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The Effect of a Pre-Load Meal Containing Resistant Starch on Spontaneous Food Intake and Glucose and Insulin Responses

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Abstract: The effects of resistant starch and dietary fiber on food intake, satiety and postprandial metabolic responses remain controversial. To assess the effect of a meal containing resistant starch on food intake, satiety, glucose and insulin responses. Twenty-two healthy subjects (13 male, 9 female; age 26±4 year; BMI 23.7±2.4 kg m^{-2}) undertook 5 study meals, taken in random order, consisting of cereal bars and beverages; control meal taken twice (0 g Resistant Starch (RS)) and three doses of RS (5, 10 and 25 g). Subjects rated their satiety level and symptoms and blood samples were collected prior to and at 15, 30, 45, 60, 90 and 120 min after consuming the study meals. Postprandial glucose and insulin levels were measured. Using the satiety ratings, the satiety quotient and appetite score were calculated. Two hours after eating, subjects were given an *ad libitum* meal and total energy intake was determined. There were no significant treatment differences in the incremental Area Under the Curve (iAUC) for glucose or insulin. However, at 90 and 120 min the incremental blood glucose and insulin levels after 25 g RS were significantly lower than that of the control ($p = 0.004$ and $p = 0.001$ for glucose, $p = 0.043$ and $p = 0.042$ for insulin, respectively). Feelings of fullness were greater with the 5 g dose of RS compared to the control, while the satiety quotient for overall appetite was significantly greater for 25 g RS in the early phase after the eating episode. The present study indicates that a meal containing RS may decrease postprandial glucose and insulin responses and enhance subjective feelings of satiety.

Key words: Resistant starch, dietary fiber, food intake and glucose, food intake and insulin, satiety, satiety quotient

INTRODUCTION

Over the past 3 decades, obesity rates have increased dramatically in both the developed and developing nations (Ogden *et al*., 2006). Obesity is a risk factor for type 2 diabetes and Coronary Heart Disease (CHD) (Lopez *et al*., 2006) and much research is underway to investigate dietary factors that may increase satiety and decrease food intake. It is now well established that foods differ in their satiating capacity and that this can in part, be ascribed to their nutritional composition (Blundell and Burley, 1987). Satiating capacity or efficiency has been defined as the capacity of a consumed food to suppress hunger and decrease subsequent food intake (Livingstone and Robson, 2000). Recently, a satiety quotient was developed to better assess the satiating ability of foods (Green *et al*., 1997). The satiety quotient is calculated by dividing the difference between the subjective ratings of motivation to eat before and after a meal by the ingested energy intake during the eating episode. This relates energy intake to the rate of return of motivation to eat during an eating episode and may therefore be more effective when measuring short term appetite control (Green *et al*., 1997).

Available carbohydrates and dietary fiber have been given much attention when it comes to studying the satiating capacity of foods. This is in part due to the fact that carbohydrates serve as the major source of energy in our diet. According to the glucostatic theory, blood glucose levels may also be involved in the regulation of appetite (Mayer, 1953).

This theory suggests that high blood glucose levels lead to satiety and termination of feeding, while low blood glucose levels trigger hunger and subsequent food intake (Mayer, 1953). In support of this theory, several studies have shown that short-term food intake and appetite are inversely associated with blood glucose response (Rogers and Blundell, 1989; Blundell *et al*., 1994;

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Anderson, 1995; Woodend and Anderson, 2001; Anderson *et al*., 2002). However, others have suggested that lower and slower glucose and insulin responses promote satiety. The reasoning being that a sharp post-meal increase in serum glucose and insulin levels is followed by a sharp decline, which may lead to hypoglycemia and increased hunger. Evidence support the effect of low-Glycemic Index (GI) foods, which are foods that lead to a lower and slower increase in blood glucose and insulin levels following a meal, on satiety (Ludwig, 2000, 2002; Roberts, 2000). Similarly, research indicates that foods that are high in fiber are more satiating (Ludwig, 2000; Anne *et al*., 2006).

Resistant starch is a dietary fiber that has been suggested to enhance satiety, yet few studies have been done in this area and the results to date have been inconsistent (Raben *et al*., 1994; Willis *et al*., 2009). A reason for this inconsistency may be due to different doses used in different trials. Resistant starch can occur naturally in foods such as cooked potatoes and lentils and it can also be manufactured and added to foods to increase fiber content. As more consumers are looking for healthier food choices and foods that will help them feel fuller for longer, it is important to determine if adding resistant starch to food items will promote satiety and reduce subsequent meal intake. Furthermore, if an effect is observed, it is important to determine, whether the effect is dose dependant. In this study, we evaluated a new, heat stable resistant starch available on the market, at three different doses to determine its effect on postprandial glucose, insulin, satiety and subsequent meal energy intake.

MATERIALS AND METHODS

Subjects: A total of 22 subjects (13 male, 9 female), aged 26±4 year (mean±SD) with a Body Mass Index (BMI) of 23.7±2.4 kg m^{-2} participated in the study. Subjects were healthy and were not taking any medications that would interfere with glucose metabolism. In order to determine whether the subjects were unrestrained eaters an eating habits questionnaire was used (Herman and Polivy, 1980). This evaluation separates people into restrained and unrestrained eaters. Whereas restrained eaters are thought to let cues other than satiety/satiation determine the cessation of eating, unrestrained eaters are thought to rely more on physiological cues.

Study protocol: Subjects were recruited from the clinic volunteer roster. All subjects were screened prior to the start of the study and underwent treatments on separate days, with each subject not performing >1 test $week^{-1}$. The study used a randomized, double blind crossover design in which subjects acted as their own control.

On each test day, subjects came to the clinic in the morning after a 10-14 h overnight fast. After being weighed and having recorded their level of satiation by answering 4 questions using a visual analogue scale (motivation to eat questionnaire), a finger-prick blood sample was obtained for glucose and insulin analysis. Subjects then consumed the test meal within 10 min and palatability was recorded. Subjective appetite was assessed by a motivation to eat questionnaire at 15, 30, 45, 60, 90, 120 and 150 min after the start of the test meal. Blood samples were taken at 15, 30, 45, 60, 90 and 120 min. Subjects were also asked to fill out a physical comfort questionnaire. At 120 min subjects were seated separately and served 2 small pizzas, 3 times at 10 min intervals. Pizzas were served in quarters and subjects were instructed to eat as much as they desired until they were comfortably full. Subjects were also given a bottle of 1.5 L mineral water. Weighed intakes of food and water were recorded.

Test meals: Three test meals containing 5, 15 and 25 g of RS and two control meals with 0 g of RS each consisting of a cereal bar and beverage were assessed. All products were provided by Tate and Lyle Ingredients America, Decatur, IL. The resistant starch used was PROMITOR™ Resistant Starch (Tate and Lyle Ingredients America, Decatur, IL). This is a RS3 type resistant starch produced from heat-moisture treated high amylose maize starch. It contains 58% fiber by AOAC method 991.43 and has an average MW of 100,000 Da and is very heat stable (up to 115°C). The energy of each meal and the amounts of the powder and water used to prepare each beverage are summarized in Table 1. The subsequent meal 'lunch' consisted of small pizzas (Deep 'N Delicious, McCain Foods Canada, Florenceville, NB) baked in the oven at 425°C for 10 min according to the manufacturer's instructions. Food intake was measured by weighing each plate of food immediately before it was served and then weighing it again after the subject had finished eating. Water was served in bottles, which were weighed before and after consumption. The energy consumed over lunch was calculated by determining the weight of food consumed and using the macronutrient profile of the pizzas provided by the manufacturer.

Measurements

Blood samples: Each finger-prick sample consisted of a total of 8-10 drops of blood obtained by finger-prick and divided into two separate vials. Two to three drops of capillary blood were collected into flat-bottomed 5 mL plastic tubes with a push cap containing a small amount of sodium fluoride and potassium oxalate as an anticoagulant and preservative. These samples were

Table 1: Test meal energy content and product information

Test meal	Energy (k cal)	Beverage powder (g)	Water added to beverage (g)	Water consumed (mL)
Control	367	0.7	224	150
Resistant starch (5 g)	356	3.2	226	150
Resistant starch (15 g)	355	13.2	224	150
Resistant starch (25 g)	354	23.1	222	150

used for analyzing capillary blood glucose levels. The remaining 6-8 drops of capillary blood were collected into a microvette CB300 (Sarsted) vial, which were used for insulin analysis.

Biochemical analysis: The finger-prick samples for glucose analysis were initially placed in a refrigerator and at the end of 2 h, placed in a -20°C freezer until analysis, which was performed within 1 week of collection. Glucose analysis was performed on a YSI model 2300 STAT analyzer (Yellow Springs, OH). The microvette tubes were centrifuged and serum transferred to labeled polypropylene tubes and stored at -20°C prior to analysis of insulin. Insulin levels were measured using the Human Insulin EIA Kit (Alpco Diagnostics).

Motivation to eat and average appetite: Subjective measurements of motivation to eat and physical comfort were assessed using Visual Analog Scales (VAS). Each of the questions on the VAS was a 100 mm line anchored at each end with opposing statements (Rogers and Blundell, 1979). Subjective appetite was assessed using the motivation to eat questionnaire, which included 4 questions:

Q1: How strong is your desire to eat? (very weak to very strong).

Q2: How hungry do you feel? (not hungry at all to as hungry as I have ever felt).

Q3: How full do you feel? (not full at all to as full as I have ever felt).

Q4: How much do you think you could eat now? (nothing at all to a large amount).

Average appetite scores were calculated as a summary measure using the following equation:

$$\text{Average appetite} = \frac{[Q1 + Q2 + Q4 + (100 - Q3)]}{4}$$

The satiety quotient was calculated using the formula by Green *et al.* (1997):

$$\text{Satiety quotient} = \frac{\text{Rating pre-eating rating post-eating episode}}{\text{Caloric intake of eating episode}}$$

Palatability: Palatability was rated on a 100 mm visual analogue scale anchored at very unpalatable at one end and very palatable at the other one. Therefore, the higher the number the higher was the perceived palatability of the product.

Subjective physical comfort: Physical comfort was assessed using a similar VAS as subjective appetite. However, participants marked either yes or no after each marker of physical comfort. If they marked yes they were instructed to rate the severity of the side effect on a 100 mm VAS and to provide any comments they felt were necessary. The physical comfort scale includes bloating, belching, diarrhea, flatulence, nausea, headache and a category for other.

Statistical analysis: All data are presented as mean±SEM, unless otherwise indicated. Incremental areas under the plasma glucose curves (iAUC) were calculated using the trapezoid rule and ignoring area beneath baseline. iAUC values were subjected to Analysis of Variance (ANOVA) examining for the effect of the test meal with the random variable representing individual subjects to denote the crossover nature of the study design. To estimate the iAUC of a 25 g glucose tolerance, the conversion factor of 0.6174 was applied. Since no significant heterogeneity was found, no further tests pairing the different levels of fiber intake were undertaken. Since, for neither the insulin nor satiety quotient iAUC did ANOVA result in rejection of the overall null hypothesis of no treatment effect, no further comparisons were assessed. Pearson moment-product correlation was also employed to assess the linear associations among AUCs, fiber dose and palatability. Data were considered significantly different at $p<0.05$.

RESULTS AND DISCUSSION

Palatability and symptoms: The RS study meals were rated as being less palatable than the control meal (Table 2). However, all of the meals were well tolerated, even at the highest RS dose (25 g) and no significant differences were found between the study meals in reported symptoms (belching, bloating, diarrhea, flatulence or nausea). All scores were very low on the symptom scale and were not considered clinically significant. The current acute study supports studies in

Table 2: Palatability of the test meals and their effect on postprandial glucose insulin and second meal energy intake

Test meal	Palatability (mm)	Glucose iAUC (mmol/min/L)	Insulin iAUC (Mu/min/L)	Second meal energy intake (k cal)
Control	60±4[a]	208.8±18.7	3287.8±453.6	755±97
Resistant starch (5 g)	48±5[b]	208.8±19.2	3386.7±681.4	755±101
Resistant starch (15 g)	51±4[b]	237.0±28.6	4018.4±647.2	834±106
Resistant starch (25 g)	34±5[c]	198.4±20.2	3082.2±392.3	867±103

Values are means+SEM. Values within a couple not sharing a common superscript are significantly different ($p<0.05$)

which relatively high intakes of RS (30 g day^{-1}), in the form of cereal and muffins, were well tolerated for 2 weeks periods even though fecal bulk and short chain fatty acid production were significantly increased (Jenkins *et al.*, 1998).

Blood glucose and insulin: There were no significant differences between treatments in the iAUC for blood glucose or insulin (Table 2). However, consumption of the highest dose (25 g) of RS generally resulted in lower blood glucose and insulin levels when compared to the other meals (Fig. 1).

These differences reached statistical significance later in the post-meal period (60-120 min). At 90 min the incremental blood glucose level after the 25 g RS dose was significantly lower compared with the 15 g RS and control meals ($p = 0.035$). In addition, the highest dose of RS resulted in a significant reduction in postprandial blood glucose levels at 90 and 120 min compared with the control (Fig. 1).

For the insulin response curve, the 25 g RS dose resulted in lower serum insulin concentrations, being significantly lower by comparison to the 15 g RS dose at 60 min ($p = 0.035$) and also to the control meal at 90 and 120 min (Fig. 2). The reduction in the glucose and insulin response curves only with higher levels of RS supports the findings of other studies (Behall and Hallfrisch, 2002; Higgins, 2004). For example, Behall and Hallfrisch (2002) showed that low doses of RS are often insufficient to reduce blood glucose and insulin and that a significant reduction may only be achieved when the RS content reaches as high as 50%.

Furthermore, another study found that the addition of low concentrations of RS (~8 g) had no effect on the GI (a marker of postprandial glycemic response resulting from ingestion of a carbohydrate food) of breakfast cereals or muffins (Jenkins *et al.*, 1998). It would therefore appear that higher doses of RS are required to lower the glycemic and insulinemic response curves acutely. Evidence also suggests that RS can have a positive effect on insulin sensitivity, thereby increasing glucose uptake from the blood stream (Robertson *et al.*, 2003, 2005). This may also explain the significant reduction in postprandial glucose that occurs later in the post-meal period.

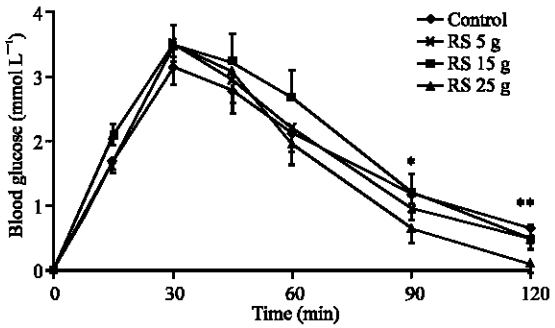

Fig. 1: Effect of RS dose on postprandial incremental blood glucose response; Significant differences between 25 g RS and the control (0 g RS) are presented: *p = 0.004; **p = 0.001

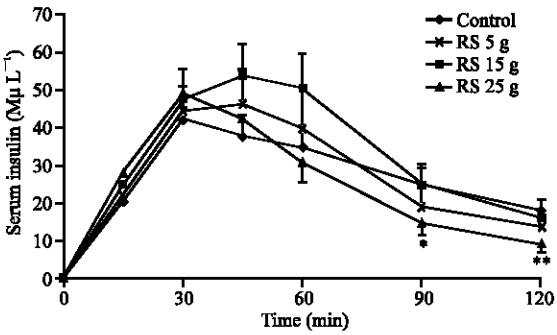

Fig. 2: Effect of RS dose on postprandial incremental serum insulin response. Significant differences between 25 g RS and the control (0 g RS) are presented: *p = 0.043; **p = 0.042

Motivation to eat, satiety quotient second meal food intake: The 5 g RS dose was significantly different than the control for the Satiety Quotient (SQ) for Q3 indicating enhanced fullness 60 min after the test meal ($p<0.04$; Table 3). In addition, the SQ for overall appetite score was increased at 15, 30 and 45 min after the 25 g RS meal compared with the control ($p = 0.097$, 0.075 and 0.036, respectively). Furthermore, the average appetite SQ over the 2 h post-meal time period was greater than the control group although this only approached significance ($p = 0.137$) (Fig. 3).

These results indicate that 25 g RS triggers an initial but transient increase in feelings of satiety >5 and 15 g

Table 3: Effect of resistant starch dose on the satiety quotient (n = 19)

Questions	Control	Resistant starch (5 g)	Resistant starch (15 g)	Resistant starch (25 g)
Desire to eat?	9±2	7±2 (p = 0.455)	8±2 (p = 0.729)	10±2 (p = 0.440)
How hungry?	8±2	8±2 (p = 0.698)	8±1 (0.755)	9±2 (0.663)
How full?	-9±2	-6±2 (p = 0.040)*	-7±1 (p = 0.137)	-10±2 (p = 0.883)
How much can you eat?	6±2	7±2 (p = 0.953)	7±2 (p = 0.667)	7±2 (p = 0.730)

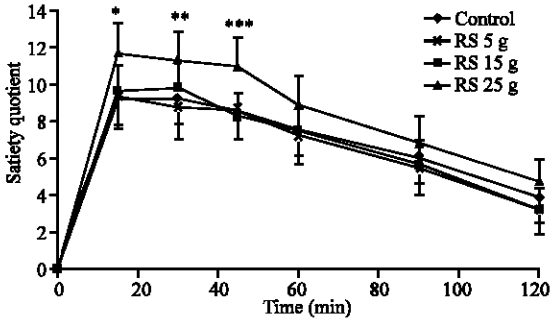

Fig. 3: Effect of RS dose on the mean appetite Satiety Quotient (SQ) for the 2 h post-meal period. For the 25 g RS dose vs. control (0 g RS): *p = 0.097; **p = 0.075; ***p = 0.036. The difference in SQ area under the curve over the 2 h post-meal period approached significance (p = 0.137)

doses. Feelings of fullness were enhanced with the 5 g dose, however, this was only at one time point and none of the other measures (i.e., hunger, desire to eat, overall appetite) were affected. After 45 min the appetite SQ for the 25 g dose, while remaining elevated, did not differ from the other treatments, which may explain why there was no difference in later meal intakes. The transient increase in SQ with no resulting change in meal intake could also be related to the glycemic response. At 60 min, there is a fairly steep drop in blood glucose with the 25 g dose, which then remains lower than all other treatments, until 120 min.

Similarly, at 60 min there is a drop in the satiety quotient. By 120 min, the blood glucose level for the 25 g dose is significantly less than the control. This is in agreement with the glucostatic theory that a drop in glucose may be a trigger for hunger and hence, why there was a decrease in satiety and ultimately no difference in meal intake. However, other studies show that glycemic responses following a pre-load are not related to hunger and satiety (Holt *et al*., 1992, 1996; Granfeldt *et al*., 1994; Lavin *et al*., 1996; Flint *et al*., 2006). In this study, the glycemic response at the 25 g dose drops below the other treatments at 60 min, however, the corresponding drop in the SQ does not fall below the other treatments but remains elevated for the duration of the measurement period. Therefore, other mechanisms beyond the glucose response, such as increased bulk from undigested fiber in the intestine, influence on satiety hormones, or fermentation in the colon (Slavin and Green, 2007), may be responsible for keeping the SQ slightly elevated over the other treatments.

Overall, there is much inconsistency in the findings. Two studies have shown that high doses of RS (e.g., 30 g) have no effect on satiety by comparison to other foods and fibers (Raben *et al*., 1994; De Roos *et al*., 1995), while another has shown that while 48 g of RS may not effect self-reported appetite, it did significantly lower 24 h food intake (Bodinham *et al*., 2008). The potential effect of palatability on satiety and food intake along with study design differences may have resulted in the different results reported.

CONCLUSION

In the present study, RS at levels of 5-25 g, were found to be well tolerated and did not result in any symptoms when incorporated into test foods, cereal bars and beverages. In addition, the highest level of RS intake generally resulted in lower postprandial glucose and insulin responses, which reached statistical significance later in the response curves. The satiety quotient for overall appetite was greater with the 25 g dose of RS for 45 min following the eating episode indicating an early but transient increase in satiety. Additionally, there was a trend to increased satiety over a 2 h period. This finding suggests that the satiety quotient, which accounts not only for subjective feelings of satiety but also the caloric intake, may be a more effective method of measuring short-term appetite control. This data also suggests there may be a relationship between glycemic response and hunger as a sharp drop in blood glucose from 45-60 min at the 25 g dose concomitantly showed a decrease in satiety quotient.

However, the glycemic responses to these different study products were not different enough to draw definite conclusions between glycemic and insulinemic response and satiety. While, this study did not show changes in subsequent meal intake, the changes in subjective feelings of hunger and fullness suggest that the role of RS in weight management and appetite control deserve further study. More acute studies, looking at higher doses of RS are required to determine whether a dose response relationship exists. Furthermore, long-term randomized clinical trials are required to determine, whether there is a link between intake of resistant starch, satiety and obesity related markers such as, weight and fat mass.

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