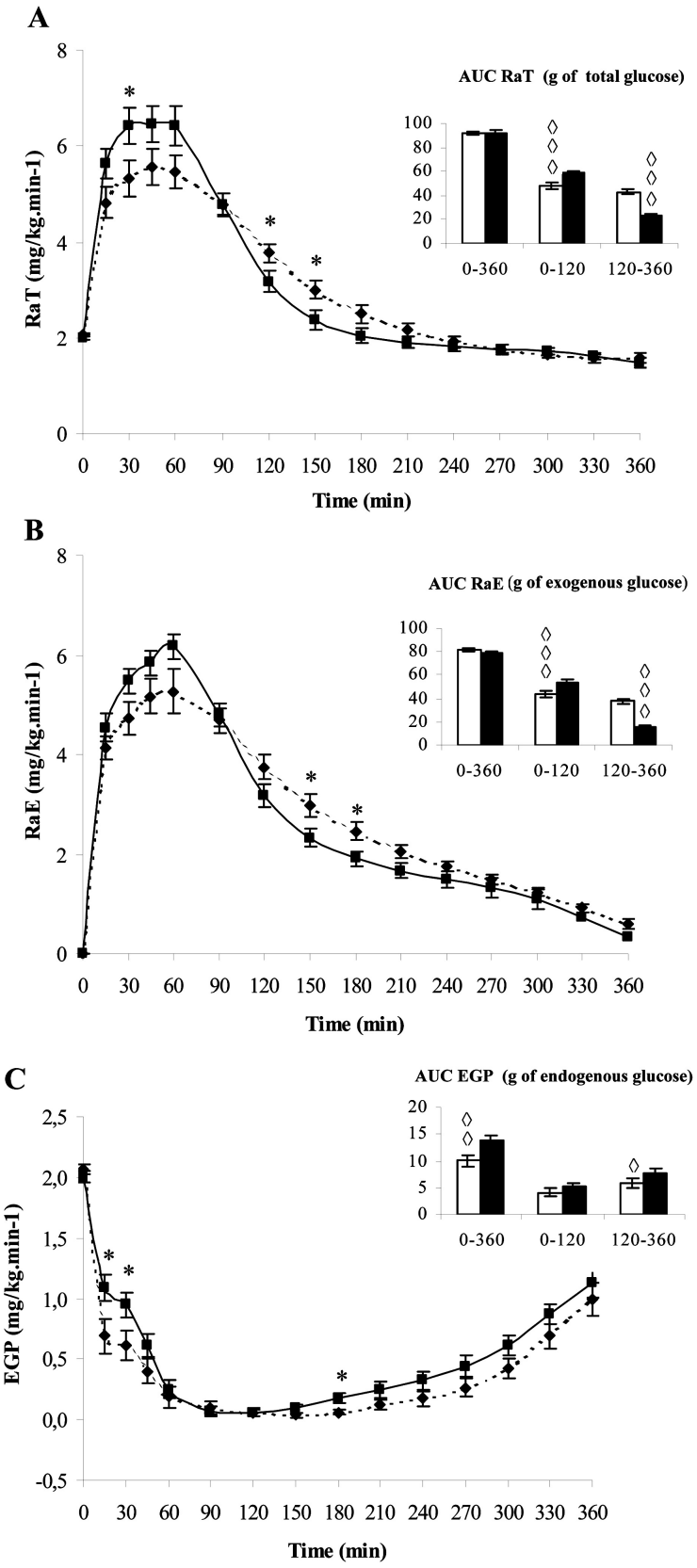

Figure 2. Rate of appearance and 360, 0–120, and 120–360 min plasma appearance (AUC) of (RaT) (A), RaE (B) and of EGP (C) after subjects ingested either the test meal with Pol alone (—, ■) or the test meal with Pol + BG (- - -, ◆). Values are means ± SEM, $n = 12$. A two-way ANOVA showed a significant diet × time interaction ($p < 0.05$) for the RaT, the RaE and the EGP. At specific time points, significant difference between the two test meals is indicated by * ($p < 0.05$, Tukey's test). A paired *t*-test showed a significant meal effect on 0–120 min and 120–360 min RaT and RaE AUCs and on 0–360 min and 120–360 min EGP AUC (◇ $p < 0.05$, ◇◇ $p < 0.01$ and ◇◇◇ $p < 0.0001$).

Considering plasma insulin response, there was neither a meal × time interaction nor a significant difference in the peak of secretion. After 90 min, insulin secretion for the Pol + BG meal was prolonged and higher, with a secondary peak at 150 min. In parallel, the AUC of insulin was significantly higher with the Pol + BG meal between 120 and 360 min ($p < 0.01$). These findings were confirmed by the C-peptide kinetics which paralleled those of the insulin, and also showed a secondary peak at 150 min (data not shown).

Plasma NEFA responses are shown in Fig. 1. There was a significant meal × time interaction for the NEFA concentrations ($p = 0.004$). First, NEFA release was less inhibited after the Pol meal. And the return of NEFA release started earlier with the Pol meal, with significant higher NEFA concentrations (T150, T180 and T270). TG concentrations were not altered by the meals (data not shown).

Thus, the Pol + BG meal tended to decrease and *prolong glycemic* response compared to the Pol meal. This was associated *with extended* insulin secretion with a secondary peak at 150 min and *prolonged* inhibition of lipolysis.

3.2 Glucose turnover: Plasma rate of appearance of total and exogenous glucose, of glucose disposal and endogenous glucose production

Figure 2 shows the changes in RaT, RaE and EGP during the 360 min after the meals. The kinetics of RaT were parallel to those of the *glycemia*, with a significant meal × time interaction ($p < 0.05$). The increase of RaT was lower after the Pol + BG meal, then the subsequent decrease was slower, so that RaT returned later to the baseline value. The Pol + BG curve was above the Pol curve after 90 min. After integration of the area under the RaT curve, we estimated the quantity of total glucose appearing in plasma over the 0–360, 0–120 and 120–360 min following ingestion. There was no difference in the quantity of total glucose appearing over the 360 min. But, according to the kinetics, less total glucose appeared with the Pol + BG meal during the first 120 min (48.11 ± 2.44 g *vs.* 58.86 ± 2.40 g, $p < 0.0001$) and between 120 and 360 min, the inverse phenomenon occurred, with more total glucose appearing with the Pol + BG diet (43.05 ± 1.51 g *vs*. 23.42 ± 1.52 g, $p < 0.0001$).

Over the 360 min, there was a significant meal × time interaction for the RaE ($p = 0.02$), but no difference in the total quantity of exogenous glucose appearing in plasma (81.59 ± 1.75 g for the Pol + BG meal *vs*. 78.55 ± 2.12 g for the Pol meal). The RaE kinetics were altered by the kind of meal ingested, as seen in Fig. 2B. The RaE was lower after the Pol + BG meal until 90 min. But after that, RaE remained significantly higher with the Pol + BG meal than it did with the Pol meal; significantly so between 150 and 180 min. Consequently, it manifested itself by a difference in the overall integrated exogenous glucose appearing in the blood when the two periods were considered separately. For the 0–120 min period, 53.98 ± 2.02 g of exogenous glucose appeared with the Pol meal, *versus* 44.10 ± 2.88 g for the Pol + BG meal ($p < 0.0001$). For the 120–360 min period, 15.70 ± 1.09 g of exogenous glucose appeared with the Pol meal, *versus* 37.49 ± 2.37 g for the Pol + BG meal ($p < 0.0001$). For the Rd, there was no significant difference between meals whether considering Rd kinetics or AUC (data not shown).

EGP is calculated by subtracting the RaE from the RaT at each timepoint. As a consequence, EGP appeared to be significantly more inhibited after the Pol + BG for the whole postprandial period, with a meal × time interaction ($p = 0.05$). Over the 360 min period, the quantity of EGP was 10.02 ± 1.10 g for the Pol + BG meal, whereas it was 13.76 ± 1.15 g for the Pol meal ($p < 0.01$).

3.3 Substrate oxidation

The total quantity of carbohydrate oxidised over the 360 min period was not statistically different between the two meals (61.06 ± 3.02 g for the Pol + BG meal *vs*. 57.06 ± 4.22 g for the Pol meal, data not shown). Moreover, no significant difference between meals was found concerning total lipid oxidation (data not shown).

4 Discussion

The added BG delayed and slowed down the absorption of glucose from Pol, as may be seen from glucose, insulin, total and exogenous glucose kinetics. This delay did not reduce the absorption of Pol. In fact the main finding was that the kinetics of total and exogenous glucose differed significantly between the meals, but not the final quantities of glucose appearing in plasma. Less total and exogenous glucose appeared during the first 120 min after the Pol + BG meal, then the phenomenon was reversed ($p < 0.0001$). The use of glucose stable isotopes allowed precise measurement of the total, exogenous and endogenous glucose kinetics so as to better understand the mechanisms of the observed effects. The quantity of exogenous glucose *appearing* in plasma during the first 120 min was 18% lower with BG. But the analysis of data from plasma glucose stable isotope enrichment for the whole test showed that there were no differences neither in the overall quantity of exogenous glucose nor in total glucose over 360 min (for Pol + BG *vs*. Pol: RaT = 91.15 ± 1.35 g *vs*. 91.61 ± 2.40 g, RaE = 81.59 ± 1.75 g *vs*. 78.55 ± 2.12 g). In the study of Battilana *et al*. [16], the addition of ^{13}C-labelled glucose to the meal containing BG showed that the appearance of exogenous ^{13}C-glucose in plasma was also 21% lower than without BG, and was associated with a modest decrease in insulin secretion. The authors concluded on delayed and also probably reduced glucose absorption with BG. In the study conducted by Schenk, the kinetics of appearance (Ra) and dis-

appearance (Rd) of glucose were studied, following the ingestion of breakfast cereals with different glycemic index. The authors showed that the different glycemic indices of breakfast cereals could be partially related to the different rates of glucose removal from blood by tissue and not to the difference in glucose appearance in plasma. This is some discrepancy with the present results, as no significant difference was found for the Rd in our study, although the Ra of exogenous and total glucose was significantly altered by BG, in parallel to the glycemic response. It should be noted however, that the two studies differed in meals composition (fibre, carbohydrate and available carbohydrate) and that in Schenk's study, the kinetics of exogenous glucose were not measured. Thus, further isotope analysis will be necessary to understand the glucose kinetics of other products (differing in glycemic index and/or fibre content and/or available carbohydrate composition), since their effect on postprandial glucose response may be related to different underlying effects on glucose kinetics.

In our study, we can quote that the final quantity of exogenous glucose which appeared in plasma was 10% higher than the quantity of exogenous glucose ingested with the test meals. This may be explained by the recycling of ^{13}C-labelled carbon during the 6 h test leading to overestimate the glucose appearing in plasma. As the same experimental conditions were used for the two meals, it did not alter our qualitative analysis but should be taken into account for further quantitative analysis.

As for the insulin and glucose kinetics, it seemed sensible to split the responses to the meals in two phases with different metabolic profiles: from 0 to 120 min and from 120 to 360 min after ingestion. During the first 120 min, there was a trend for the *glycemic* response to be lowered by BG, but this was not significant. Over the same time period, even if the insulinaemic response was not significantly altered in intensity by fibre ingestion, the decrease in insulin secretion was delayed with the BG. In parallel to a lower appearance of exogenous glucose (44.10 ± 2.88 g for the Pol + BG meal *vs*. 53.98 ± 2.02 g for the Pol meal), the appearance of total glucose in plasma was also reduced (48.11 ± 2.44 g for the Pol + BG meal *vs*. 58.86 ± 2.40 g for the Pol meal) ($p < 0.0001$). This confirms our finding of a trend to a lower *glycemia* before 120 min. After 120 min, metabolic responses were different. With BG, the *glucose* response returned to baseline more slowly. In the same way, insulin secretion was sustained with a second peak at 150 min, which was probably parallel to the exogenous glucose appearing to a greater *extent* in plasma after 120 min after BG ingestion and indicating a prolonged glucose absorption.

Several studies in healthy and diabetic subjects have shown that there are decreased postprandial glucose and insulin responses following the ingestion of BG, linked to a decreased nutrient absorption rate [1–4, 12–14]. The present work did not exactly induce the same effect *in term of parallel glucose and insulin responses*; such discordances between glucose and insulin responses have also been found in other studies. For example, in the study of Juntunen *et al*. [15], the ingestion of 50 g rye bread containing 5.4 g of BG (3% total meal weight) reduced the insulinaemic response without a parallel reduction in the *glucose* response. In the study carried out by Bourdon *et al*. [4], the subjects ingested either enriched barley pasta (containing 5 g of BG) or normal pasta. No effect on 0–120 min *glycemia* was noticed, even though insulinaemia was reduced, in parallel with a decreased absorption rate. But, interestingly, the breakdown of the responses into two phases, before and after 120 min, like in our study, showed differences in glucose response kinetics, indicating the importance of studying metabolic responses for more than 2 h. Different studies underlined that the acute postprandial glucose and insulin responses (within 120–180 min. from meals) are among the main determinants of the risk of cardiovascular disease in healthy and diabetic subjects [29–32]. This study also demonstrated the difficulty of focusing and concluding only on the effect of BG fibre, when part of a meal's matrix. Concerning the amount of BG ingested, it has been demonstrated that the reduced glucose response was achieved by increasing the quantity of fibre (in most cases ranging from 5 to 10 g), linearly to the viscosity [2, 3, 33, 34]. Biorklund *et al*. [12] showed a significant reduction in postprandial *glucose* response after the consumption of a beverage enriched with 5 g of oat BG for 8 wk, but not with barley BG. These reported discrepancies between intervention studies could be due to differences in study design, such as the duration of the dietary intervention, the quantity and form of BG, the chosen food matrix, the presence of others nutrients, the test population, whether healthy, overweight or diabetic. Several authors have shown that proteins in combination with carbohydrate intake could induce an increase in insulin secretion and also differed in their ability to stimulate insulin secretion, according to their nature, structure or structure form in food matrix [35–37]. The BG preparation added to Pol contained proteins (diets were matched for carbohydrate content), which could have had a synergistic effect on insulin secretion and could therefore have modulated or compensated the effect on the sole BG on insulinaemia. Moreover, our population was normo-glycemic and since *glycemia* is a well-regulated parameter, adding 5 g of BG to a single test-meal may not have been sufficient to affect significantly the 6 h area under the glucose curve. But the reduction of the 0–120 min rate of glucose absorption could be of interest, because of the link between acute postprandial glucose response and cardiovascular risks [29–32]. The overweight subjects also showed heterogeneity in postprandial insulin secretion in response to the meals, and this could have blurred the effects of BG on the insulin response, more particularly during the first postprandial phase (higher SD).

There was no difference in total lipid or carbohydrate oxidation between the two meals, suggesting that 5 g of BG

could not alter the overall nutrient oxidation pattern over the 360 min. This was in accordance with the literature concerning BG [16] or *glycemic* index and *nutrient* oxidation. In a review compiling several studies about *glycemic* index and fuel partitioning, Diaz *et al*. [38] showed that other short-, mid- or long-term interventions failed to modify fuel partitioning. Similarly, in a study of obese women, ingestion of either a low or high *glycemic* index breakfast induced a 1.8-fold change in the AUC of serum insulin which was not enough to influence fuel oxidation over 5 h [39].

Consequent to the delay in glucose absorption which prolonged the *glycemic* and insulinemic responses to the BG containing meal, the longer insulinaemic response had a prolonged inhibitory effect on the EGP throughout the test ($p < 0.001$). After BG ingestion, EGP was lower throughout the 360 min of the test. This is an interesting finding, since the reduction of the postprandial EGP could decrease the postprandial *glycemic* response in our overweight population at higher risk of developing type 2 diabetes mellitus. A longer inhibition of lipolysis was observed after the Pol + BG meal, as the plasma NEFA concentration, which is very sensitive to insulin variations [40], tended to have a delayed return to baseline values after the Pol + BG meal ($p < 0.05$).

In conclusion, over the 360 min study, the quantity of total and exogenous glucose which appeared in the plasma was the same; but the kinetics of their appearance differed, as well as *glycemic* response. The addition of BG delayed exogenous glucose absorption, resulting in a longer-lasting insulin secretion that exerted a prolonged inhibition of EGP and lipolysis. This study confirmed the importance of a longer follow-up of metabolic parameters to *capture relevant* long-lasting metabolic changes; in fact the 0 – 120 min AUC for insulin secretion was not significantly different between the meals. The use of glucose stable isotopes was of prime importance in detecting the delayed, but not reduced, BG-induced absorption underlying the postprandial glucose and insulin response. These mechanisms may modulate the postprandial metabolic responses to a BG-enriched meal and should be taken into account in the context of BG enriched product development.

We thank all the staff of the Centre de Recherche en Nutrition Humaine Rhône-Alpes and more particularly Christine Maitrepierre, Jocelyne Peyrat, Corinne Louche-Pélissier and Monique Sothier for their excellent technical assistance in subject recruitment, sample collection and analysis, and dietary surveys, and all the subjects for taking part in this trial. We also thank Mickael R. Skilton and Lynn Richardson for their helpful assistance in translation/revison of the manuscript. Beta-glucan fiber was produced by Oatly AB, Landskrona, Sweden. This study was part of the European project, contract No. QLK1-CT-2000-00535, supported and funded by the European Union.

Conflict of interest statement: The beta-glucan fiber was produced by Oatly AB, Landskrona, Sweden. A. O. Triantafyllou is employee of Oatly AB.

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