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Positive impact of a functional ingredient on hunger and satiety after ingestion of two meals with different characteristics

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ABSTRACT

The ingestion of unavailable carbohydrates – functional ingredients – has presented an inverse relationship with the risk for chronic non-communicable diseases. The objective of this work was to evaluate the effects of addition of inulin to two ready-to-eat frozen meals on the release of gastrointestinal hormones and other parameters related to hunger and satiety. Prototypes of two different kinds of frozen meals were elaborated by the food industry: control meal (C1 and C2); and test meals, added inulin (T1 and T2). Three sequential clinical assays were performed with healthy volunteers: 1) evaluation of glycemic response (n = 16); 2) evaluation of gastrointestinal hormones related to satiety (n = 15); and 3) evaluation of satiety (by Visual Analogue Scale — VAS and energy intake) (n = 52). The meals showed low glycemic index and glycemic load, and T1 showed a decreased glycemic response peak compared to C1. The addition of inulin (~8 g) to the test meals (lunch) provided significant satiety, resulting in an decrease in energy intake of 419 (group 1) and 586 kJ (group 2) in the two subsequent meals (after 180 min and 360 min) and a decrease in hunger and increase in satiety at 120 and 180 min when comparing with control meals. A positive post-prandial variation was observed in the plasmatic levels of ghrelin and insulin in relation to the control meal (hormones related to hunger in high levels), after the intake of both two test meals. Inulin is an ingredient that presents several positive characteristics for the elaboration of products that stimulate healthy eating habits. These effects are currently being evaluated in medium-term trials.

1. Introduction

The energy balance can be regulated by the size and frequency of meals, which in turn respond to a number of biomarkers related to the digestive tract, such as ghrelin or to the energy reserve, such as insulin and leptin (Woods & D'Alessio, 2008). Thus, meals that act on the suppression of these hormones can contribute to the energy balance.

The appetite, central point of the energy balance, can be divided into three components: hunger, satiation and satiety. Hunger is related to the sensations that promote the consumption of food and involves metabolic, sensory and cognitive factors. Satiation is related to the decrease of appetite, and can be measured by the duration or size of the current meal (Mattes, Hollis, Hayes, & Stunkard, 2005). Satiety is related to the next meal, and may reduce its volume or decrease the time interval between them, those being some of the satiety parameters assessed (Burton-Freeman, 2000; Mattes et al., 2005).

The brain control on energy intake is a response to sensory and cognitive perception of the food consumed and gastric distension (Benelam, 2009). The hypothalamus is the structure of the nervous system responsible for the control of food intake (short-term regulation of hunger and satiety) and body weight (long-term regulation). It receives many signals in the form of hormones such as ghrelin that stimulates hunger, and adrenaline, insulin, cholecystokinin, leptin and PYY protein that stimulate satiation and/or satiety.

Foods that contain unavailable carbohydrates have shown greater efficacy in satiety control, insulin resistance and improve plasma levels of glucose, insulin and lipids (Cani, Joly, Horsmans, & Delzenne, 2006; Jakobsdottir, Nyman, & Fak, 2014; WHO/FAO, 2003). Fructans, such as inulin, are considered functional ingredients because they are associated with favorable effects on metabolism such as absorption of minerals and change in gastrointestinal hormones related to the control of food intake, and blood levels of glucose and insulin (Nair, Kharb, & Thompkinson, 2010). Certain types of dietary fiber (DF) present in food can contribute to a reduced response in blood levels of glucose and insulin, and to satiety as well, due to its viscosity and low energy density, in addition to promoting satiation by gastric distension (Slavin, 2013).

The objective of this work was to evaluate the effects of the addition of inulin to ready-to-eat frozen meals on the release of gastrointestinal hormones and other parameters related to hunger and satiety.

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2. Materials and methods

2.1. Studied meals

The four meals studied were developed in a pilot plant, semi-industrial scale, by BRF SA (São Paulo unit), according to good manufacturing practices (Brasil, 2014). Microbiological analyses were performed, and they were according to all microbiological limits established by RDC 12/01 from ANVISA (Brasil, 2001). The control (C1 and C2) and test meals (T1 and T2) with ~8 g of inulin, contained the following ingredients: C1 — chicken breast fillet, herb sauce, polished rice with carrots and green beans, creamed corn, and boiled carrots; T1 — C1 added inulin; C2 — wholewheat fusilli pasta, Bolognese sauce with textured soy protein and vegetables; and T2 — C2 added inulin. The meals were stored at −20 °C until the moment of consumption.

2.2. Carbohydrates and proximal composition analysis

Frozen samples (n = 3 from three different lots) were lyophilized (Freeze Dryer, model Super Modulyo 220 TC60 Tray Cell, Thermo Fisher Scientific, Waltham, MA, USA), had the lipids removed and were ground to 250 μm for chemical analysis. The analyses of proximal composition were conducted in triplicate and were determined according to the A.O.A.C. methods (Horwitz & Latimer, 2006); the results were expressed as g/100 g of wet weight (w.w.). DF was quantified by the enzymatic–gravimetric method according to the A.O.A.C. method 991.43 (Lee, Prosky, & Devries, 1992) with modifications. The modifications were proposed in order to exclude fructans from the dietary fiber fraction (McCleary & Rossiter, 2004). Fructans were determined according to the A.O.A.C. method 999.03 (McCleary, Murphy, & Mugford, 2000) using a Megazyme fructan kit (Megazyme International Ireland Ltd., Wicklow, IRL). Total unavailable carbohydrate was determined as the sum of DF (without fructans), fructans and resistant starch.

TS was determined after starch solubilization, precipitation and hydrolysis, with amyloglucosidase (Sigma A-7255, 28 U/mL — Sigma Chemical Co., St. Louis, MO, USA) (Cordenunsi & Lajolo, 1995); wheat starch (Sigma-1514 — Sigma Chemical Co., St. Louis, MO, USA) was used as a standard reference material (SRM). RS analysis was conducted by the AOAC 2002.02 method (McCleary, McNally, & Rossiter, 2002); a sample of boiled beans was used as an in-house reference material. Available starch was calculated as the difference between the content of TS and RS.

Soluble sugars were extracted and analyzed by HPAE-PAD (Dionex Corporation, Sunnyvale, CA, USA). The analytical column employed was a Carbopac PA1 (250 × 4 mm, 5-μm particle size). The mobile phase was 18 mmol NaOH and the flow rate was kept constant at 1.0 mL/min. Injections (25 μL) were made using an AS 500 autosampler. Glucose, fructose, galactose, maltose and sucrose were used as SRM (B. Cordenunsi, Shiga, & Lajolo, 2008).

2.3. Determination of the glycemic response

The blood glucose level was determined (triplicate) by the enzymatic method (glucose oxidase/peroxidase) in capillary blood (Brouns et al., 2005). For the capillary puncture the Accu-Chek® Softclix® Pro-Roche lancing device with a disposable lancet was used (Accu-Chek® Softclix® Pro-Lancet Roche — Roche Diagnostics®, Indianapolis, IN, USA), which was replaced at every blood collection. The device for glucose quantification used was Accu-Chek® Go-Roche (Roche Diagnostics®, Indianapolis, IN, USA).

2.4. Determination of gastrointestinal hormones

Blood samples were collected in Vacutainer® tubes containing EDTA. Next, blood aliquots (1 mL) were transferred to Eppendorf tubes containing [4-(2-aminoethyl)-benzenesulfonyl fluoreide] (Pefabloc®, Sigma Aldrich, Switzerland) (1 mg/mL of blood) and were centrifuged (800 *g*) at 4 °C, for 15 min. Plasma was stored in an ultra-freezer until the analysis. The values were expressed in pg/mL of plasma or serum. The hormones ghrelin and insulin were analyzed (duplicate) through specific LINCOplex® kits (Linco Research Inc., St Charles, MO, USA) according to Luminex™ xMAP technology (Luminex Corporation, Austin, TX, USA).

2.5. Clinical assays

2.5.1. Participants

All assays were performed with healthy volunteers, aged between 18 and 40 years and BMI between 18.5 and 24.9 kg/m^2 (WHO, 1997). Individuals with a previous diagnosis of diabetes mellitus or family history, renal and gastrointestinal diseases, hyperthyroidism, pregnancy, breastfeeding or hormone therapy, or treatment of any kind were not included. Volunteers with high-dietary fiber intake or possible eating disorders, assessed through a questionnaire on eating behavior were excluded (Stunkard & Messick, 1985). The volunteers were asked to maintain a balanced diet, avoiding alcohol consumption in the days before each assay day.

The assays were approved by the Ethical Research Committee of the Faculty of Pharmaceutical Science, University of Sao Paulo (USP)[1] and University Hospital HU/USP (CEP-HU/USP 878). The collection of biological material was performed in the Center of Clinical and Epidemiological Research HU/USP. All volunteers signed an informed consent form before the start of the interventions.

2.5.2. Experimental designs

Three sequential trials were performed testing 4 meals (C1, T1, C2, T2). The meals were randomly offered for all of them. Every week the volunteers consumed one meal (400 g) with 250 mL of water, within 10 to 15 min.

2.5.2.1. Glycemic response — study design. The glycemic response to the meals was determined according to the protocol proposed by FAO (Brouns et al., 2005; FAO/WHO, 1998). Healthy volunteers (n = 16), both genders, aged 29.3 ± 9.0 (standard deviation) years old and normal body mass index (BMI) of 21.8 ± 1.3 kg/m^2 participated in the study. White bread (reference food) was tested three times in the three first weeks; in the subsequent weeks (until the seventh week), the volunteers consumed one of the four meals, in a randomized way. Each portion of bread (61 g) or meals (C1 — 387 g, T1 — 375 g, C2 — 390 g, T2 — 398 g) contained 25 g of available carbohydrates. In order to elaborate the glycemic curve, blood sugar levels of the following timepoints were used: 0, 15, 30, 45, 60, 90 and 120 min.

The glycemic index (GI) of the meals was calculated using the ratio between the area under the curve produced by the meals (C1, T1, C2, T2) and by the white bread (reference = 100%). The area under the curve was calculated geometrically, applying the trapezoidal rule, with the fasting state line as the base. To obtain the GI value expressed with glucose as reference food, the GI value with bread as reference was multiplied by 0.7, both present high GI, but the bread GI is 30% lower than the glucose GI. The GL of each meal was calculated by the following equation: GL = GI (reference = glucose) × content of available carbohydrate (g) in the ingested portion × 1 / 100 (Salmeron et al., 1997). The Harvard (2014) reference was used to classify GI and GL.

2.5.2.2. Gastrointestinal hormones — study design. The study was conducted in healthy volunteers (n = 15 females), aged 29.3 ± 5.3 years old and BMI of 22.5 ± 2.0 kg/m^2. The volunteers came to the Center for Clinical and Epidemiological Research in University Hospital (HU/USP) in the morning, with weekly intervals, after a 10–12 h fasting and had

[1] Glycemic response CEP-FCF/USP 456; gastrointestinal hormones CEP-FCF/USP 496; hunger/satiety CEP-FCF/USP 523.

their vein cannulated. Before and after the consumption of the meal, blood samples (8 mL) were collected at the timepoints: 0, 30, 60, 120 and 180 min, when the volunteers' catheter was removed.

2.5.2.3. Hunger and satiety — study design. The study was performed in healthy volunteers (n = 52 females), aged between 18 and 40 years old who came to the laboratory of the Department of Food and Experimental Nutrition of FCF/USP at lunch time (4 h after breakfast), for the *Visual Analogue Scale* test (before and after the consumption of meals) and evaluation of energy consumption in subsequent standardized meals.

2.6. Hunger and satiety measurements

Visual Analogue Scale (VAS) Test: The volunteers completed the test (Flint, Raben, Blundell, & Astrup, 2000) with 5 questions assessing hunger, thirst, satiety, prospective food consumption and desire to eat.

Bipolar scale used by Holt, Miller, Petocz, and Farmakalidis (1995): It is a 7-point bipolar scale anchored by extremes, from extremely hungry (−3) to extremely satiated (+3). For the assessment of hunger/satiety scores for the questions, the area under the curve (AUC) for 180 min, from time zero (t = 0), and after the consumption of one of the meals on times 30, 60, 90, 120 and 180 min, was calculated.

Assessment of energy consumption in subsequent standardized meals (Ball et al., 2006; Ludwig et al., 1999): Two meals, with a known composition, were offered for consumption *ad libitum*: a snack (a slice of white bread with cheese spread — 452 kJ/unit) after 180 min accompanied by ready-to-drink mate tea (200 mL — 167 kJ), and a dinner after 360 min from time zero (frozen lasagna with tomato sauce and ground beef — 504 kJ/100 g).

2.7. Statistical analysis

For the analysis of the results the Statistica Software version 10.0 (StatSoft Inc. Tulsa, OK, USA) was used.

The plasma concentration of gastrointestinal hormones has a large inter-personal variation, so the variations after consumption of group 1 and 2 meals were normalized as a percentage variation from time zero (t0).

Chemical results were presented as mean ± standard deviation (SD) and the differences were determined by Student's *t* test. In some analyses, repeated measures ANOVA was also used and the differences were determined by the *post hoc* Tukey test.

The results for glycemic index were presented as mean ± standard error (SE) and the differences were determined by Student's *t* test.

For parameters with large inter-personal variation (gastrointestinal hormones, hunger/satiety assay, food consumption), the results were presented as mean, median, minimum and maximum, in addition to the percentage variation. Repeated measures ANOVA was used and the differences were determined by the *post hoc* Tukey test; for food consumption Student's *t* test was used. Differences for p values <0.05 were considered significant.

3. Results and discussion

The four meals were hyperproteic, and those from group 1 (C1 and T1) showed higher concentration of proteins and lipids and also energy value. All meals had similar content of total and available starch. The group 2 meals (C2 and T2) contained a higher concentration of unavailable carbohydrates (DF + RS + fructans) (C2 = 2.81% and T2 = 5.44%) when compared to group 1 (C1 = 2.40% and T1 = 4.29%) (Table 1S — supplementary material). This fact is consistent with the composition of group 2 meals, composed of pasta produced with whole-wheat flour, an important source of DF, while the group 1 meals are composed of polished rice and corn starch.

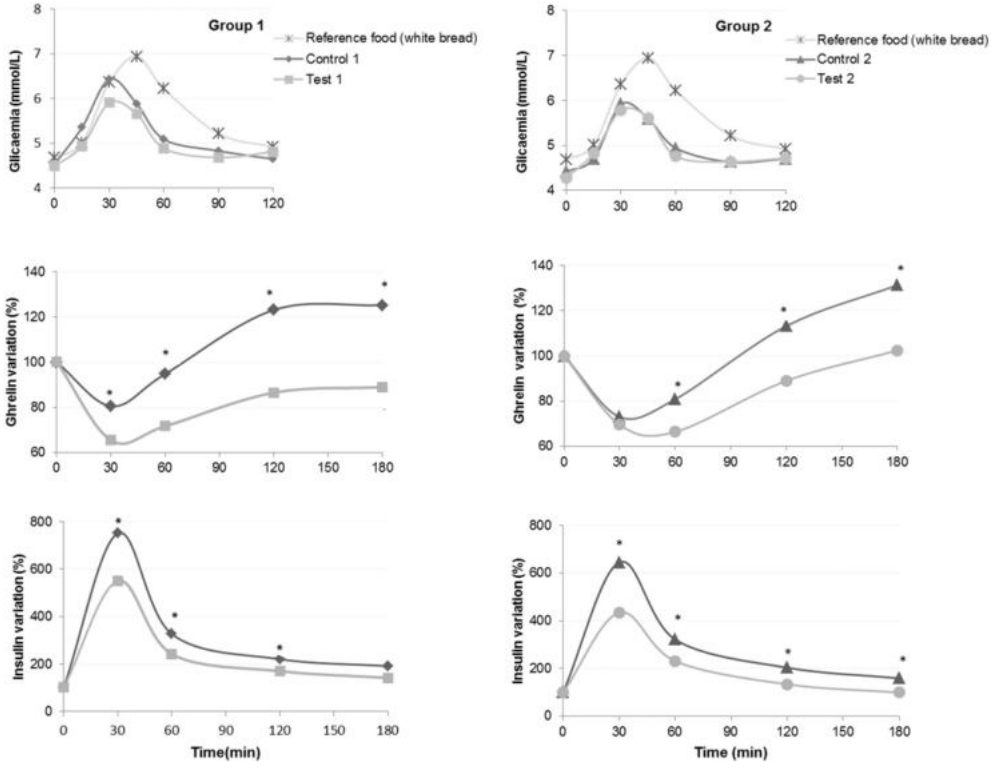

Fig. 1. Glycemic response in 120 min (n = 16) and percentage variation compared to fasting in ghrelin and insulin plasma concentrations up to 180 min (n = 15), after consumption of control meals (C1 and C2) and test meals, added inulin (T1 and T2) by healthy volunteers. *Significant difference for the same grouptype (Tukey test; $p < 0.05$).

Since the two groups of meals have different characteristics, the variations were compared within the same group (C1 × T1 and C2 × T2). All the responses obtained concerning satiety-related parameters were mainly due to the addition of inulin to test meals (T1 and T2), since the chemical composition and energy density within each group were similar, except the carbohydrate content.

All studied meals had a low glycemic index (between 35 and 42%) and low glycemic load (9–10). The glycemic response produced by group 1 meals showed that the addition of inulin modified the curve profile, reducing the glycemic response peak (Fig. 1), as well as the area under the curve (AUC). The presence of this DF may have altered glucose absorption and delayed its release into the bloodstream. Group 2 meals showed very similar curves, possibly due to the presence of wholemeal ingredients in their formulation (wholewheat pasta, textured soy protein, vegetables). A review on functional oligosaccharides shows that the structure and viscosity of DF can modulate blood glucose (Qiang, YongLie, & QianBing, 2009).

The high concentration of ghrelin is responsible for the hunger sensation during fasting and it can rise above the fasting concentrations few hours after consumption of a meal, which could increase the hunger sensation. This orexigenic hormone produced by the stomach is affected not only by the composition of the meal or type of nutrient ingested, but also by gastric distension, responding to pre- and post-absorptive stimuli (Williams, Cummings, Grill, & Kaplan, 2003).

The test meals showed a lower increase in ghrelin compared to their respective control from 30 or 60 min (Table 2S — supplementary material). For C1 and T1 meals the variations were significant at all timepoints (t30 min, $p = 0.0161$; t60, $p = 0.0197$; t120, $p = 0.0182$; t180, $p =$

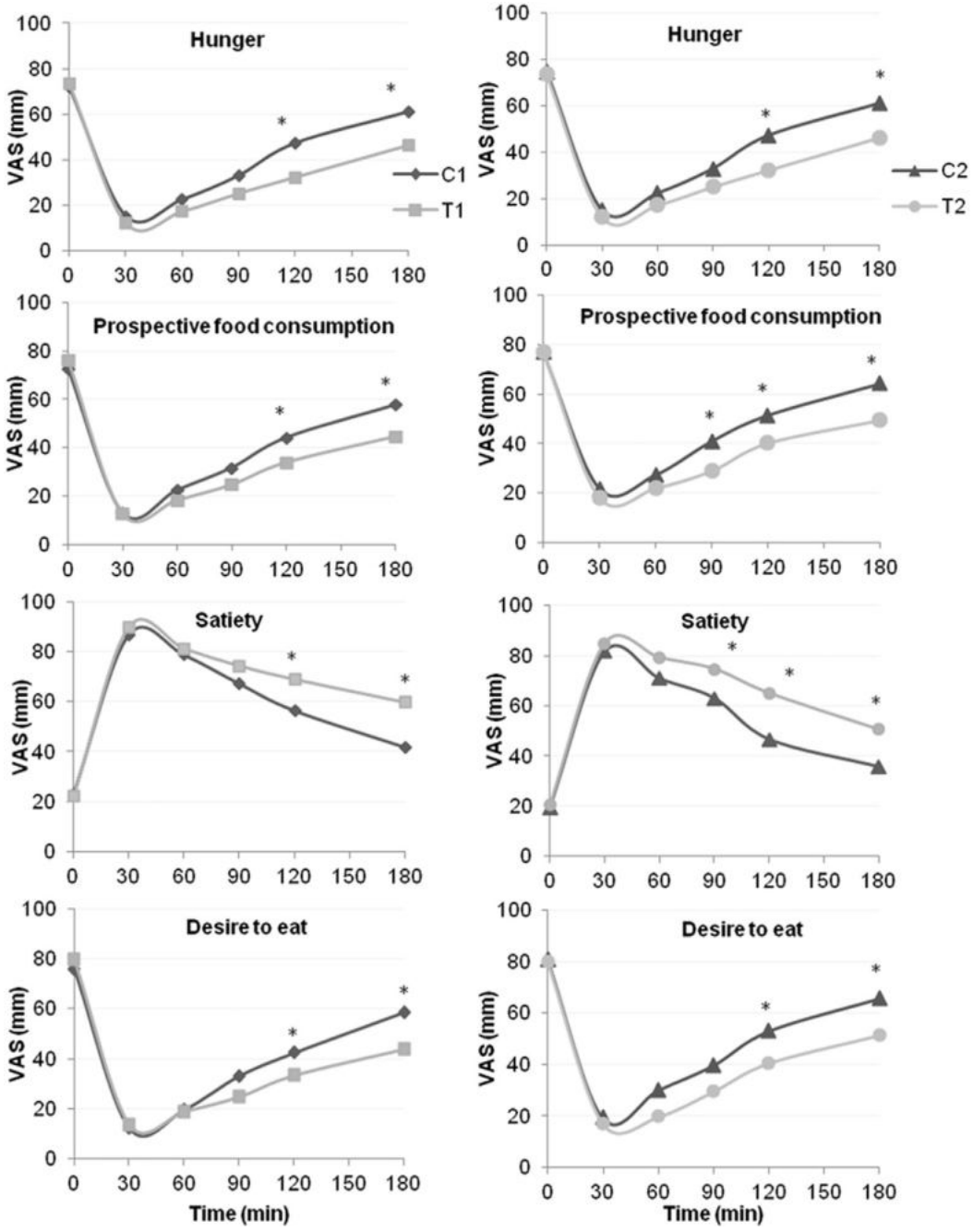

Fig. 2. Variation in hunger, prospective food consumption, satiety and desire to eat during 180 min, after the consumption of control (C1 and C2) and test meals, added inulin (T1 and T2) by healthy volunteers (n = 52). C1 and C2 — 1 and 2 control meals; T1 and T2 — C1 and C2 meals added inulin. *Significant difference for the same group (Tukey test; $p < 0.05$).

0.0368); for C2 and T2 meals the variations were significant at t60 ($p = 0.0474$), t120 ($p = 0.0082$) and t180 ($p = 0.0023$). This ghrelin plasma variation signalizes a possible reduction in hunger in the subsequent meal after consumption of meals added inulin, which caused a lower increase when compared to their control. The lower ghrelin plasma concentration at 180 min may cause higher satiety and a lower energy requirement on the next meal (Cani et al., 2006; El Khoury, Obeid, Azar, & Hwalla, 2006; Genta et al., 2009; Verhoef, Meyer, & Westerterp, 2011).

As it is a dietary fiber with a soluble characteristic, the inulin added to the studied meals is a component that causes gastric distension and may alter the absorption of nutrients, which are factors related to ghrelin suppression after food consumption (Williams et al., 2003). Several studies show that the fructans are rapidly fermented in the proximal part of the colon, producing short chain fatty acids (SCFA — acetate, butyrate and propionate), that are able to modulate the release of ghrelin (Sanchez, Miguel, & Aleixandre, 2012; Wang et al., 2007), which would happen a few hours after intake. Although this mechanism is not totally clear, Suginoa et al. (2011) suggest that the SCFA can suppress plasma ghrelin concentration through stimulation of vagal afferents, or as a response of the increase in YY peptide, stimulated by acetate (Freeland & Wolever, 2010).

Regarding insulin, T1 and T2 meals added inulin had their peaks reduced compared to controls. The variations in plasma insulin concentrations for T1 compared C1 meals (Table 3S — supplementary material) were lower at t30 ($p = 0.0174$), t60 ($p = 0.0124$) and t120 ($p = 0.0232$). Between T2 and C2 meals there was a decrease at all timepoints (t30, $p = 0.0055$; t60, $p = 0.0342$; t120, $p = 0.0307$; t180, $p = 0.0077$).

The low insulinic response is due to the consumption of low glycemic load meals (Fig. 1), however the SCFA inulin fermentation process can also contribute. Acetate has been related to the regulation of glucose-stimulated insulin secretion, possibly through Free Fatty Acid Receptor 2 (FFAR2) and 3 (FFAR3), expressed in pancreatic β cells (Priyadarshini et al., 2015).

Rats fed fructose rich-diet added inulin show improved glucose and insulin levels as well as lipid profile, possibly due to the ability to change the blood level of adipocytokines (Nassar, Ismail, El-Damarawi, & El-Din, 2013). Karhunen et al. (2010) also observed that feeds enriched with DF interfered positively in glucose, insulin and ghrelin plasma levels in healthy young adults.

In a study by El Khoury et al. (2006), a carbohydrate rich-diet showed greater suppression of ghrelin and higher insulin peak, and the authors believe that this strengthens the hypothesis that insulin produced by the consumption of a meal can induce a greater suppression of ghrelin (Purnell, Weigle, Breen, & Cummings, 2003). In the present study, the highest peak regarding the percentage variation in insulin is not associated with greater percentage suppression in ghrelin, yet these hormones had a good inverse correlation (−0,53; $p < 0,05$). A study by Erdmann, Töpsch, Lippl, Gussmann, and Schusdziarra (2004) confirmed the existence of a negative association ($r = -0.44$; $p < 0.001$) between those hormones, because it found a reduced increase in ghrelin levels as a function of a small or moderate increase in insulin.

By the VAS test it was observed that the hunger sensation was reduced by the addition of inulin in both test meals at t120 and t180 (Fig. 2). Those results are consistent with the results for ghrelin and insulin related to hunger only at those timepoints, since the meals showed differences in those hormones in almost all timepoints evaluated. Differences at those same timepoints (120 and 180 min) were found in the parameters reflecting the prospective food consumption, satiety and desire to eat. The group 2 meals also showed a difference at 60 min for consumption perspective (B) and satiety (C). No differences were found in the parameter thirst.

In the initial period the individuals are more satiated, therefore the difference found in hormone concentration in this period between control and test meals (Tables 2S and 3S — supplementary material) was not enough to be noticed in a test considered subjective such as the VAS. Using this same instrument, Isaksson, Fredriksson, Andersson, Olsson, and Aman (2009) verified that different dose–responses (5 and 8 g DF) increased the satiety compared to control before lunch, but did not change hunger or urge to eat. Thus, it is important that VAS is used with other tools in order to confirm those parameters.

Foods with low GI are responsible for the greater power of satiety due to the lower insulin response produced. Ball et al. (2006) observed that the greater amount of insulin resulting from a high GI meal produced less satiety, since it reduced the time to request the next meal compared to low GI meals. Thus, low GI foods, such as the meals in the present study, produce reduced glucose and insulin blood levels (Fig. 1), which can explain the hunger sensation reduction after their ingestion (Fig. 2). The lower ghrelin observed after intake of meals (Fig. 1) with inulin possibly contributed to the variations observed in VAS concerning hunger, satiety, consumption perspective and urge to eat for test meals at 120 and 180 min (Fig. 2).

The areas under the curve (AUCs) for the satiety produced by the test meals, calculated from the bipolar scale used by Holt et al. (1995), were superior for both groups compared to areas from control meals ($p = 0.0005$), which means that they provided higher satiety (Fig. 3).

In the assessment of energy consumption from subsequent meals (180 min and 360 min), it was observed that there was a reduction in energy intake after the test meals. For the T1 meal the reduction was 18% in the snack and 12% at dinner compared to C1; in the comparison between T2 and C2 the reduction was 11% in the snack and 17% at dinner. This percentage difference observed in the present study represented an average reduction of 419–578 kJ (100–138 kcal) of the total energy intake after consuming meals added inulin (Fig. 4). These reduction confirm the relation with the lowest ghrelin and insulin plasma concentration observed, as well as a reduction in hunger, prospective food consumption and desire to eat and increase in satiety of test meals verified in VAS at 180 min.

In a review study, Klosterbuer, Greaves, and Slavin (2012) concluded that DF is associated with a decrease in post-prandial ghrelin and this modulation may be due to the colonic fermentation of fructans (Sanchez et al., 2012; Tarini & Wolever, 2010; Wang et al., 2007). In another review realized by Wanders et al. (2011), it was observed that there are different short- and long-term effects dependent on the DF chemical structure concerning appetite, energy intake and body weight, and the authors suggest that it is not possible yet to establish a dose–response as a function of interaction between multiple mechanisms and different physical–chemical properties of the various DF types. According to Clark and Slavin (2013), the association between DF intake and weight control may be due to the increase of satiation and other effects related to insulin release, and not necessarily to appetite control.

Unlike the observations described herein, acute studies sometimes do not allow associations between DF and hunger/satiety (Clark & Slavin, 2013; Karalus et al., 2012; Lafond, Greaves, Maki, Leidy, & Romsos, 2015), because some effects are not due only to the presence of the nutrients in the digestive system, but can occur many hours

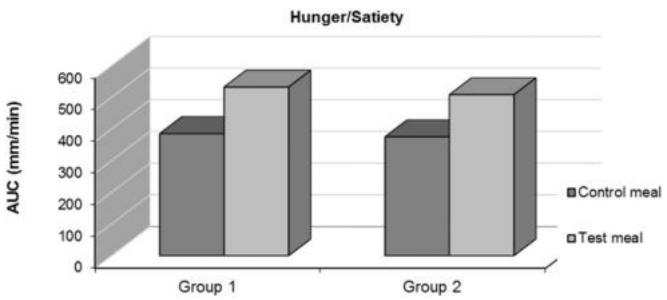

Fig. 3. Area under the curve (AUC) for Holt bipolar scale for the assessment of hunger/satiety (extreme hunger = 0) after the consumption of control (C1 and C2) and test meals, added inulin (T1 and T2) by healthy volunteers ($n = 52$). C1 and C2 — 1 and 2 control meals; T1 and T2 — C1 and C2 meals added inulin. *Significant difference for the same group (Tukey test; $p < 0.05$).

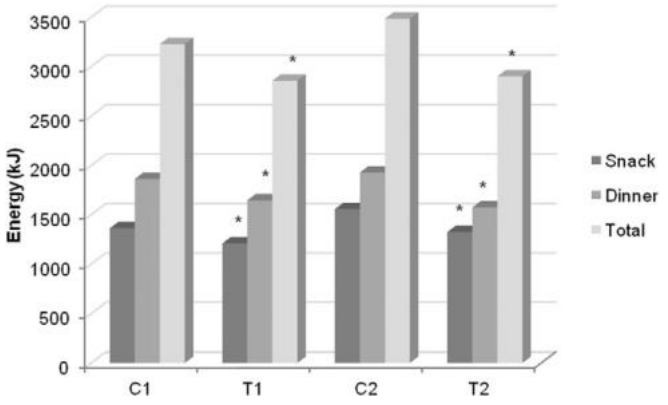

Fig. 4. Subsequent energy intake (snack — 180 min and dinner — 360 min) after the consumption of control (C1 and C2) and test meals, added inulin (T1 and T2) by healthy volunteers (n = 52). C1 and C2 — 1 and 2 control meals; T1 and T2 — C1 and C2 meals added inulin. *Significant difference for the same group (Tukey test; $p < 0.05$).

after consumption. However, the fermentation and consequent SCFA production may contribute to long-term effects such as reduced food intake at subsequent meals due to hormonal control like other authors have also suggested (Jakobsdottir et al., 2014; Nilsson, Ostman, Holst, & Björck, 2008; Roberfroid et al., 2010).

In this study, the addition of inulin in meals altered ghrelin and insulin plasmatic levels; however, Klosterbuer et al. (2012), in a review, did not observe those effects in all clinical trials. The authors believe that the controversial results are due to the limitation of some studies regarding sample size, lack of *crossover* or inappropriate control.

4. Conclusions

The addition of inulin to both groups of ready-to-eat frozen meals provided a positive change in post-prandial plasma profiles of the gastrointestinal hormones ghrelin and insulin compared to control meals. These changes may explain the greater satiety and lower hunger sensation, consumption perspective and desire to eat observed after the meals with inulin, as well as lower energy intake in two subsequent meals. Further studies are necessary in order to verify if these benefits can be maintained with regular intake.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.foodres.2015.06.038.

Conflict of interest

All the authors declare that there is no conflict of interest.

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References

Ball, S., Keller, K., Moyer-Mileur, L., Ding, Y., Donaldson, D., & Jackson, W. (2006). Prolongation of satiety after low versus moderately high glycemic index meals in obese adolescents. *Pediatrics*, *111*(3), 488–494.

Benelam, B. (2009). Satiation, satiety and their effects on eating behaviour. *Nutrition Bulletin*, *34*, 127–174.

Brasil (2001). *Resolução - RDC n° 12. Aprova o Regulamento Técnico sobre padrões microbiológicos para alimentos.* Ministério da Saúde. Agência Nacional de Vigilancia Sanitária (ANVISA).

Brasil (2014). *Legislação de Boas Práticas de Fabricação.* Ministério da Saúde. Agência Nacional de Vigilância Sanitária (ANVISA) (Retrieved cited 2014 mar 31).

Brouns, F., Bjorck, I., Frayn, K.N., Gibbs, A.L., Lang, V., Slama, G., et al. (2005). Glycaemic index methodology. *Nutrition Research Reviews*, *18*(1), 145–171.

Burton-Freeman, B. (2000). Dietary fiber and energy regulation. *Journal of Nutrition*, *130*(2S Suppl.), 272S–275S.

Cani, P.D., Joly, E., Horsmans, Y., & Delzenne, N.M. (2006). Oligofructose promotes satiety in healthy human: A pilot study. *European Journal of Clinical Nutrition*, *60*(5), 567–572.

Clark, M., & Slavin, J. (2013). The effect of fiber on satiety and food intake: A systematic review. *Journal of the American College of Nutrition*, *32*(3), 200–211.

Cordenunsi, B.R., & Lajolo, F.M. (1995). Starch breakdown during banana ripening — Sucrose synthase and sucrose-phosphate synthase. *Journal of Agriculture and Food Chemistry*, *43*, 347–351.

Cordenunsi, B., Shiga, T., & Lajolo, F. (2008). Non-starch polysaccharide composition of two cultivars of banana (*Musa acuminata* L.: cvs Mysore and Nanicao). *Carbohydrate Polymers*, *71*(1), 26–31.

El Khoury, D., Obeid, O., Azar, S., & Hwalla, N. (2006). Variations in postprandial ghrelin status following ingestion of high-carbohydrate, high-fat, and high-protein meals in males. *Annals of Nutrition and Metabolism*, *50*(3), 260–269.

Erdmann, J., Töpsch, R., Lippl, F., Gussmann, P., & Schusdziarra, V. (2004). Postprandial response of plasma ghrelin levels to various test meals in relation to food intake, plasma insulin, and glucose. *Journal of Clinical Endocrinology and Metabolism*, *89*(6), 3048–3054.

FAO/WHO (1998). Carbohydrates in human nutrition. *Report of a joint FAO/WHO expert consultation (FAO food and nutrition paper 66)*. Rome: Food and Agriculture Organization (April 14–18, 1997. (140 pp.)).

Flint, A., Raben, A., Blundell, J., & Astrup, A. (2000). Reproducibility, power and validity of visual analogue scares in assessment of appetite sensations in single test meal studies. *International Journal of Obesity*, *24*(1), 38–48.

Freeland, K.N., & Wolever, T.M.S. (2010). Acute effects on intravenous and rectal acetate on glucagon-like peptide-1, peptide YY, ghrelin, adiponectin and tumor necrosis factor-α. *British Journal of Nutrition*, *103*(3), 460–466.

Genta, S., Cabrera, W., Habib, N., Pons, J., Carillo, I.M., Grau, A., et al. (2009). Yacon syrup: Beneficial effects on obesity and insulin resistance in humans. *Clinical Nutrition*, *28*(2), 182–187.

Harvard (2014). Carbohydrates and the glycemic load. Retrieved nov 2014, from http://www.hsph.harvard.edu/nutritionsource/carbohydrates-and-the-glycemic-load/

Holt, s., Miller, j., Petocz, p., & Farmakalidis, E. (1995). A satiety index of common foods. *European Journal of Clinical Nutrition*, *49*(9), 675–690.

Horwitz, W., & Latimer, G.W., Jr. (2006). *Official methods of analysis of AOAC International* (18th ed.). Maryland: AOAC International.

Isaksson, H., Fredriksson, H., Andersson, R., Olsson, J., & Aman, P. (2009). Effect of rye bread breakfasts on subjective hunger and satiety: A randomized controlled trial. *Nutrition Journal*, *8*, 39 (8 pp.).

Jakobsdottir, G., Nyman, M., & Fak, F. (2014). Designing future prebiotic fiber to target metabolic syndrome. *Nutrition*, *30*(5), 497–502.

Karalus, M., Clark, M., Greaves, K., Thomas, W., Vickers, Z., Kuyama, M., et al. (2012). Fermentable fibers do not affect satiety or food intake by women who do not practice restrained eating. *Journal of the Academy of Nutrition and Dietetics*, *112*(9), 1356–1362.

Karhunen, L., Juvonen, K., Flander, S., Liukkonen, K., Lahteenmaki, L., Siloaho, M., et al. (2010). A psyllium fiber-enriched meal strongly attenuates postprandial gastrointestinal peptide release in healthy young adults. *Journal of Nutrition*, *140*(4), 737–744.

Klosterbuer, A.S., Greaves, K.A., & Slavin, J. (2012). Fiber intake inconsistently alters gut hormone levels in humans following acute or chronic intake. *Journal of Food Research*, *1*(2), 255–273.

Lafond, D.W., Greaves, K.A., Maki, K.C., Leidy, H.J., & Romsos, D.R. (2015). Effects of two dietary fibers as part of ready-to-eat cereal (RTEC) breakfasts on perceived appetite and gut hormones in overweight women. *Nutrients*, *7*(2), 1245–1266.

Lee, S.C., Prosky, L., & Devries, J.W. (1992). Determination of total, soluble and insoluble dietary fiber in foods. Enzymatic–gravimetric method, MES-TRIS buffer: Collaborative study. *Journal of AOAC International*, *75*, 395–416.

Ludwig, D., Majzoub, J., Al-Zahrani, A., Dallal, G., Blanco, I., & Roberts, S. (1999). High glycemic index foods, overeating, and obesity. *Pediatrics*, *103*(3).

Mattes, R.D., Hollis, J., Hayes, D., & Stunkard, A.J. (2005). Appetite: Measurement and manipulation misgivings. *Journal of American Dietetic Association*, *105*(5 Suppl. 1), S87–S97.

McCleary, B.V., McNally, M., & Rossiter, P. (2002). Measurement of resistant starch by enzymatic digestion in starch and selected plant materials: Collaborative study. *Journal of AOAC International*, *85*(5), 1103–1111.

McCleary, B.V., Murphy, A., & Mugford, D.C. (2000). Measurement of total fructan in foods by enzymatic/spectrophotometric method: Collaborative study. *Journal of AOAC International*, *83*(2), 356–364.

McCleary, B.V., & Rossiter, P. (2004). Measurement of novel dietary fibers. *Journal of AOAC International*, *87*(3), 707–717.

Nair, K., Kharb, S., & Thompkinson, D. (2010). Inulin dietary fiber with functional and health attributes — A review. *Food Reviews International*, *26*(2), 189–203.

Nassar, S.E., Ismail, G.M., El-Damarawi, M.A., & El-Din, A.A.A. (2013). Effect of inulin on metabolic changes produced by fructose rich diet. *Life Science Journal*, *10*(2), 1807–1814.

Nilsson, A.C., Ostman, E.M., Holst, J.J., & Björck, I.M. (2008). Including indigestible carbohydrates in the evening meal of healthy subjects improves glucose tolerance, lowers inflammatory markers, and increases satiety after a subsequent standardized breakfast. *Journal of Nutrition*, *138*(4), 732–739.

Priyadarshini, M., Villa, S.R., Fuller, M., Wicksteed, B., Mackay, C.R., Alquier, T., et al. (2015). An acetate-specific GPCR, FFAR2, regulates insulin secretion. *Molecular Endocrinology*http://dx.doi.org/10.1210/me.2015-1007 (Jun 15: 11 pp.).

Purnell, J., Weigle, D., Breen, P., & Cummings, D. (2003). Ghrelin levels correlate with insulin levels, insulin resistance, and high-density lipoprotein cholesterol, but not with gender, menopausal status, or cortisol levels in humans. *Journal of Clinical Endocrinology and Metabolism*, *88*(12), 5747–5752.

Qiang, X., YongLie, C., & QianBing, W. (2009). Health benefit application of functional oligosaccharides. *Carbohydrate Polymers*, *77*(3), 435–441.
Roberfroid, M., Gibson, G., Hoyles, L., McCartney, A., Rastall, R., Rowland, I., et al. (2010). Prebiotic effects: Metabolic and health benefits. *British Journal of Nutrition*, *104*, S1–S63.
Salmeron, J., Ascherio, A., Rimm, E., Colditz, G., Spiegelman, D., Jenkins, D., et al. (1997). Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes Care*, *20*(4), 545–550.
Sanchez, D., Miguel, M., & Aleixandre, A. (2012). Dietary fiber, gut peptides, and adipocytokines. *Journal of Medicinal Food*, *15*(3), 223–230.
Slavin, J. (2013). Fiber and prebiotics: Mechanisms and health benefits. *Nutrients*, *5*(4), 1417–1435.
Stunkard, A., & Messick, S. (1985). The 3-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research*, *29*(1), 71–83.
Suginoa, T., Hasegawab, Y., Kojimac, M., Kangawad, K., Obitsua, T., Taniguchi, K., et al. (2011). Plasma ghrelin concentration is decreased by short chain fatty acids in wethers. *Domestic Animal Endocrinology*, *41*, 50–55.
Tarini, J., & Wolever, T.M. (2010). The fermentable fibre inulin increases postprandial serum short-chain fatty acids and reduces free-fatty acids and ghrelin in healthy subjects. *Applied Physiology, Nutrition, and Metabolism*, *35*(1), 9–16.
Verhoef, S.P., Meyer, D., & Westerterp, K.R. (2011). Effects of oligofructose on appetite profile, glucagon-like peptide 1 and peptide YY3-36 concentrations and energy intake. *British Journal of Nutrition*, *106*(11), 1757–1762.
Wanders, A., van den Borne, J., de Graaf, C., Hulshof, T., Jonathan, M., Kristensen, M., et al. (2011). Effects of dietary fibre on subjective appetite, energy intake and body weight: A systematic review of randomized controlled trials. *Obesity Reviews*, *12*(9), 724–739.
Wang, Z.Q., Zuberi, A.R., Zhang, X.H., Macgowan, J., Qin, J., Ye, X., et al. (2007). Effects of dietary fibers on weight gain, carbohydrate metabolism, and gastric ghrelin gene expression in mice fed a high-fat diet. *Metabolism*, *56*(12), 1635–1642.
WHO (World Health Organization) (1997). *L'epidemie d'obesite expose des millions de personnes a d'autres maladies. Vol. 46*, Geneve: Communiqué OMS.
WHO/FAO (2003). Diet, nutrition and the prevention of chronic diseases. *Report of a joint WHO/FAO expert consultation. WHO technical report*, *916*. Geneva: World Health Organization.
Williams, D., Cummings, D., Grill, H., & Kaplan, J. (2003). Meal-related ghrelin suppression requires postgastric feedback. *Endocrinology*, *144*(7), 2765–2767.
Woods, S., & D'Alessio, D. (2008). Central control of body weight and appetite. *Journal of Clinical Endocrinology and Metabolism*, *93*(11), S37–S50.