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Research report

Appetite and gut peptide responses to exercise and calorie restriction. The effect of modest energy deficits ☆

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ABSTRACT

Weight loss is the result of a sustained negative energy balance, which is typically achieved by decreasing food intake and/or increasing physical activity. Current evidence suggests that acute energy deficits of ~4820 kJ elicit contrasting homeostatic responses when induced by exercise and food restriction but the response to government-recommended energy deficits is unknown. Twelve healthy men (mean(SD): age 24(5) years, body mass index 23.8(2.7) kg m^{-2}, maximum oxygen uptake 55.4(9.1) mL kg^{-1} min^{-1}) completed three 8 h trials (control (Con), exercise-induced energy deficit (Ex-Def) and food restriction (Food-Def)) separated by 1 week. Thirty minutes of cycling at 64.5(3.2)% of maximum oxygen uptake was performed in Ex-Def from 0 to 0.5 h, which induced an energy deficit of 1469(256) kJ. An equivalent energy deficit was induced in Food-Def (1478(275) kJ) by reducing the energy content of standardised test meals at 1 h and 4 h. Appetite ratings, acylated ghrelin and peptide YY_{3-36} concentrations were measured throughout each trial. An ad libitum meal was provided at 7 h. Appetite was higher in Food-Def than Ex-Def from 4 to 8 h ($P = 0.033$) and tended to be higher across the entire 8 h trial ($P = 0.059$). However, energy intake at the ad libitum meal did not differ between trials ($P = 0.634$; Con 4376 (1634); Food-Def 4481 (1846); Ex-Def 4217 (1850) kJ). Acylated ghrelin was not related to changes in appetite but plasma PYY_{3-36} concentrations were higher in Ex-Def than Food-Def ($P < 0.05$) and negatively correlated with changes in appetite across the entire 8 h trial ($P = 0.037$). An energy deficit of ~1475 kJ stimulated compensatory increases in appetite when induced via calorie restriction but not when achieved by an acute bout of exercise. Appetite responses were associated with changes in plasma PYY_{3-36} but not acylated ghrelin concentrations and did not influence subsequent energy intake.

Introduction

Obesity is characterised by an excess accumulation of body fat and is associated with an increased prevalence of chronic diseases including type 2 diabetes, osteoarthritis, cardiovascular disease and some forms of cancer (Bray, 2004). Consequently, overweight and obesity has recently been classified as one of the top five global risk factors for mortality and one of the top 10 risk factors for morbidity (World Health Organization, 2009). However, weight loss as little as 3% has been associated with favourable changes in chronic disease risk factors and therefore represents a major public health priority (Donnelly et al., 2009).

For weight loss to occur, a sustained negative energy balance is required and is typically achieved by decreasing energy intake (i.e. dieting) and/or increasing energy expenditure (i.e. exercising). Although both interventions induce a negative energy balance, current research suggests that exercise and caloric restriction elicit contrasting homeostatic responses. In this regard, acute caloric restriction appears to stimulate rapid compensatory increases in appetite and energy intake that do not occur in response to equivalent energy deficits induced by exercise (Hubert, King, & Blundell, 1998; King et al., 2011a). Furthermore, King et al. (2011a) reported immediate decreases in circulating concentrations of the anorectic gut hormone PYY_{3-36} and increases in the orexigenic gut hormone acylated ghrelin in response to food restriction but no compensatory changes in response to exercise. Such findings suggest that these appetite-regulating gut hormones have a mediating role in the immediate appetite and energy intake responses to acute energy deficits but this requires further investigation.

Although these studies have provided interesting information regarding energy homeostasis and the regulation of appetite, large and abrupt methods of energy restriction have been employed as

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calorie intake was reduced by ~1820 kJ at a single meal (Hubert et al., 1998) and ~4820 kJ across two meals (King et al., 2011a). Such substantial decreases in energy intake at individual meals increases the likelihood that compensatory increases in appetite will occur and does not represent a practical strategy for energy restriction. In this regard, research has demonstrated that compensatory changes in gastrointestinal hormones and increases in appetite persist for at least 1 year after weight loss induced by a very low energy diet, despite increases in body weight (Sumithran et al., 2011).

The current UK government and American College of Sports Medicine (ACSM) guidelines recommend a minimum of 150 min per week of moderate intensity physical activity, spread over most days of the week (British Heart Foundation, 2009; Donnelly et al., 2009). This may be interpreted as five 30 min exercise bouts performed on separate days of the week and is considered to be sufficient to reduce chronic disease risk, prevent significant weight gain, and elicit modest weight loss in overweight and obese populations (Donnelly et al., 2009). The appetite and energy intake response to such a practical energy deficit achieved via exercise and food restriction is unknown. This requires further investigation as compensatory increases in appetite contribute to the difficulty of maintaining an energy deficit in current society where energy dense, highly palatable foods are abundant and easily accessible. Furthermore, increases in appetite are commonly cited as a reason for unsuccessful dieting (Ikeda, Lyons, Schwartzman, & Mitchell, 2004) and are inversely related to exercise-induced weight loss (King, Hopkins, Caudwell, Stubbs, & Blundell, 2008).

The purpose of this study was to investigate the appetite, acylated ghrelin, PYY_{3-36} and energy intake responses to a 30 min bout of moderate intensity cycling compared with an equivalent energy deficit achieved via caloric restriction. This study also enables further investigation into the sensitivity of the appetite-regulating system and the role of acylated ghrelin and PYY_{3-36} in energy homeostasis via the utilisation of small, yet practical, energy deficits. It was hypothesised that appetite and acylated ghrelin would increase, and that PYY_{3-36} would decrease in response to food restriction but that these variables would remain unaffected by exercise, resulting in a higher energy intake in the food restriction trial.

Methods

Participants

This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human participants were approved by the Loughborough University Ethics Advisory Committee (reference number: R12-P61). Written informed consents were obtained from all participants. Participants were male, non-smokers, not taking medication, weight stable for at least 6 months before the study and were not dieting. The physical characteristics of participants (mean (SD)) were as follows: age 24 (5) years, body mass index (BMI) 23.8 (2.7) $kg.m^{-2}$, body mass 75.3 (10.3) kg, body fat 14.2 (4.0)%, waist circumference 80.3 (6.6) cm, maximum oxygen uptake (VO_2 max) 55.4 (9.1) mL kg^{-1} min^{-1}.

Preliminary trials

Prior to main trials participants visited the laboratory for two preliminary trials. During the first visit, preliminary anthropometric measurements were collected and participants completed a maximal exercise test to determine VO_2 max. Height and body weight were measured and BMI was subsequently calculated. Body fat percentage was estimated via skinfold measurements of the biceps, triceps, sub-scapular and suprailiac sites (Durnin & Womersley, 1974) and waist circumference determined as the narrowest part of the torso between the xiphoid process and the iliac crest. Maximum oxygen uptake was determined using a continuous incremental cycle test to exhaustion as described previously (Deighton, Barry, Connon, & Stensel, 2013a). Acceptability of the food items to be provided during the main trials was assessed by completion of a food preference questionnaire. The questionnaire required participants to rate preselected food items on a scale ranging from 1 (dislike extremely) to 10 (like extremely). Any volunteers that scored ≤5 for any of the pre-selected food items to be presented were excluded from participating in the study.

Participants visited the laboratory on a second occasion for a familiarisation trial. Participants performed 30 min of continuous cycling exercise on an electromagnetically braked cycle ergometer (Lode Excalibur Sport V2, Groningen, Netherlands) at a work rate predicted to elicit 65% of VO_2 max. Samples of expired air were collected at 6, 18 and 30 min during exercise to monitor the intensity of the cycle bout, with adjustments made to the work rate if necessary. Heart rate (Polar T31; Polar Electro, Kempele, Finland) and ratings of perceived exertion (RPE) (Borg, 1973) were also measured at these times. Energy expenditure of exercise was calculated using the equation of Frayn (Frayn, 1983), for the determination of energy provision during the main trials.

Experimental protocol

Participants performed three 8 h experimental trials (control, exercise-induced energy deficit and diet-induced energy deficit) separated by 1 week in a counterbalanced Latin Square design. Participants completed a weighed food diary in the 24 h before the first main trial and replicated this before each subsequent trial. Alcohol, caffeine and strenuous physical activity were not permitted during this period. Participants arrived at the laboratory at 0800 h after an overnight fast of at least 10 h and exerted themselves minimally when travelling to the laboratory, using motorised transport when possible. Verbal confirmation of dietary and exercise standardisation was obtained at the beginning of each experimental trial.

During each trial, appetite perceptions (hunger, satisfaction, fullness and prospective food consumption) (Flint, Raben, Blundell, & Astrup, 2000) were assessed at baseline, 0.25, 0.5 h and every 30 min thereafter using 100 mm visual analogue scales. An overall appetite rating was calculated as the mean value of the four appetite perceptions after inverting the values for satisfaction and fullness (Stubbs et al., 2000).

Test meals

At 1 h (~9 am) participants were provided with a standardised breakfast, which consisted of toasted white wheatgerm bread, margarine, strawberry jam, banana and orange juice. The macronutrient content of the meal was 72.9% carbohydrate, 9.5% protein and 17.6% fat. A standardised lunch was provided at 4 h (~12 pm) and consisted of a tuna and mayonnaise sandwich, salted crisps, chocolate muffin and green apple. The macronutrient content of the meal was 47% carbohydrate, 17.6% protein and 35.4% fat.

Energy deficits

Participants rested within the laboratory throughout all trials (sitting reading, working at a desk or watching television), except from 0 to 0.5 h during the exercise-induced energy deficit (Ex-Def) trial where participants replicated the exercise bout performed during the familiarisation trial. To calculate the net energy expenditure of exercise (gross energy expenditure of exercise minus energy expenditure at rest), expired gas was collected into Douglas bags for 5 min every 10 min between 0 and 0.5 h during the control (Con) and diet-induced energy deficit (Food-Def) trials (Frayn, 1983).

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