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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.