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L-rhamnose as a source of colonic propionate inhibits insulin secretion but does not influence measures of appetite or food intake

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ABSTRACT

Activation of free fatty acid receptor (FFAR)2 and FFAR3 *via* colonic short-chain fatty acids, particularly propionate, are postulated to explain observed inverse associations between dietary fiber intake and body weight. Propionate is reported as the predominant colonic fermentation product from L-rhamnose, a natural monosaccharide that resists digestion and absorption reaching the colon intact, while effects of long-chain inulin on appetite have not been extensively investigated. In this single-blind randomized crossover study, healthy unrestrained eaters ($n = 13$) ingested 25.5 g/d L-rhamnose, 22.4 g/d inulin or no supplement (control) alongside a standardized breakfast and lunch, following a 6-d run-in to investigate if appetite was inhibited. Postprandial qualitative appetite, breath hydrogen, and plasma glucose, insulin, triglycerides and non-esterified fatty acids were assessed for 420 min, then an *ad libitum* meal was provided. Significant treatment x time effects were found for postprandial insulin ($P = 0.009$) and non-esterified fatty acids ($P = 0.046$) with a significantly lower insulin response for L-rhamnose ($P = 0.023$) than control. No differences between treatments were found for quantitative and qualitative appetite measures, although significant treatment x time effects for meal desire ($P = 0.008$) and desire to eat sweet ($P = 0.036$) were found. Breath hydrogen was significantly higher with inulin ($P = 0.001$) and L-rhamnose ($P = 0.009$) than control, indicating colonic fermentation. These findings suggest L-rhamnose may inhibit postprandial insulin secretion, however neither L-rhamnose or inulin influenced appetite.
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1. Introduction

Inverse associations between dietary fiber intake and body weight (Du et al., 2010; Howarth, Huang, Roberts, & McCrory, 2005), hunger and energy intake (EI) following non-digestible carbohydrate ingestion in randomized controlled trials (Wanders et al., 2011), indicate dietary fiber and other non-digestible carbohydrates may have a role in the prevention and treatment of obesity. Postulated mechanisms include an increased viscosity of intestinal contents (Kristensen & Jensen, 2011), a reduced energy density due to the bulking effect of non-digestible carbohydrates (Burton-Freeman, 2000), and an inhibition of EI arising from effects of non-digestible carbohydrate on satiation and satiety (Burton-Freeman, 2000), possibly mediated by actions of colon derived short-chain fatty acids (SCFA).

Physiological serum SCFA concentrations are low, in the region of 1, 2 and 65 μmol/L for fasting serum butyrate, propionate and acetate (Fernandes, Vogt, & Wolever, 2011). Postprandially SCFA concentrations appear to increase significantly in response to ingestion of some non-digestible carbohydrates including resistant starch (Robertson, Bickerton, Dennis, Vidal, & Frayn, 2005). Physiological SCFA concentrations have been shown to activate two G-protein coupled receptors, free fatty acid receptor (FFAR) 2 and

Abbreviations: AUC, Area under curve; EI, Energy intake; FFAR, Free fatty acid receptor; GLP-1, Glucagon-like receptor-1; HOMA, Homeostasis Assessment Model; iAUC, Incremental area under curve; L-Rha, L-rhamnose; PYY, Peptide YY; SCFA, Short-chain fatty acid; VAS, Visual analogue scale.

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FFAR3 (Brown et al., 2003; Le Poul et al., 2003), with propionate reported as the most potent agonist (Le Poul et al., 2003). FFAR2 and FFAR3 are co-localized in colonic enteroendocrine L-cells with peptide YY (PYY) and glucagon-like peptide 1 (GLP-1) (Karaki et al., 2006; Karaki et al., 2008; Tazoe et al., 2009), both hormones which are postulated to play roles in the physiological regulation of appetite (Hussain & Bloom, 2013; Lean & Malkova, 2015). *In vivo* administration of SCFA increases plasma PYY in rats (Cherbut et al., 1998; Psichas et al., 2015) and pigs (Cuche, Cuber, & Malbert, 2000), and of propionate increases GLP-1 and PYY *via* FFAR2 activation in rodents (Psichas et al., 2015). *In vitro* and *in vivo* evidence in rodents further indicates SFCA-induced FFAR2 and FFAR3 activation upregulates leptin expression in adipose tissue (Covington, Briscoe, Brown, & Jayawickreme, 2006; Xiong et al., 2004). Thus SCFA, particularly propionate, may be postulated to influence energy homeostasis and insulin secretion.

L-Rhamnose (L-Rha), a natural monosaccharide that resists digestion and absorption reaching the colon intact (Vogt, Pencharz, & Wolever, 2004b), shows promise as a suitable candidate to investigate effects on colonic propionate on appetite. Propionate is reported as the primary SCFA produced during fermentation of L-Rha *in vitro* (Fernandes, Rao, & Wolever, 2000), and L-Rha ingestion increased serum propionate concentrations in humans acutely (Vogt et al., 2004b) and chronically (Vogt, Ishii-Schrade, Pencharz, & Wolever, 2004a). Effects of L-Rha ingestion on metabolic response have been previously investigated (Vogt et al., 2004a, b); albeit not extensively, however effects on appetite have not.

Effects of supplementing with inulin-type fructans on appetite are more extensively investigated; however, results are contradictory due to variable dosages, differing types of inulin-type fructans and limitations in study design (reviewed by (Darzi, Frost, & Robertson, 2011)). While a number of studies have investigated effects on appetite of short-chain inulin-type fructans, also termed oligofructose and fructooligosaccharides (for example Hess, Birkett, Thomas, & Slavin, 2011; Parnell & Reimer, 2009; Pedersen et al., 2013; Peters, Boers, Haddeman, Melnikov, & Qvyjt, 2009; Verhoef, Meyer, & Westerterp, 2011), few investigations of long-chain inulin effects exist (Archer, Johnson, Devereux, & Baxter, 2004; Karalus et al., 2012; Tarini & Wolever, 2010). The present study therefore aimed to investigate the acute effects of providing L-Rha or long-chain inulin, following a 6-d run-in, on postprandial appetite and metabolite concentrations compared to control (no supplement).

2. Methods

2.1. Participants

Healthy, non-smoking unrestrained eaters 18–55 y were recruited *via* e-mail advertisement at the University of Surrey and attended the Clinical Investigation Unit (CIU) in a fasted state for screening. Inclusion criteria were BMI between 19 and 26 kg/m^2, fasting blood glucose <6.0 mmol/l, weight stable for at least 3 months, non-smoker and reported habitual alcohol intake ≤20 units. Exclusion criteria included following a weight reducing diet, presence of gastrointestinal, endocrine or cardiovascular disorders, history of depression, eating disorders or substance abuse, pregnancy or lactation, taking regular medication (except birth control medication), and high dietary restraint (score ≥3.5 on the Dutch Eating Behaviour Questionnaire restraint scale (Van Strien, Frijters, Bergers, & Defares, 1986)). The study was conducted according to the Declaration of Helsinki and all procedures involving human participants were approved by the University of Surrey Ethics Committee (Ref: EC/2008/53/FHMS). Participants gave written informed consent.

2.2. Study protocol

This single-blind 3-way randomized crossover study was conducted from September 2008 to May 2009. Participants commenced 1-wk study periods during which L-Rha, long-chain inulin or control (no supplement) were consumed an order randomly assigned using www.randomizer.org. The condition randomized to was concealed by providing participants with ready prepared jelly (Jell-O) and mousse containing the supplement or control (no supplement) to disguise the treatment. Each study period comprised a 6-d run-in with a study day at the CIU on Day 7 and were separated by a washout period of ≥1-wk. Prior to commencing the study, all participants who had not previously participated in an appetite study attended an initial study morning at the CIU to familiarise them with with the techniques being used. To control for effects of hormonal variations throughout the menstrual cycle (Asarian & Geary, 2013), female participants not using birth control medication attended the study day at approximately the same point of the menstrual cycle for each study day during the mid-follicular phase (between days 8 and 12). Participants were informed that the study aimed to compare the effects of two fiber supplements compared to a placebo (no fiber) on appetite and metabolic response.

2.3. Six day run-in

The supplement dosages used were based on the reported dose used in previous investigations of L-Rha (Vogt, Ishii-Schrade, Pencharz, Jones, & Wolever, 2006; Vogt, et al., 2004a, b), with the equivalent inulin dose calculated to be matched by pentose/hexose equivalents. The target dose of 25.5 and 22.4 g/d for L-Rha and inulin was reached by Day 4, increasing from one-third, half and two-thirds target dose during Days 1–3. The supplement (inulin or L-Rha) or control (no supplement) was provided within two portions of jelly (Hartleys Sugar Free Jelly) during run-in to be consumed alongside participants' usual diet. Sugar free jelly was chosen as the vehicle to provide the supplement, as it did not contribute greatly to the EI, it disguised the supplement or control, and made it easy for the participant to consume the supplement, as no additional preparation was required. The jellies were collected by or were dropped off to participants every few days. Compliance, gastrointestinal symptoms and the taste of the jellies were assessed using a daily monitoring diary, and a 4-day food diary was completed from Days 3–6 using household measures. Gastrointestinal symptoms (stomach pain, diarrhoea, constipation, belching, flatulence, nausea, acid regurgitation, heartburn and bloating) were monitored on a five point scale (1: none, 3: moderate, 5: debilitating), and taste was assessed on a nine point Likert Scale.

2.4. Study day (Day 7)

Participants arrived at the CIU in a fasted state after consuming a standard low fiber meal the previous evening and avoiding alcohol and unaccustomed exercise for 24-h. Participants were required to stay in the CIU for the entire study duration and water was provided *ad libitum*. Upon arrival anthropometric measurements were taken and an intravenous cannula was inserted into an antecubital vein. Two fasting blood samples were taken 30 min and 5 min before breakfast, and hydrogen concentrations in expired breath were measured using a Gastrolyser 2 portable hand held breath hydrogen monitor (Bedfont Scientific Ltd, Rochester, UK). Following each baseline blood and breath sample appetite was subjectively assessed by 100 mm visual analogue scale (VAS) questionnaires for fullness, hunger, prospective food consumption, desire to eat meal/snack/sweet/savoury/salty/fatty and nausea as previously

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