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Research report

Impaired oral fatty acid chemoreception is associated with acute excess energy consumption ☆

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ABSTRACT

Excessive consumption of dietary fat is implicated with development of obesity. Impaired oral and gastrointestinal chemoreception to the breakdown products of dietary fat, fatty acids, may be associated with increased energy consumption. The objective of this study was to determine if impaired oral fatty acid chemoreception influences energy intake and perceived satiety. Subjects ($n = 24$) attended six laboratory sessions. Impaired fatty acid chemoreception was defined as subjects who could not identify >3.8 mM oleic acid (C18:1). Subjects participated in a blinded crossover study and consumed each of three high macronutrient breakfasts (high fat, high protein, high carbohydrate) and a balanced macronutrient breakfast on four separate days. Following breakfast, subjects were required to consume a buffet-style lunch until comfortably full. The amount consumed (MJ and g) was measured, as was perceived satiety prior to and following meals. Following the high fat breakfast, subjects with impaired fatty acid chemoreception ($n = 10$) consumed significantly more energy (2.1 ± 0.8 MJ) and grams (237.70 ± 46.37 g) of food at lunch compared to other subjects ($P < 0.05$). There were no significant differences in energy, grams of food consumed at lunch and perceived satiety, between subjects for the other breakfasts ($P > 0.05$). Impaired oral fatty acid chemoreception was associated with excess energy consumption following a high fat meal.

Introduction

Obesity is one of the leading preventable conditions associated with the development of negative health outcomes and is prevalent in a large percentage of the world's population (James, 2008). While multiple factors undoubtedly contribute to the development of obesity, it is widely acknowledged that an excess consumption of dietary fat plays a dominant role, (Martinez, 2000; Snoek, Huntjens, Van Gemert, De Graaf, & Weenen, 2004) and taste may influence fat consumption.

Oral chemoreception, including the sense of taste, is a system that indicates the presence of macronutrients in food such as carbohydrate (sugars) and protein (amino acids) (Bachmanov & Beauchamp, 2007). Recent evidence suggests that there may be an oral chemoreception component involved with detection of the other macronutrient, fat via its breakdown products fatty acids (Chale-Rush, Burgess, & Mattes, 2007; Newman & Keast, 2013; Tucker & Mattes, 2013). Putative receptor mechanisms for detection of fatty acids include transporters (CD36, (homologous to fatty acid transporter (FAT) in animals), GPCRs, ion channels (delayed rectifying potassium (DRK) channels) and enzymes (lingual lipase) which have been located in the oral cavity on taste receptor cells within the circumvallate and fungiform papillae (Galindo et al., 2011; Gilbertson, Fontenot, Liu, Zhang, & Monroe, 1997; Pepino, Love-Gregory, Klein, & Abumrad, 2012; Simons, Kummer, Luiken, & Boon, 2011). For a review of fatty acid chemoreception see Newman, Haryono, and Keast (2013). Receptors in the oral cavity for the detection of fat are homologous in the gastrointestinal tract (GIT) raising the possibility of a coordinated alimentary canal response to dietary fat (Mattes, 2005). Indeed, a link between oral fatty acid chemoreception and GIT responses to fatty acid has been established with obese individuals having impaired response to fatty acid in the oral cavity and the GIT (Brennan et al., 2012; Pepino et al., 2012; Samra, 2010;

Abbreviations: C18:1, oleic acid; GIT, gastrointestinal tract; BMI, body mass index; VAS, visual analogue scale; CCK, cholecystokinin; PYY, peptide YY.

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Stewart & Keast, 2012; Stewart et al., 2011) compared to healthy weight subjects.

The presence of fats in the small intestine in healthy, normal weight subjects generates potent satiety signals (Stewart, Feinle-Bisset, & Keast, 2011). Gastric emptying is slowed, gut hormones CCK and PYY are released, and ghrelin is inhibited (Blundell & Macdiarmid, 1997; Feinle et al., 2003), altogether causing suppression of energy intake. These physiological satiety mechanisms may be impaired in the obese with subjects voluntarily consuming twice as much energy from fat products as non-obese (Blundell, Burley, Cotton, & Lawton, 1993; Stewart et al., 2011). In addition, psychological mechanisms such as restrained eating behaviours (Stunkard & Messick, 1985) may be at play as lean subjects demonstrated a decreased desire to eat following consumption of high fat meals in comparison with the obese subjects who retained a residual hunger (Snoek et al., 2004). This suggests that the obese have an attenuated physiological and psychological response to dietary fat in comparison to healthy weight subjects (Stewart et al., 2011).

The majority of satiation and satiety studies have focused on preloads that vary in form (liquid, semi-solid, solid) or macronutrient composition and analysis of group effects or associations with BMI or gender (Almiron-Roig et al., 2013). Other individual factors such as oral chemoreception of macronutrients are plausible factors that may be associated with satiety (Stewart et al., 2011).

Identifying mechanisms causal in excess fat consumption is an important step in developing long-term strategies to combat obesity. Further investigation expanding the link between individual differences in oral chemoreception to fatty acids, oral consumption of fats and the corresponding effect upon perceived satiety is required to target one of the potential contributors to obesity. Therefore, the aim of this study was to assess if impaired fatty acid chemoreception influenced satiety, energy or mass consumption following high fat, carbohydrate or protein meals, with the hypothesis that impaired oral fatty acid chemoreception is associated with decreased satiety following a high fat meal.

Methods

Subjects

Subjects ($n = 24$, 14 males, age 24 ± 8.4 years., BMI 22.8 ± 2.6 kg/m^2, 10 females, age 32 ± 14.3 years., BMI 23.5 ± 4.7 kg/m^2) were recruited by flyer drops around Deakin University, Melbourne. Inclusion criteria included no cold or flu symptoms (good health), no food allergies and over 18 years of age. Smokers were excluded from the study as smoking is known to interfere with the taste process (Sinnot & Rauth, 1937). The required study sample size was determined using a power calculation. Using energy intake at the buffet lunch of 5 MJ (SD of 1 MJ) we would want to identify differences in intake of 0.75 MJ (Lucas, Riddell, Liem, Whitelock, & Keast, 2011; Stewart et al., 2011). With an alpha of 95% and 90% power 23 subjects were required for this study. Ethical approval to conduct this study was obtained from the Deakin University Human Research Ethics Committee (HEAG-H20_2012) and all subjects provided informed, written consent prior to participation. This trial was registered with the Australian New Zealand Clinical Trials Registry (ACTRN12613000055707), http://www.anzctr.org.au.

Study overview

The study comprised of six sessions and subjects were blinded to the real reason for the study and were told the purpose was to assess the influence of diet on taste thresholds. Prior to session 1 subjects completed a Food Frequency Questionnaire (FFQ) and a 4-day diet diary. Anthropometric measurements were recorded to calculate BMI in session 1. Oral fatty acid sensitivity was determined in duplicate during sessions 1 and 6. In sessions 2–5 subjects consumed a high fat, high carbohydrate, high protein or an equal fat, protein and carbohydrate breakfast on four separate days (days could have been consecutive or non-consecutive, depending on subject availability). Subjects then returned to the laboratory at the same time on each testing day and consumed a buffet style lunch where they ate a variety of foods until comfortably full. Measurements of perceived hunger and fullness were taken before and after all meals (breakfast and lunch). Mass of food consumed in grams was recorded and converted into energy (MJ). All sensory data were collected using the Compusense five v 4.6 (Compusense, Guelph, Canada) data collection system.

Study protocol

C18:1 samples. To test oral fatty acid sensitivity, samples were prepared as previously described (Newman & Keast, 2013). Briefly, C18:1 was mixed at varying concentrations (0.02, 0.06, 1, 1.4, 2, 2.8, 3.8, 5, 6.4, 8, 9.8 and 12 mM) with long-life non-fat milk (Devondale, Cobram, Victoria, Australia). To minimise textural cues due to the addition of fat, samples were mixed with 5% (w/v) gum acacia (Deltagen, Boronia, Victoria, Australia) and liquid paraffin (Merck, Darmstadt, Germany). To prevent oxidation of C18:1, samples were mixed with 0.01% w/v EDTA (Merck). Samples were homogenised for 30 seconds/100 mL solution (Silverson L4RT homogeniser, Longmeadow, Massachusetts, USA), prepared fresh on the day of testing and served at room temperature. Control samples were prepared in the same manner, but without the addition of C18:1. To prevent confounding from non-oral sensory inputs, tests were conducted with subjects wearing nose clips.

Forced choice methodology. Samples were presented to subjects in a set of three containing two control samples and one sample with a given concentration of C18:1. Oral detection thresholds were determined with triangle tests using ascending concentrations of C18:1 samples. Subjects were instructed to taste all three samples and to pick the odd sample. If correctly identified, subjects were presented with another set of samples where C18:1 remained at the same concentration. If incorrect, subjects were presented with three more samples where the C18:1 concentration was increased to the next concentration. Subjects rinsed their mouths with filtered water between sets of samples. This procedure continued until the subject identified the C18:1 sample at the same concentration three consecutive times, with a 3.7% chance of guessing correctly. The concentration at which this occurred was defined as the subject's oral detection threshold for C18:1. In line with previous research, we classified individuals as hyposensitive (impaired fatty acid chemoreception) if they required concentrations of C18:1 > 3.8 mM. Subjects correctly identifying C18:1 < 3.8 mM were classified as hypersensitive (Stewart, Newman, & Keast, 2011).

Subjects were tested in duplicate on both testing days to ensure consistency in line with previous research (Newman & Keast, 2013). Two hours prior to testing subjects were required to fast from all foods.

Anthropometry measurements. Weight and height were collected for all subjects without shoes and in light clothing using dedicated scales (Tanita Body Scan Composition Monitor Scales, Cloverdale, Western Australia, Australia) and a portable stadiometer (Seca, MedShop Australia, Fairfield, Victoria, Australia) at baseline. From this, BMI was calculated (weight (kg)/height (m^2)), and subjects were divided into groups based on cut-off values for lean BMI <24.9 kg/m^2 and overweight (OW) ≥ 25–29.9 kg/m^2 or obese (OB) ≥ 30 kg/m^2.