



LEEDS
BECKETT
UNIVERSITY

Citation:

Deighton, K and Karra, E and Batterham, RL and Stensel, DJ (2013) Appetite, energy intake, and PYY3-36 responses to energy-matched continuous exercise and submaximal high-intensity exercise. *Applied physiology, nutrition, and metabolism = Physiologie appliquee, nutrition et metabolisme*, 38 (9). 947 - 952. ISSN 1715-5312 DOI: <https://doi.org/10.1139/apnm-2012-0484>

Link to Leeds Beckett Repository record:

<https://eprints.leedsbeckett.ac.uk/id/eprint/209/>

Document Version:

Article (Accepted Version)

The aim of the Leeds Beckett Repository is to provide open access to our research, as required by funder policies and permitted by publishers and copyright law.

The Leeds Beckett repository holds a wide range of publications, each of which has been checked for copyright and the relevant embargo period has been applied by the Research Services team.

We operate on a standard take-down policy. If you are the author or publisher of an output and you would like it removed from the repository, please [contact us](#) and we will investigate on a case-by-case basis.

Each thesis in the repository has been cleared where necessary by the author for third party copyright. If you would like a thesis to be removed from the repository or believe there is an issue with copyright, please contact us on openaccess@leedsbeckett.ac.uk and we will investigate on a case-by-case basis.

Appetite, energy intake and PYY₃₋₃₆ responses to energy-matched continuous exercise and submaximal high intensity exercise.

Kevin Deighton¹, Efthimia Karra², Rachel Louise Batterham², David John Stensel¹

¹School of Sport, Exercise and Health Sciences, Loughborough University, UK

²Centre for Obesity Research, Department of Medicine, University College London, UK

Correspondence

Kevin Deighton

School of Sport, Exercise and Health Sciences

Loughborough University,

Leicestershire

LE11 3TU

United Kingdom

Phone: +44(0)1509 226352

Fax: +44(0)1509 226301

E-mail: K.Deighton@lboro.ac.uk

E-mail addresses

Efthimia Karra: e.karra@ucl.ac.uk

Rachel Louise Batterham: r.batterham@ucl.ac.uk

David John Stensel: D.J.Stensel@lboro.ac.uk

Abstract

High intensity intermittent exercise induces similar physiological adaptations as energy-matched continuous exercise but the comparative appetite and energy balance responses are unknown. Twelve healthy males (mean(SD): age 22(3) years, body mass index 23.7(3.0) kg.m⁻², maximum oxygen uptake (VO₂ max) 52.4(7.1) mL.kg⁻¹.min⁻¹) completed three 8h trials (control, steady state exercise (SSE), high intensity intermittent exercise (HIIE)) separated by one week. Trials commenced upon completion of a standardised breakfast. Exercise was performed from 2-3h. Sixty minutes of cycling at 59.5(1.6)% of VO₂ max was performed in SSE. In HIIE, ten 4 min cycling intervals were completed at 85.8(4.0)% of VO₂ max with 2 min rest between each interval. A standardised lunch and ad libitum afternoon meal were provided at 3.75 and 7h. Appetite ratings and peptide YY₃₋₃₆ concentrations were measured throughout each trial. Appetite was acutely suppressed during exercise but more so during HIIE (P<0.05). Peptide YY₃₋₃₆ concentrations increased significantly upon cessation of exercise in SSE (P=0.002) but were highest in the hours after exercise in HIIE (P=0.05). Exercise energy expenditure was not different between HIIE and SSE (P=0.649) but perceived exertion was higher in HIIE (P<0.0005). Ad libitum energy intake did not differ between trials (P=0.833). Therefore *relative* energy intake (energy intake minus the net energy expenditure of exercise) was lower in SSE and HIIE than control (P<0.0005; control 4759(1268); SSE 2362(1224), HIIE 2523(1402) kJ). An acute bout of energy-matched continuous exercise and high intensity intermittent exercise were equally effective at inducing an energy deficit without stimulating compensatory increases in appetite.

Key words: peptide YY, appetite-regulating hormones, exercise-induced anorexia, compensation, energy balance, high intensity exercise, steady state exercise

Introduction

High intensity intermittent exercise (HIIE) appears to induce many similar health and performance-related physiological adaptations as traditional continuous steady state exercise (SSE) (Gibala et al. 2012). However, the effect of HIIE on body composition remains a contentious issue, with studies showing greater (Irving et al. 2008; Trapp et al. 2008; Tremblay et al. 1994), lesser (Helgerud et al. 2007; Nybo et al. 2010) or no difference (Moholdt et al. 2009; Rognmo et al. 2004; Tjønnå et al. 2008; Warburton et al. 2005; Wisløff et al. 2007) in the weight and fat loss responses to HIIE compared with SSE.

Despite such conflicting findings, it has been repeatedly postulated that HIIE has the potential to facilitate greater fat loss than SSE due to a greater reduction in appetite during the post-exercise period (Boutcher 2011; Heydari et al. 2012; Trapp et al. 2008; Tremblay et al. 1994). However, thus far, only one published study has investigated the appetite response to HIIE and demonstrated that appetite perceptions and daily energy balance were higher after six 30 s Wingate tests than after 60 min of continuous cycling at ~68% of VO_2 max. The mechanisms underlying this increase in appetite are unknown but occurred despite a lower exercise energy expenditure and greater appetite suppression during the sprint exercise (Deighton et al. 2012).

The practical application of such supramaximal exercise has recently been questioned due to the high levels of nausea previously experienced by participants and the increased risk of acute cardiovascular events during such intense exercise (Deighton et al. 2012; Gibala et al. 2012; Richards et al. 2010; Whyte et al. 2010). Conversely, high volume HIIE involving repeated 4 min exercise intervals at ~ 90 % of VO_2 max appears to be well-tolerated, even in clinical populations (Moholdt et al. 2009; Rognmo et al. 2004; Tjønnå et al. 2008; Wisløff et al. 2007).

High volume HIIE represents an intermediate between SSE and supramaximal interval exercise (Talanian et al. 2007). It is therefore unclear whether this exercise mode will stimulate compensatory increases in appetite similar to supramaximal interval exercise (Deighton et al. 2012), or whether the appetite response will be more akin to continuous submaximal exercise, which does not seem to elicit any compensatory increases in appetite (King et al. 2010; 2011). Understanding the appetite response to popular exercise protocols is important in determining the most effective method of inducing a negative energy balance without stimulating compensatory increases in appetite, which are inversely associated with exercise-induced weight loss (King et al. 2008). Furthermore, high volume HIIE allows comparisons with SSE to be matched for duration, total work and energy expenditure. Therefore, investigations into this exercise protocol may help to elucidate the influence of exercise mode on appetite and energy intake responses.

The purpose of this investigation was to examine the appetite and energy intake responses to ten 4 min cycling bouts at 85-90 % of VO_2 max compared with 60 min of continuous cycling at 60 % of VO_2 max. Circulating concentrations of peptide YY₃₋₃₆ (PYY₃₋₃₆) were also measured, as previous research suggests that this anorexigenic gut hormone may be implicated in the appetite response to exercise (King et al. 2011; Ueda et al. 2009a). We hypothesised that high intensity intermittent cycling would stimulate compensatory increases in appetite during the hours after exercise but that this would not occur in response to continuous steady state cycling.

Methods

Participants

Following the approval of Loughborough University's Ethics Advisory Committee, twelve healthy males (18 - 30 years) gave their written informed consent to participate. Participants

were 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 22 (3) years, body mass index (BMI) 23.7 (3.0) kg.m⁻², body mass 75.0 (12.0) kg, body fat 13.7 (3.9) %, waist circumference 78.6 (6.4) cm, maximum oxygen uptake 52.4 (7.1) mL.kg⁻¹.min⁻¹.

Participant Screening

Prior to the main trials participants visited the laboratory to undergo screening, familiarization, preliminary anthropometric measurements and a maximal exercise test to determine VO₂ max. Height and body weight were measured and body mass index subsequently calculated. Body fat percentage was estimated via skinfold measurements (Durnin and 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 et al. 2012). The test started at a work rate of 95 W with increments of 35 W every 3 min until volitional fatigue. Expired gas samples were collected into Douglas bags during the third minute of each stage and during the final minute of the test for the determination of oxygen uptake. A food preference questionnaire was completed to ensure acceptability of all food items to be consumed during the main trials.

Experimental Protocol

Participants performed three experimental trials (control, steady state exercise and high intensity intermittent exercise) separated by one week in a counterbalanced Latin square design. All trials were identical except that 60 min of exercise was performed in the steady state exercise (SSE) and high intensity intermittent exercise (HIIE) trials. Exercise commenced at 2 h and finished at 3 h. No exercise was performed in the control trial (CON).

Participants rested within the laboratory for the remainder of the day in all trials (sitting reading, working at a desk or watching television).

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 8.30 am after an overnight fast of at least 10 h and exerted themselves minimally when travelling to the laboratory, using motorized transport when possible.

During each trial, appetite perceptions (hunger, satisfaction, fullness and prospective food consumption (PFC)) (Flint et al. 2000) were assessed at baseline, 0.25, 2 h and every 30 min thereafter using 100 mm visual analogue scales with descriptors anchored at each end. An overall appetite score was calculated as the mean value of the four appetite perceptions after inverting the values for satisfaction and fullness (Stubbs et al. 2000).

Steady State Exercise Session

Participants performed 60 min of continuous cycling exercise on an electromagnetically braked cycle ergometer (Lode Excalibur Sport V2, Groningen, Netherlands) at a work rate predicted to elicit 60 % of VO_2 max. Samples of expired air were collected at 10, 20, 30, 45 and 60 min during exercise to monitor the intensity of the exercise, with adjustments made to the work rate if necessary. Heart rate and ratings of perceived exertion (RPE) (Borg 1973) were also assessed at these times. Energy expenditure of the session was subsequently calculated from VO_2 and VCO_2 values using the equation of Frayn (1983).

High Intensity Intermittent Exercise Session

The high intensity exercise session consisted of ten 4 min cycling bouts at a work rate predicted to elicit 85 – 90 % of VO_2 max separated by 2 min of rest. Samples of expired air

were collected during the last minute of repetition number 1, 3 5, 8 and 10 to monitor the intensity of the exercise and to calculate the energy expenditure of the session (Frayn 1983). Adjustments were made to the work rate based on the intensity and the participants' tolerance to the exercise. Heart rate and RPE were monitored during the final minute of each repetition.

Net Energy Expenditure of Exercise

In order to estimate the net energy expenditure of exercise (gross energy expenditure of exercise minus resting energy expenditure), expired air was collected into Douglas bags for 5 min every 15 min from 2 - 3 h during the control trial to quantify resting energy expenditure (Frayn 1983). Data from previous experiments suggested that the net energy expenditure of exercise should be comparable between the high intensity and steady state exercise sessions (Leggate et al. 2010; Warren et al. 2009).

Standardised test meals and ad libitum meal

After the collection of baseline measures, participants were provided with a standardised breakfast, which consisted of toasted white wheatgerm bread, margarine, strawberry jam, banana and orange juice. Participants were instructed to consume the breakfast within 15 min and the 8 h trial commenced upon completion of the meal. The macronutrient content of the meal was 72.9 % carbohydrate, 9.5 % protein and 17.6 % fat. A standardised lunch was provided at 3.75 h 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. The breakfast meal provided 30 % and the lunch meal 35 % of the estimated daily energy needs for each individual for a sedentary day, which was calculated using the Mifflin-St Jeor equation and a physical activity factor of 1.4 (Mifflin et al. 1990). The mean (SD) calculated daily energy need for the participants in this study was 10,276 (806) kJ.

At 7 h an ad libitum meal was provided, consisting of fusilli pasta that was cooked in a microwave for 12 min in unsalted water and served in a bolognaise sauce. The macronutrient composition of the meal was 77.5 % carbohydrate, 13.8 % protein and 8.7 % fat. Participants were provided with a small bowl, which was repeatedly filled with the pasta meal before the participant had emptied it in an attempt to blind the participant to the amount of food eaten. No time limit was set for eating and participants were instructed to eat until 'comfortably full'. Each participant consumed the meal separately in the presence of a sole experimenter and any discussions about food were avoided. Food intake was determined by weighing the food presented before and after eating and energy intake was subsequently determined using manufacturers' values.

Water was available ad libitum throughout each trial.

Blood Sampling

Upon arrival to the laboratory, participants rested in a semi-supine position and a cannula (Venflon, Becton Dickinson, Helsinborg, Sweden) was inserted into an antecubital vein. Blood samples were collected into pre-chilled syringes containing 10 µl DPP-IV inhibitor (Millipore, Watford, UK) per mL of blood at: baseline, 2, 3, 3.75, 5, 6, 7 and 8 h. Syringes were immediately inverted and the blood dispensed into pre-chilled 2 mL EDTA tubes containing aprotonin (Nordic Pharma, Reading, UK), 500 KIU per mL of blood. Blood tubes were promptly centrifuged at $1165 \times g$ for 10 min at 4 °C (Heraeus Labofuge 400R, Thermo Electron, Osterode, Germany). The plasma supernatant was then aliquoted into 2 mL Eppendorf tubes prior to storage at -20°C for later analysis of PYY₃₋₃₆ concentrations.

All samples were collected in the semi-supine position. Measurements of haemoglobin and haematocrit were taken to estimate changes in plasma volume (Dill and Costill 1974). The

mean coefficient of variation for blood haemoglobin and haematocrit measures was 0.7 % and 0.8 %, respectively.

Biochemical Analysis

Plasma concentrations of PYY₃₋₃₆ were determined using a commercially available radioimmunoassay (Millipore, Watford, UK). All samples were run in duplicate. To eliminate interassay variation, samples from each participant were analysed in the same run. The within batch coefficient of variation for the assay was 7.2 %.

Statistical Analysis

Data was analysed using IBM SPSS statistics version 19 for Windows. Time-averaged area under the curve (AUC) values were calculated using the trapezoidal method. Exercise responses in SSE and HIIT were compared using Students paired t-tests. One-way repeated measures ANOVA was used to assess trial-based differences in energy intake at the ad libitum meal as well as baseline and AUC values for appetite and PYY₃₋₃₆. Repeated measures, two-factor ANOVA was used to examine differences between trials over time for appetite and PYY₃₋₃₆. Where significant main and interaction effects were found, post-hoc analysis was performed using Holm-Bonferroni correction for multiple comparisons.

Correction of PYY₃₋₃₆ values for changes in plasma volume did not alter the interpretation of the results; therefore, for simplicity, the unadjusted values are presented. Statistical significance for this study was accepted as $P \leq 0.05$. Results in text and tables are presented as mean (SD). Graphical representations of results are presented as mean (SEM) to avoid distortion of the graphs. Based on previous data from our laboratory (Deighton et al. 2012), a sample size of 12 participants was determined as sufficient to detect a 10 % difference in appetite perceptions during the post-exercise period. This calculation was performed using G*power with an alpha value of 5 % and a power of 80 % (Faul et al. 2007).

Results

Exercise Responses

The exercise responses for SSE and HIIE are detailed in Table 1. Work rate, oxygen consumption, heart rate, RPE and RER were significantly higher during HIIE than SSE (all $P < 0.0005$). However, due to the rest periods during HIIE, the net energy expenditure of exercise and external work performed did not differ between protocols (both $P > 0.64$).

Appetite

Overall appetite scores did not differ significantly between trials at baseline ($P = 0.345$). Two-factor ANOVA revealed a main effect of time and a trial x time interaction for appetite perceptions (both $P < 0.0005$) but no main effect of trial (Figure 1). Post-hoc analysis of trial x time interactions revealed suppressed appetite during and upon completion of exercise in HIIE compared with CON ($P < 0.05$). One-way ANOVA revealed a trend towards a main effect of trial for appetite AUC from 0 – 3.5 h, indicating lower appetite in HIIE than CON ($P = 0.06$). There were no between trial differences in appetite AUC for 3.5 – 8 h and 0 – 8 h (Table 2).

Energy Intake

Absolute energy intake at the ad libitum meal was not significantly different between trials ($P = 0.833$; CON 4759 (1268); SSE 4813 (1316); HIIE 4952 (1351) kJ). One-way ANOVA revealed a significant main effect of trial for relative energy intake (REI) (energy intake minus the net energy expenditure of exercise), demonstrating lower REI in SSE than CON (50.3%; $P < 0.0005$) and HIIE than CON (44.7%; $P < 0.0005$) but no difference between SSE and HIIE ($P = 0.625$; CON 4759 (1268); SSE 2362 (1224); HIIE 2523 (1402) kJ). Closer

inspection of the data revealed that all 12 participants had the highest REI during CON. Seven participants had a higher REI in HIIE than SSE, while 5 participants had a higher REI in SSE than HIIE.

Plasma PYY₃₋₃₆ concentrations

Due to problems with venous cannulation, blood sampling was not possible for one participant. Therefore, data for PYY₃₋₃₆ are presented from 11 participants. Fasting PYY₃₋₃₆ concentrations did not differ significantly between trials (P = 0.356; CON 76.5 (13.4); SSE 72.5 (13.7); HIIE 71.9 (9.5) pg.mL⁻¹). Two-factor ANOVA revealed a significant trial (P = 0.019), time (P < 0.0005) and trial x time interaction (P = 0.025) for delta PYY₃₋₃₆ concentrations (Figure 2). Post-hoc analysis of between trial differences revealed higher plasma PYY₃₋₃₆ concentrations in HIIE than CON (P = 0.015). Post-hoc analysis of trial x time interactions demonstrated elevated PYY₃₋₃₆ concentrations upon completion of exercise in SSE compared with CON (P = 0.002). One-way ANOVA revealed a significant main effect of trial for area under the delta PYY₃₋₃₆ concentration versus time curve for 0 – 3.75 h, 3.75 – 8 h and for the total 8 h trial (Table 3).

Peptide YY₃₋₃₆ responses to exercise were highly variable between participants with delta AUC values for the 8 h trial being highest in HIIE for 6 participants and SSE for 5 participants. Values were lowest in the control trial for 8 of 11 participants.

Discussion

The primary finding of this investigation is that an acute bout of SSE and high volume HIIE did not stimulate any compensatory increases in appetite or energy intake, which resulted in a substantial negative daily energy balance in both exercise trials relative to control. Plasma

PYY₃₋₃₆ concentrations were increased in the hours after exercise but to a greater extent after HIIE.

It is well established that appetite is acutely suppressed during exercise $\geq 60\%$ of VO₂ max (Blundell et al. 2003). However, the comparative appetite responses to exercise intensities above this threshold remain unclear (Deighton et al. 2012; Imbeault et al. 1997; Thompson et al. 1988; Ueda et al. 2009a). In the present study, appetite was suppressed during both exercise bouts but to a greater extent during HIIE. This supports previous findings from supramaximal interval exercise (Deighton et al. 2012) and suggests that modern day HIIE protocols above $\sim 85\%$ of VO₂ max elicit greater appetite suppression during exercise than moderate intensity exercise at 60 – 65 % of VO₂ max. The lack of clarity among previous findings is likely to be a result of the lower exercise intensities employed (Imbeault et al. 1997; Thompson et al. 1988; Ueda et al. 2009a).

The HIIE protocol employed in this study did not stimulate any compensatory increases in appetite during the hours after exercise, which suggests that previously observed increases in appetite after supramaximal interval exercise (Deighton et al. 2012) are likely to be a result of the extreme intensity rather than the intermittent nature of exercise. It remains plausible that a threshold exercise intensity may exist for the stimulation of appetite during the post-exercise period but this requires further investigation.

In accordance with previous research, circulating concentrations of PYY₃₋₃₆ increased upon completion of exercise (Broom et al. 2009; King et al. 2011; Ueda et al. 2009a). However, this response was highly variable between participants and a statistically significant effect was only found upon completion of SSE, despite appetite-suppression being greatest upon completion of HIIE. Although surprising, this supports previous findings (Deighton et al. 2012) and suggests that appetite suppression during exercise is not solely dependent on

increases in PYY₃₋₃₆. Furthermore, plasma PYY₃₋₃₆ concentrations were highest in the hours after HIIE but appetite did not differ between trials. These findings highlight the complex nature of appetite regulation, which involves the integration of a wide range of neuroendocrine and psychological factors (Evero et al. 2012; Morton et al. 2006) and is therefore unlikely to be explained by the measurement of a single appetite-regulating hormone. Nevertheless, increases in circulating PYY₃₋₃₆ in response to exercise represent a physiological adjustment to promote satiety, which contrasts the decreases observed with food restriction (King et al. 2011).

The mechanisms underlying such increases in PYY₃₋₃₆ in response to exercise are unknown. However, it seems reasonable to postulate that such changes may be related to an increase in sympathetic nervous system activity, which occurs during exercise (Hagberg et al. 1979) and has been shown to stimulate PYY secretion (Brechet et al. 2001; Zhang et al. 1993).

Sympathetic activity increases with exercise intensity (Perini et al. 1989), which may explain the more-prolonged increase in PYY₃₋₃₆ after HIIE. However, this theory does not explain the significant increase in PYY₃₋₃₆ concentrations immediately upon completion of SSE but not HIIE. This confliction may be a result of intestinal blood flow, which decreases with exercise intensity (Clausen 1977; Gil et al. 1998) and may have therefore reduced the transport of PYY₃₋₃₆ into the peripheral circulation immediately after exercise in HIIE (Mailman 1982). These are the first postulations to be made regarding the mechanisms of exercise-induced increases in PYY and although this would explain the findings of the present study, it must be noted that this is speculation and requires future investigation.

Energy intake at the ad libitum meal did not differ between trials. In combination with the appetite data, this would suggest that the exercise protocols utilised in this study did not stimulate a physiological or conscious drive to eat. The absence of any compensatory increases in energy intake resulted in a substantial negative daily energy balance in both

exercise trials compared with control. However, for the same energy expenditure of exercise, HIIE was significantly more strenuous than SSE, as reflected by higher heart rates and perceived exertion. Therefore, although a single bout of HIIE has been demonstrated to be more enjoyable than SSE (Bartlett et al. 2011), exercise practitioners must also be aware that HIIE elicits greater physical stress than energy-matched SSE. . Research suggests that SSE and high volume HIIE also induce similar improvements in VO₂ max, maximal mitochondrial enzyme activity and fat oxidation during submaximal exercise (Perry et al. 2008; Talanian et al. 2007; 2010). Therefore a beneficial and sustainable approach to exercise training may be to include a combination of SSE and HIIE based on the preferences of individual participants.

Some authors have postulated that HIIE may be more beneficial for fat loss than SSE due to greater post-exercise decreases in appetite and increases in resting energy expenditure (Boutcher 2011; Heydari et al. 2012; Trapp et al. 2008; Tremblay et al. 1994). However, despite greater appetite suppression during HIIE, present and previous research (Deighton et al. 2012) suggests that post-exercise appetite perceptions are not reduced compared with SSE. The HIIE protocols investigated thus far represent both extremes of the HIIE spectrum, i.e. high volume, submaximal interval exercise in the present study and very low volume, supramaximal interval exercise in previous work (Deighton et al. 2012). We are therefore confident that HIIE does not elicit lower appetite perceptions in the hours after exercise compared with SSE. Additionally, although not measured in the present study, previous research indicates that energy-matched SSE and HIIE induce comparable changes in resting metabolism during the recovery from exercise (McGarvey et al. 2005; Warren et al. 2009). These findings suggest that any additional fat loss benefits of HIIE are mediated by other unknown mechanisms.

The findings of the present study are limited by the population sample, as participants were all healthy young males. Although previous research suggests that exercise elicits similar appetite and energy intake responses in lean and obese participants (Ueda et al. 2009b), it remains important to perform further investigations in overweight and obese populations as this is where weight management strategies hold the most clinical relevance.

In conclusion, an acute bout of SSE and high volume HIIE did not stimulate any compensatory increases in appetite or energy intake during the hours after exercise. This contrasts previously observed increases in appetite after supramaximal interval exercise and suggests that a threshold exercise intensity may exist for the stimulation of appetite after exercise. Despite speculation from previous authors, this study presents evidence that a single bout of HIIE does not elicit greater appetite suppression than SSE during the post-exercise period. Further research is required to investigate whether differences in appetite occur in response to repeated bouts of HIIE and SSE.

Acknowledgements

The authors thank Hannah Bateson and Rachel Malcolm for their help with the data collection and all of the volunteers for their participation in this study.

References

- Bartlett, J. D., Close, G. L., MacLaren, D. P. M., Gregson, W., Drust, B., and Morton, J. P. (2011). High-intensity interval running is perceived to be more enjoyable than moderate-intensity continuous exercise: implications for exercise adherence. *J Sports Sci* **29**: 547–53. DOI: 10.1080/02640414.2010.545427.
- Blundell, J. E., Stubbs, R. J., Hughes, D. A., Whybrow, S., and King, N. A. (2003). Cross talk between physical activity and appetite control: does physical activity stimulate appetite? *Proc Nutr Soc* **62**: 651–61. DOI: 10.1079/PNS2003286.
- Borg, G. A. (1973). Perceived exertion: a note on 'history' and methods. *Med Sci Sports* **5**: 90-3. PMID: 4721012.
- Boutcher, S. H. (2011). High-intensity intermittent exercise and fat loss. *J Obes* 868305. DOI: 10.1155/2011/868305.
- Brechet, S., Plaisancié, P., Dumoulin, V., Chayvialle, J. A., Cuber, J. C., and Claustre, J. (2001). Involvement of beta1- and beta2- but not beta3-adrenoceptor activation in adrenergic PYY secretion from the isolated colon. *J Endocrinol* **168**: 177–83. DOI: 10.1677/joe.0.1680177.
- Broom, D. R., Batterham, R. L., King, J. A, and Stensel, D. J. (2009). Influence of resistance and aerobic exercise on hunger, circulating levels of acylated ghrelin, and peptide YY in healthy males. *Am J Physiol Regul Integr Comp Physiol* **296**: R29–35. DOI: 10.1152/ajpregu.90706.2008.
- Clausen, J. P. (1977). Effect of physical training on cardiovascular adjustments to exercise in man. *Physiol Rev* **57**: 779–815. PMID: 333481.

Deighton, K., Barry, R., Connon, C. E., and Stensel, D. J. (2012). Appetite, gut hormone and energy intake responses to low volume sprint interval and traditional endurance exercise. *Eur J Appl Physiol*: DOI: 10.1007/s00421-012-2535-1 (Epub ahead of print).

Dill, D. B., and Costill, D. L. (1974). Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol* **37**: 247–8. PMID: 4850854.

Durnin, J. V., and Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *Br J Nutr* **32**: 77–97. DOI: 10.1079/BJN19740060.

Evero, N., Hackett, L.C., Clark, R.D., Phelan, S., and Hagobian, T.A. (2012). Aerobic exercise reduces neuronal responses in food reward brain regions. *J Appl Physiol* **112**: 1612-9. DOI: 10.1152/jappphysiol.01365.2011.

Faul, F., Erdfelder, E., Lang, A.G., and Buchner, A. (2007). G*Power 3: a flexible statistical power analysis program for the social, behavioural, and biomedical sciences. *Behav Res Methods* **39**: 175-91. DOI: 10.3758/BF03193146.

Flint, A., Raben, A., Blundell, J. E., and Astrup, A. (2000). Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *Int J Obes Relat Metab Disord* **24**: 38–48. DOI: 10.1038/sj.ijo.0801083.

Frayn, K. N. (1983). Calculation of substrate oxidation rates in vivo from gaseous exchange. *J Appl Physiol* **55**: 628–34. PMID: 6618956.

Gibala, M. J., Little, J. P., Macdonald, M. J., and Hawley, J. A. (2012). Physiological adaptations to low-volume, high-intensity interval training in health and disease. *J Physiol* **590**: 1077–84. DOI: 10.1113/jphysiol.2011.224725.

Gil, S. M., Yazaki, E., and Evans, D. F. (1998). Aetiology of running-related gastrointestinal dysfunction. How far is the finishing line? *Sports Med* **26**: 365–78. DOI: 10.2165/00007256-199826060-00001.

Hagberg, J. M., Hickson, R. C., McLane, J. A., Ehsani, A. A., and Winder, W. W. (1979). Disappearance of norepinephrine from the circulation following strenuous exercise. *J Appl Physiol* **47**: 1311–4. PMID: 536302.

Helgerud, J., Høydal, K., Wang, E., Karlsen, T., Berg, P., Bjerkaas, M., et al. (2007). Aerobic high-intensity intervals improve VO₂max more than moderate training. *Med Sci Sports Exerc* **39**: 665–71. DOI: 10.1249/mss.0b013e3180304570.

Heydari, M., Freund, J., and Boutcher, S. H. (2012). The effect of high-intensity intermittent exercise on body composition of overweight young males. *J Obes* 480467. DOI: 10.1155/2012/480467.

Imbeault, P., Saint-Pierre, S., Alméras, N., and Tremblay, A. (1997). Acute effects of exercise on energy intake and feeding behaviour. *Br J Nutr* **77**: 511–21. DOI: <http://dx.doi.org/10.1079/BJN19970053>.

Irving, B. A., Davis, C. K., Brock, D. W., Weltman, J. Y., Swift, D., Barrett, E. J., et al. (2008). Effect of exercise training intensity on abdominal visceral fat and body composition. *Med Sci Sports Exerc* **40**: 1863–72. DOI: 10.1249/MSS.0b013e3181801d40.

King, J. A., Miyashita, M., Wasse, L. K., and Stensel, D. J. (2010). Influence of prolonged treadmill running on appetite, energy intake and circulating concentrations of acylated ghrelin. *Appetite* **54**: 492–8. DOI: 10.1016/j.appet.2010.02.002.

King, J. A., Wasse, L. K., Ewens, J., Crystallis, K., Emmanuel, J., Batterham, R. L., et al. (2011). Differential acylated ghrelin, peptide YY3-36, appetite, and food intake responses to equivalent energy deficits created by exercise and food restriction. *J Clin Endocrinol Metab* **96**: 1114–21. DOI: 10.1210/jc.2010-2735.

King, N. A., Hopkins, M., Caudwell, P., Stubbs, R. J., and Blundell, J. E. (2008). Individual variability following 12 weeks of supervised exercise: identification and characterization of compensation for exercise-induced weight loss. *Int J Obes* **32**: 177–84. DOI: 10.1038/sj.ijo.0803712.

Leggate, M., Nowell, M.A., Jones, S.A., and Nimmo, M.A. (2010). The response of interleukin-6 and soluble interleukin-6 receptor isoforms following intermittent high intensity and continuous moderate intensity cycling. *Cell Stress Chaperones* **15**: 827-33. DOI: 10.1007/s12192-010-0192-z.

Mailman, D. (1982). Blood flow an intestinal absorption. *Fed Proc* **41**: 2096–100. PMID: 6122606.

McGarvey, W., Jones, R., and Petersen, S. (2005). Excess post-exercise oxygen consumption following continuous and interval cycling exercise. *Int J Sport Nutr Exerc Metab* **15**: 28–37. PMID: 15902987.

Mifflin, M. D., St Jeor, S. T., Hill, L. A., Scott, B. J., Daugherty, S. A., and Koh, Y. O. (1990). A new predictive equation for resting energy expenditure in healthy individuals. *Am J Clin Nutr* **51**: 241–7. PMID: 2305711.

Moholdt, T. T., Amundsen, B. H., Rustad, L. A., Wahba, A., Løvø, K. T., Gullikstad, L. R., et al. (2009). Aerobic interval training versus continuous moderate exercise after coronary

artery bypass surgery: a randomized study of cardiovascular effects and quality of life. *Am Heart J* **158**: 1031–7. DOI: 10.1016/j.ahj.2009.10.003.

Morton, G. J., Cummings, D. E., Baskin, D. G., Barsh, G. S., and Schwartz, M. W. (2006). Central nervous system control of food intake and body weight. *Nature*, **443**: 289–95. DOI: 10.1038/nature05026.

Nybo, L., Sundstrup, E., Jakobsen, M. D., Mohr, M., Hornstrup, T., Simonsen, L., et al. (2010). High-intensity training versus traditional exercise interventions for promoting health. *Med Sci Sports Exerc* **42**: 1951–8. DOI: 10.1249/MSS.0b013e3181d99203.

Perini, R., Orizio, C., Comandè, A., Castellano, M., Beschi, M., and Veicsteinas, A. (1989). Plasma norepinephrine and heart rate dynamics during recovery from submaximal exercise in man. *Eur J Appl Physiol Occup Physiol* **58**: 879–83. DOI: 10.1007/BF02332222.

Perry, C. G. R., Heigenhauser, G. J. F., Bonen, A., and Spriet, L. L. (2008). High-intensity aerobic interval training increases fat and carbohydrate metabolic capacities in human skeletal muscle. *Appl Physiol Nutr Metab* **33**: 1112–23. DOI: 10.1139/H08-097.

Richards, J. C., Johnson, T. K., Kuzma, J. N., Lonac, M. C., Schweder, M. M., Voyles, W. F., et al. (2010). Short-term sprint interval training increases insulin sensitivity in healthy adults but does not affect the thermogenic response to beta-adrenergic stimulation. *J Physiol* **588**: 2961–72. DOI: 10.1113/jphysiol.2010.189886.

Rognmo, Ø., Hetland, E., Helgerud, J., Hoff, J., and Slørdahl, S. A. (2004). High intensity aerobic interval exercise is superior to moderate intensity exercise for increasing aerobic capacity in patients with coronary artery disease. *Eur J Cardiovasc Prev Rehabil* **11**: 216–222. DOI: 10.1097/01.hjr.0000131677.96762.0c.

Stubbs, R. J., Hughes, D. A., Johnstone, A. M., Rowley, E., Reid, C., Elia, M., et al. (2000). The use of visual analogue scales to assess motivation to eat in human subjects: a review of their reliability and validity with an evaluation of new hand-held computerized systems for temporal tracking of appetite ratings. *Br J Nutr* **84**: 405–15. DOI: 10.1017/S0007114500001719.

Talanian, J. L., Galloway, S. D. R., Heigenhauser, G. J. F., Bonen, A., and Spriet, L. L. (2007). Two weeks of high-intensity aerobic interval training increases the capacity for fat oxidation during exercise in women. *J Appl Physiol* **102**: 1439–1447. DOI: 10.1152/jappphysiol.01098.2006.

Talanian, J. L., Holloway, G. P., Snook, L. A., Heigenhauser, G. J. F., Bonen, A., and Spriet, L. L. (2010). Exercise training increases sarcolemmal and mitochondrial fatty acid transport proteins in human skeletal muscle. *Am J Physiol Endocrinol Metab* **299**: E180–8. DOI: 10.1152/ajpendo.00073.2010.

Thompson, D. A., Wolfe, L. A., and Eikelboom, R. (1988). Acute effects of exercise intensity on appetite in young men. *Med Sci Sports Exerc* **20**: 222–7. DOI: 10.1249/00005768-198806000-00002.

Tjønnå, A. E., Lee, S. J., Rognum, Ø., Stølen, T. O., Bye, A., Haram, P. M., et al. (2008). Aerobic interval training versus continuous moderate exercise as a treatment for the metabolic syndrome: a pilot study. *Circulation* **118**: 346–54. DOI: 10.1161/CIRCULATIONAHA.108.772822.

Trapp, E. G., Chisholm, D. J., Freund, J., and Boutcher, S. H. (2008). The effects of high-intensity intermittent exercise training on fat loss and fasting insulin levels of young women. *Int J Obes* **32**: 684–91. DOI: 10.1038/sj.ijo.0803781.

Tremblay, A., Simoneau, J. A., and Bouchard, C. (1994). Impact of exercise intensity on body fatness and skeletal muscle metabolism. *Metabolism* **43**: 814–8. DOI: 10.1016/0026-0495(94)90259-3.

Ueda, S., Yoshikawa, T., Katsura, Y., Usui, T., and Fujimoto, S. (2009a). Comparable effects of moderate intensity exercise on changes in anorectic gut hormone levels and energy intake to high intensity exercise. *J Endocrinol* **203**: 357–64. DOI: 10.1677/JOE-09-0190.

Ueda, S., Yoshikawa, T., Katsura, Y., Usui, T., Nakao, H., and Fujimoto, S. (2009b). Changes in gut hormone levels and negative energy balance during aerobic exercise in obese young males. *J Endocrinol* **201**: 151–9. DOI: 10.1677/JOE-08-0500.

Warburton, D. E. R., McKenzie, D. C., Haykowsky, M. J., Taylor, A., Shoemaker, P., Ignaszewski, A. P., et al. (2005). Effectiveness of high-intensity interval training for the rehabilitation of patients with coronary artery disease. *Am J Cardiol* **95**: 1080–4. DOI: 10.1016/j.amjcard.2004.12.063.

Warren, A., Howden, E. J., Williams, A. D., Fell, J. W., and Johnson, N. A. (2009). Postexercise fat oxidation: effect of exercise duration, intensity, and modality. *Int J Sport Nutr Exerc Metab* **19**: 607–23. PMID: 20175430.

Whyte, L. J., Gill, J. M. R., and Cathcart, A. J. (2010). Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. *Metabolism* **59**: 1421–8. DOI: 10.1016/j.metabol.2010.01.002.

Wisløff, U., Støylen, A., Loennechen, J. P., Bruvold, M., Rognum, Ø., Haram, P. M., et al. (2007). Superior cardiovascular effect of aerobic interval training versus moderate continuous

training in heart failure patients: a randomized study. *Circulation* **115**: 3086–94. DOI: 10.1161/CIRCULATIONAHA.106.675041.

Zhang, T., Uchida, T., Gomez, G., Lluís, F., Thompson, J. C., and Greeley, G. H. (1993). Neural regulation of peptide YY secretion. *Regul Pept* **48**: 321–8. DOI: 10.1016/0167-0115(93)90160-A.

Table 1. Exercise responses during steady state exercise (SSE) and high intensity intermittent exercise (HIIE).

		SSE	HIIE	P
Exercise intensity	% of VO ₂ max	59.5 (1.6)	85.8 (4.0)	<0.0005*
Heart rate	beats.min ⁻¹	143 (8)	171 (10)	<0.0005*
RPE	6 - 20	13 (1)	17 (1)	<0.0005*
RER		0.93 (0.04)	1.00 (0.03)	<0.0005*
Power output	W	146 (16)	222 (24)	<0.0005*
External work	kJ	527 (58)	525 (65)	0.877
Net energy expenditure	kJ	2451 (208)	2429 (266)	0.649

Values are mean (SD). N = 12. *Different between SSE and HIIE (P < 0.05). RPE = rating of perceived exertion, RER = respiratory exchange ratio.

Table 2. Time-averaged area under the curve values for overall appetite perceptions in the control (CON), steady state exercise (SSE) and high intensity intermittent exercise (HIIE) trials.

	Morning (0 – 3.5 h)	Afternoon (3.5 – 8 h)	Total Trial (0 – 8 h)
Overall Appetite (0 – 100)			
CON	51 (15)	43 (10)	47 (11)
SSE	48 (13)	50 (10)	49 (10)
HIIE	41 (14)	45 (11)	43 (11)
<i>P</i>	0.060	0.228	0.256

Values are mean (SD). N = 12.

Table 3. Time-averaged area under the curve values for delta PYY₃₋₃₆ concentrations in the control (CON), steady state exercise (SSE) and high intensity intermittent exercise (HIIE) trials.

	Morning (0 – 3.75 h)	Afternoon (3.75 – 8 h)	Total Trial (0 – 8 h)
PYY₃₋₃₆ (pg.mL⁻¹)			
CON	4.7 (2.9) [†]	11.2 (9.6) [‡]	8.2 (5.9) [‡]
SSE	11.2 (8.4)	15.1 (11.4)	13.3 (9.3)
HIIE	12.7 (10.9)	20.1 (13.5)	16.6 (11.9)
<i>P</i>	0.027	0.050	0.026

Values are mean (SD). N = 11. [†]SSE different from CON, [‡]HIIE different from CON, P ≤ 0.05.

Figure 1. Overall appetite perceptions in CON (▼), SSE (●) and HIIE (○). Values are mean (SEM). N = 12. Hatched shaded rectangles indicate standardised test meals, lightly shaded rectangle indicates exercise, black rectangle indicates ad libitum meal. ‡HIIE different from CON, P < 0.05.

Figure 2. Delta PYY₃₋₃₆ concentrations in CON (▼), SSE (●) and HIIE (○). Values are mean (SEM). N = 11. Hatched shaded rectangles indicate standardised test meals, lightly shaded rectangle indicates exercise, black rectangle indicates ad libitum meal. †SSE different from CON, P < 0.05.

Figure 1

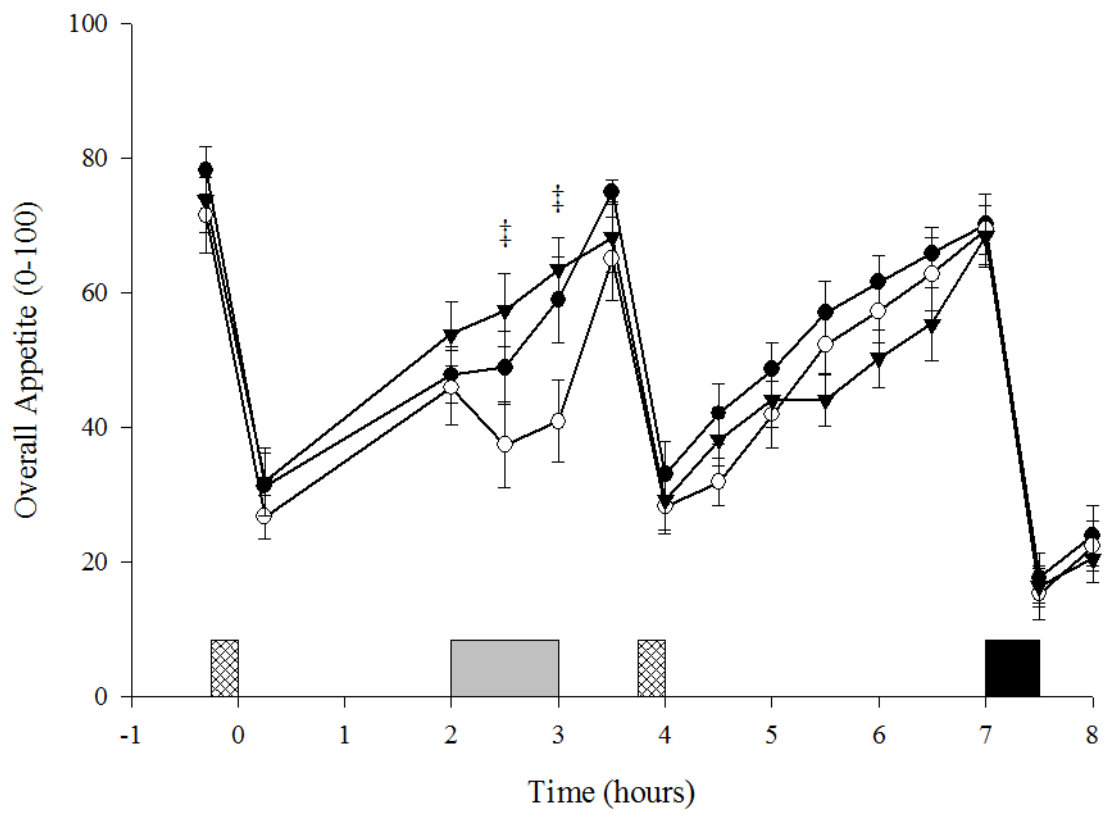


Figure 2

