



LEEDS
BECKETT
UNIVERSITY

Citation:

Deighton, K and Stensel, DJ (2014) Creating an acute energy deficit without stimulating compensatory increases in appetite: is there an optimal exercise protocol? *The Proceedings of the Nutrition Society*, 73 (2). 352 - 358. ISSN 0029-6651 DOI: <https://doi.org/10.1017/S002966511400007X>

Link to Leeds Beckett Repository record:

<https://eprints.leedsbeckett.ac.uk/id/eprint/138/>

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.

Creating an acute energy deficit without stimulating compensatory increases in appetite: is there an optimal exercise protocol?

Kevin Deighton^{1,2} and David J. Stensel²

¹School of Sport, Leeds Metropolitan University, Leeds, LS6 3QS, United Kingdom

²School of Sport, Exercise and Health Sciences, Loughborough University, Leicestershire, LE11 3TU, United Kingdom

Correspondence

Kevin Deighton

School of Sport,

Leeds Metropolitan University,

Leeds,

LS6 3QS,

United Kingdom.

Phone: +44 (0)113 81 24029

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

Key words: physical activity, energy balance, compensation

Abstract

Recent years have witnessed significant interest from both the scientific community and the media regarding the influence of exercise on subsequent appetite and energy intake responses. This review demonstrates a consensus among the majority of scientific investigations that an acute bout of land-based endurance exercise does not stimulate any compensatory increases in appetite and energy intake on the day of exercise. Alternatively, preliminary evidence suggests that low volume, supramaximal exercise may stimulate an increase in appetite perceptions during the subsequent hours. In accordance with the apparent insensitivity of energy intake to exercise in the short term; the daily energy balance response to exercise appears to be primarily determined by the energy cost of exercise. This finding supports the conclusions of recent training studies that the energy expenditure of exercise is the strongest predictor of fat loss during an exercise program.

Introduction

Overweight and obesity are defined by a body mass index (BMI) of 25 to 29.9 kg.m⁻² and 30 kg.m⁻² or greater, respectively, and characterised by an excess accumulation of body fat. Recent decades have witnessed a global increase in BMI to the extent that 22.5 % of adults worldwide were estimated to be overweight in 2008, with an additional 11.8 % qualifying as obese⁽¹⁾. These conditions are associated with an increased prevalence of several chronic diseases⁽²⁾, which has resulted in the classification of overweight and obesity as one of the top five global risk factors for mortality and one of the top ten risk factors for morbidity⁽³⁾. 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⁽⁴⁾.

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). Interest in the appetite and energy intake responses to exercise stems from the acknowledgement that physical activity may enhance weight loss via an increase in energy expenditure⁽⁴⁾ but is dependent upon subsequent food intake, as an increase in energy consumption may negate the energy deficit of exercise. Furthermore, any compensatory increases in appetite after exercise are likely to enhance the difficulty of maintaining a negative energy balance and increase psychological discomfort within participants.

The importance of this issue has recently been highlighted in two prominent media articles, which suggested that exercise stimulates compensatory increases in appetite and food intake that prevent weight loss⁽⁵⁾ and actually increase body fat⁽⁶⁾. However, the conclusions of these articles oppose findings from the scientific literature, which indicate that appetite and energy intake remain largely unchanged in the hours after an acute bout of exercise⁽⁷⁻⁹⁾. Although this relationship cannot continue indefinitely, the insensitivity of the appetite-regulating system to exercise-induced energy deficits is in stark contrast with the powerful homeostatic responses to food restriction. In this regard, current evidence suggests that food restriction elicits rapid compensatory increases in appetite and food intake, which does not occur in response to an equivalent exercise-induced energy deficit^(10,11).

This article aims to provide a comprehensive review of the scientific literature relating to the acute effects of exercise on appetite and energy intake responses. The final sections of this review subsequently address the question of whether there is an optimal exercise protocol for minimising compensatory increases in appetite after exercise.

Exercise-induced anorexia

Strenuous exercise (≥ 60 % of maximum oxygen uptake ($\text{VO}_2 \text{ max}$)) has consistently been found to acutely suppress appetite during and shortly after the exercise bout. This is known as ‘exercise-induced anorexia’ and has been demonstrated during a variety of exercise modes including: running^(12–14), cycling^(15–21), swimming⁽²²⁾ and resistance exercise⁽²³⁾. Although significant changes in appetite have been reported in these studies, values tend to return to control values within 30 min of the cessation of exercise. Such a transient effect is unlikely to influence energy intake in the hours after exercise but may delay the initiation of feeding when food is provided immediately after exercise⁽²⁴⁾. It therefore remains most important to understand the influence of exercise on resting appetite and energy intake responses in the hours *after* exercise.

Appetite responses in the immediate post-exercise period (0 – 120 min)

The majority of studies that have investigated the appetite response to exercise have employed an observation period lasting up to 2 h after the exercise bout. The consensus among these studies is that, after the recovery from exercise-induced anorexia, appetite during the post-exercise period does not differ from a resting control trial^(13,15,17–19,25–34). However, although appetite perceptions do not appear to decrease during the post-exercise period, it must be acknowledged that some investigations have reported elevated appetite perceptions after exercise compared with a control trial^(35–37).

The majority of the studies listed above have investigated the appetite response to exercise in healthy non-obese men and demonstrated no differences in appetite between exercise and control trials during the 2 h after a range of exercise modes including: walking⁽²⁶⁾, cycling^(15,17–19,35) and running^(13,28,30). The use of male participants in the majority of investigations has led some authors to postulate that sex-based differences may occur in the appetite response to exercise due to the critical relationship between energy balance and reproductive function in females⁽³⁸⁾. However, current research suggests that females do not exhibit increased appetite perceptions during the 2 h period after exercise^(18,19,29,31–33). Furthermore, a recent study by Hagobian et al.⁽²⁹⁾ directly compared the appetite response to exercise in male and female participants and concluded that 80 min of cycling at 70 % of $\text{VO}_2 \text{ max}$ did not stimulate increases in appetite in either sex during the 40 min after exercise.

As obesity is the result of a chronic excess of energy intake over energy expenditure, it is also logical to consider that appetite regulation in response to exercise may differ between lean and obese individuals. However, current research suggests that exercise also fails to stimulate

immediate compensatory increases in appetite in overweight and obese populations. In this regard, Unick and colleagues⁽³³⁾ reported that walking at 70 - 75 % of maximum heart rate to expend 3 kcal.kg⁻¹ body mass did not stimulate any compensatory increases in appetite during the 60 min after exercise in overweight and obese women. Similarly, Tsofliou et al.⁽³²⁾ did not observe any increases in appetite during the 60 min after 20 min of brisk walking in ten obese healthy women. Furthermore, in a direct comparison between obese and normal weight young men, Ueda and colleagues⁽³⁴⁾ demonstrated that 60 min of cycling at 50 % of VO₂ max did not stimulate any increases in appetite during the 60 min after exercise in either group.

Energy intake responses in the immediate post-exercise period (0 – 120 min)

The majority of studies detailed in the previous section also assessed the food intake response to exercise by providing participants with an ad libitum meal \leq 2 h after the exercise bout. In accordance with the appetite responses detailed in the previous section, the majority of these studies demonstrated that energy intake was unaffected by exercise^(27–30,32,33,39–41). However, some studies have demonstrated increases^(18,19,37,42) or decreases^(34,43,44) in energy intake after exercise.

An early study to investigate the effect of exercise on subsequent food intake responses was performed by King and colleagues in 1994⁽⁴⁰⁾. In this study, cycling at 30 % or 70 % of VO₂ max to expend ~ 1460 kJ did not affect energy intake at an ad libitum buffet meal 15 min after exercise. Furthermore, in a second group of healthy young men, energy intake was unaffected by either 26 or 52 min of cycling at 75 % of VO₂ max. Many of these findings were replicated by Erdmann and colleagues⁽²⁵⁾ in a combined sample of normal-weight men and women, as energy intake was unaffected by 30 min of cycling at either 50 or 100 W. However, although energy intake was unchanged in response to 60 min of cycling at a fixed work rate of 50 W, increasing the duration to 120 min stimulated an increase in energy intake. Although these findings suggest that the energy intake response to exercise is dependent upon exercise duration, all trials commenced after a 12 h overnight fast with the ad libitum meal provided 15 min after exercise. Therefore the observed increases in energy intake may have been a result of the extended overnight fast rather than exercise duration *per se*.

Although these studies may be criticised for the provision of a buffet meal so close to the cessation of exercise, other authors have also demonstrated no change in energy intake after exercise when the ad libitum meal was provided up to 1 h after exercise. This relationship has been demonstrated in young men and women^(28,30,39,41), overweight and obese women^(32,33) and in response to a variety of exercise modes. Furthermore, Hagobian et al.⁽²⁹⁾ reported no change in energy intake in response to exercise in men and women matched for age and VO₂ max.

The potential influence of exercise mode on the energy intake response to exercise has been demonstrated by Larson-Meyer et al.⁽³¹⁾. In this study, energy intake from an ad libitum meal that was provided two hours after exercise was not affected by 60 min of running at 70 % of VO₂ max but increased in response to 60 min of walking exercise at the same relative intensity in a separate group of participants. Although this study suggests that exercise mode may influence energy intake responses, it is plausible that the higher percentage body fat and lower VO₂ max in the walking group may have confounded the results.

In this regard, Finlayson and colleagues⁽⁴⁵⁾ demonstrated that body fat and physical activity levels may influence the energy intake response to exercise in females. This novel study analysed the energy intake response to 50 min of cycling exercise and separated the participants into two groups: compensators and non-compensators. Compensators were defined as the participants that increased energy intake beyond the energy cost of exercise, whereas non-compensators consumed less energy than that expended during exercise. Analysis of between group differences revealed a significantly higher BMI and percentage body fat and a lower habitual exercise frequency in the compensators. Alternatively, recent evidence suggests that this relationship may not occur in men as Jokisch and colleagues⁽³⁵⁾ demonstrated that 45 min of cycling at 65 – 75 % of maximum heart rate decreased energy intake in a sample of inactive normal weight young men but not active normal weight young men.

Other studies have also demonstrated a decrease in energy intake but are confounded by the provision of the ad libitum meal within 10 min of exercise completion^(43,44), which may have prevented the recovery from exercise-induced anorexia prior to feeding. However, in support of these findings, Ueda and colleagues⁽³⁴⁾ observed a decrease in energy intake at an ad libitum meal that was provided sixty minutes after a 60 min bout of cycling at 50 % of VO₂ max. Furthermore, this reduction in energy intake occurred in both lean and obese male participants. Other authors have also reported similar energy intake responses in normal weight and overweight participants⁽²⁷⁾ but this is not a universal finding⁽⁴⁶⁾.

Some authors have demonstrated an increase in energy intake during the immediate post-exercise period^(18,19,37,42). The reasons for such variations in the energy intake response to exercise are unclear but may be influenced by a variety of factors including: participant differences, the composition and timing of the ad libitum meal, variations in exercise mode, and time of day effects. Although measures of absolute energy intake provide important information regarding feeding behaviour, King and colleagues⁽⁴⁰⁾ suggested that it may be more relevant to express the energy intake response to exercise as 'relative energy intake' (REI) after deducting the net energy cost of

exercise. Subsequently, all increases in absolute energy intake reported thus far in this review are negated after accounting for the energy expenditure of exercise. Although this approach provides an overview of energy balance, the short monitoring period of the studies described thus far bias the results towards a lower REI during exercise trials as food intake is unlikely to be upregulated sufficiently at a single feeding episode to overturn substantial energy deficits. Therefore, investigations into the energy intake response to multiple ad libitum meals are important and are discussed below.

Appetite responses beyond a single test meal (2 – 9 h)

Although some authors have suggested that exercise may stimulate appetite in response to a standardised meal in the post-exercise period⁽⁴⁷⁾, the majority of studies demonstrate that appetite does not increase above control values during the 2 – 9 hours after exercise. This includes a variety of exercise modes including: running^(14,23,48–50), cycling⁽⁵⁰⁾, walking^(51–54) and resistance exercise⁽²³⁾ in both male and female populations.

However, exceptions have been observed within the literature as King et al.⁽²²⁾ demonstrated that 60 min of intermittent swimming exercise stimulated an increase in appetite compared with a resting control trial from 1.5 – 6 h after exercise. This contrasts with previous findings from the same author as 60 min of brisk walking exercise⁽⁵³⁾ and 90 min of running exercise⁽¹⁴⁾ did not influence resting appetite perceptions during the 7 h and 8.5 h post-exercise period, respectively. These differences occurred despite utilising a similar experimental protocol and participant population of physically active young men, which suggests a potential influence of exercise mode on subsequent appetite responses. However, subtle changes in meal timing and the recruitment of different participants confound any inferences regarding exercise mode.

The potential confounding influence of these factors is demonstrated by the findings of two studies from Broom and colleagues^(12,23), as 60 min of running at 70 % of VO₂ max in young physically active males stimulated an increase in hunger from 2 – 8 h after exercise in the former but not the latter of the two studies. Such discrepancies highlight the need for within measures study designs when comparing the appetite response to different exercise protocols.

Energy intake responses beyond a single test meal (2 – 9 h)

Few studies have monitored food intake in a laboratory setting for more than two hours after an acute exercise bout. However, the available research suggests that exercise does not stimulate any changes in energy intake during the subsequent 22.5 h⁽¹⁴⁾. This finding is also supported by studies

that have used self-report measures of food intake, which have failed to discover any changes in energy intake during the 24 h⁽⁵⁵⁾, 48 h⁽⁴⁸⁾ and 72 h⁽⁵⁴⁾ after exercise.

Studies that have successfully performed prolonged monitoring of food intake in a laboratory setting have typically provided participants with one or two buffet meals during the 3 to 7.5 h after exercise. Studies from our laboratory have reported that energy intake is unchanged by exercise under these laboratory conditions at any of the provided feeding opportunities^(14,22,49,53). Furthermore, the provision of an overnight food bag upon leaving the laboratory demonstrated that energy intake continued to remain unchanged for 22.5 h after exercise⁽¹⁴⁾.

The importance of such prolonged monitoring of energy intake is demonstrated by Pomerleau et al.⁽⁵⁴⁾. In this study, energy intake at an ad libitum buffet meal was significantly higher 1 h after walking at 70 % of VO₂ max compared with a resting control. However, after the provision of an additional ad libitum meal 6.5 h after exercise and an overnight snack bag, energy intake did not differ significantly between the trials.

One weakness of the above studies is that meals were provided to participants at pre-defined time points during the trials, which constrains the opportunities for food intake and may hinder the detection of differences in energy intake. This issue was recently addressed by King et al.⁽²⁴⁾ who allowed participants unlimited access to a buffet meal during the 6 h after 60 min of running at 70 % of VO₂ max. In support of previous findings, energy intake remained unchanged after exercise compared with a resting control trial and resulted in a substantially lower REI after exercise.

Manipulating exercise protocols to produce the most beneficial responses

This review has demonstrated a consensus among the majority of the scientific literature that an acute bout of land-based exercise does not stimulate compensatory increases in appetite and energy intake during the following hours. Subsequently, recent efforts have focussed on manipulating exercise protocols to most effectively reduce appetite and energy balance in the hours after exercise.

In this regard, Cheng and colleagues⁽¹⁶⁾ recently reported that moderate intensity cycling exercise induced a more prolonged suppression of hunger when performed two hours after a high fat breakfast compared with after a 12 h overnight fast. However, a subsequent investigation from our research group at Loughborough University suggested that this effect is limited to the period of exercise-induced anorexia, as appetite and energy intake did not differ from a resting control trial during the 5 – 9 h period after fasted or postprandial exercise despite greater appetite suppression *during* the postprandial exercise bout⁽⁵⁶⁾.

More commonly, investigators have manipulated the intensity of exercise when comparing exercise protocols but initial studies yielded equivocal findings. In this regard, Thompson and colleagues⁽²⁰⁾ demonstrated greater appetite suppression during cycling exercise at 68 % of VO₂ max compared with 35 % of VO₂ max but found no differences in energy intake after exercise. Conversely, Imbeault et al.⁽⁵⁷⁾ found no differences in appetite but reported a decrease in energy intake after running at 75 % of VO₂ max compared with energy-matched walking at 35 % of VO₂ max. Alternatively, Ueda and colleagues⁽²¹⁾ and King et al.⁽⁴⁰⁾ found no differences in the appetite or energy intake responses to cycling at 50 % versus 75 % of VO₂ max and 30 % versus 70 % of VO₂ max, respectively.

Interest in the influence of exercise intensity on appetite and energy intake responses has been renewed in recent years due to the popularity of high intensity intermittent exercise (HIIE). In this regard, several authors within both the scientific literature and the media have suggested that HIIE may elicit greater weight loss than traditional endurance exercise due to greater reductions in appetite during the post-exercise period^(58–63). However, despite these postulations, research from our laboratory has demonstrated that a recently popularised sprint interval exercise protocol, consisting of six 30 s maximal sprints on a cycle ergometer, stimulated compensatory increases in appetite during the five hour monitoring period after exercise that did not occur in response to 60 min of continuous cycling at 60 % of VO₂ max. Although these differences in appetite did not influence ad libitum food intake on the day of exercise, similar absolute food intake between trials resulted in a substantial negative energy balance after endurance exercise compared with sprint exercise and a resting control trial due to a significantly higher energy expenditure during the endurance exercise than sprint exercise (mean (SD) 2640 (418) vs. 594 (50) kJ; $P < 0.0005$)⁽⁶⁴⁾.

In order to further investigate this issue, a high volume, submaximal HIIE protocol was investigated in a subsequent study and it was determined that compensatory increases in appetite and energy intake did not occur during the five hour monitoring period after ten 4 min cycling intervals at 85 – 90 % of VO₂ max or 60 min of continuous cycling at 60 % of VO₂ max⁽⁶⁵⁾. The absence of any compensatory increases in appetite in this study suggests that previously observed increases after supramaximal interval exercise 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. It is also important to note that these studies employed exercise protocols that represented both extremes of the HIIE spectrum (i.e. very low volume supramaximal interval exercise and high volume, submaximal interval exercise) and therefore provides convincing evidence that HIIE does

not elicit lower appetite perceptions during the hours after exercise than traditional endurance exercise.

Conclusions

Contrary to recent media articles, this review demonstrates a consensus among the majority of scientific investigations that an acute bout of land-based endurance exercise does not stimulate compensatory increases in appetite and energy intake during the hours after exercise. Current research also disputes the postulations of recent authors that HIIE elicits lower appetite and energy intake responses than traditional endurance exercise. Alternatively, initial investigations suggest that supramaximal exercise may stimulate increases in appetite that do not occur in response to larger energy deficits induced by moderate intensity exercise. Furthermore, in accordance with the consensus that energy intake remains unresponsive to exercise in the short term; the daily energy balance response to exercise appears to be primarily determined by the energy cost of exercise. These findings support the concept that appetite and energy intake are primarily determined via orogastric, rather than metabolic mechanisms⁽⁵¹⁾, and also support the conclusions of recent training studies that the energy expenditure of exercise is the strongest predictor of fat loss during an exercise program^(66,67).

Financial support

This review paper did not receive any financial support.

Conflict of interest

Both authors declare that there is no conflict of interest.

Authorship

KD conceived and wrote the manuscript. DJS read and approved the manuscript.

References

1. Finucane MM, Stevens GA, Cowan MJ et al. (2011) National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9·1 million participants. *Lancet* **377**, 557–67.
2. Bray GA (2004) Medical consequences of obesity. *J Clin Endocrinol Metab* **89**, 2583–9.
3. World Health Organisation (2009) Global health risks. Available at: http://www.who.int/healthinfo/global_burden_disease/GlobalHealthRisks_report_full.pdf.
4. Donnelly JE, Blair SN, Jakicic JM et al. (2009) American College of Sports Medicine Position Stand. Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Med Sci Sports Exerc* **41**, 459–71.
5. Cloud J (2009) Why exercise won't make you thin. *Time Magazine*. Available at: <http://www.time.com/time/health/article/0,8599,1914857,00.html>.
6. Gray R (2009) Health warning: exercise makes you fat. *The Daily Telegraph*. Available at: <http://www.telegraph.co.uk/science/6083234/Health-warning-exercise-makes-you-fat.html>.
7. Blundell JE & King NA (1999) Physical activity and regulation of food intake: current evidence. *Med Sci Sports Exerc* **31**, S573–83.
8. Blundell JE, Stubbs RJ, Hughes DA et al. (2003) Cross talk between physical activity and appetite control: does physical activity stimulate appetite? *Proc Nutr Soc* **62**, 651–61.
9. Martins C, Morgan L, Truby H (2008) A review of the effects of exercise on appetite regulation: an obesity perspective. *Int J Obes* **32**, 1337–47.
10. Hubert P, King NA, Blundell JE (1998) Uncoupling the effects of energy expenditure and energy intake: appetite response to short-term energy deficit induced by meal omission and physical activity. *Appetite* **31**, 9–19.
11. King JA, Wasse LK, Ewens J 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.

12. Broom DR, Stensel DJ, Bishop NC et al. (2007) Exercise-induced suppression of acylated ghrelin in humans. *J Appl Physiol* **102**, 2165–71.
13. Burns SF, Broom DR, Miyashita M et al. (2007) A single session of treadmill running has no effect on plasma total ghrelin concentrations. *J Sports Sci* **25**, 635–42.
14. King JA, Miyashita M, Wasse LK et al. (2010) Influence of prolonged treadmill running on appetite, energy intake and circulating concentrations of acylated ghrelin. *Appetite* **54**, 492–8.
15. Becker GF, Macedo RCO, Cunha GDS et al. (2012) Combined effects of aerobic exercise and high-carbohydrate meal on plasma acylated ghrelin and levels of hunger. *Appl Physiol Nutr Metab* **37**, 184–92.
16. Cheng MH-Y, Bushnell D, Cannon DT et al. (2009) Appetite regulation via exercise prior or subsequent to high-fat meal consumption. *Appetite* **52**, 193–8.
17. Evero N, Hackett LC, Clark RD et al. (2012) Aerobic exercise reduces neuronal responses in food reward brain regions. *J Appl Physiol* **112**, 1612–9.
18. Laan DJ, Leidy HJ, Lim E et al. (2010) Effects and reproducibility of aerobic and resistance exercise on appetite and energy intake in young, physically active adults. *Appl Physiol Nutr Metab* **35**, 842–7.
19. Martins C, Morgan LM, Bloom SR et al. (2007) Effects of exercise on gut peptides, energy intake and appetite. *J Endocrinol* **193**, 251–8.
20. Thompson DA, Wolfe LA, Eikelboom R (1988) Acute effects of exercise intensity on appetite in young men. *Med Sci Sports Exerc* **20**, 222–7.
21. Ueda S, Yoshikawa T, Katsura Y et al. (2009) 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.
22. King JA, Wasse LK, Stensel DJ (2011) The acute effects of swimming on appetite, food intake, and plasma acylated ghrelin. *J Obes* 351628.

23. Broom DR, Batterham RL, King JA et al. (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.
24. King JA, Wasse LK, Stensel DJ (2013) Acute exercise increases feeding latency in healthy normal weight young males but does not alter energy intake. *Appetite* **61**, 45–51.
25. Erdmann J, Tahbaz R, Lippl F et al. (2007) Plasma ghrelin levels during exercise - effects of intensity and duration. *Regul Pept* **143**, 127–35.
26. Farah NMF, Brunstrom JM, Gill JMR (2012) Using a novel computer-based approach to assess the acute effects of exercise on appetite-related measures. *Appetite* **58**, 196–204.
27. George VA & Morganstein A (2003) Effect of moderate intensity exercise on acute energy intake in normal and overweight females. *Appetite* **40**, 43–6.
28. Gonzalez JT, Veasey RC, Rumbold PLS et al. (2013) Breakfast and exercise contingently affect postprandial metabolism and energy balance in physically active males. *Br J Nutr* **110**, 721-32.
29. Hagobian TA, Yamashiro M, Hinkel-Lipsker J et al. (2013) Effects of acute exercise on appetite hormones and ad libitum energy intake in men and women. *Appl Physiol Nutr Metab* **38**, 66–72.
30. Kelly PJ, Guelfi KJ, Wallman KE et al. (2012) Mild dehydration does not reduce postexercise appetite or energy intake. *Med Sci Sports Exerc* **44**, 516–24.
31. Larson-Meyer DE, Palm S, Bansal A et al. (2012) Influence of running and walking on hormonal regulators of appetite in women. *J Obes* 730409.
32. Tsofliou F, Pitsiladis YP, Malkova D et al. (2003) Moderate physical activity permits acute coupling between serum leptin and appetite-satiety measures in obese women. *Int J Obes Relat Metab Disord* **27**, 1332–9.
33. Unick JL, Otto AD, Goodpaster BH et al. (2010) Acute effect of walking on energy intake in overweight/obese women. *Appetite* **55**, 413–9.

34. Ueda S, Yoshikawa T, Katsura Y et al. (2009) Changes in gut hormone levels and negative energy balance during aerobic exercise in obese young males. *J Endocrinol* **201**, 151–9.
35. Jokisch E, Coletta A, Raynor HA (2012) Acute energy compensation and macronutrient intake following exercise in active and inactive males who are normal weight. *Appetite* **58**, 722–9.
36. Kawano H, Mineta M, Asaka M et al. (2013) Effects of different modes of exercise on appetite and appetite-regulating hormones. *Appetite* **66**, 26–33.
37. Verger P, Lanteaume MT, Louis-Sylvestre J (1994) Free food choice after acute exercise in men. *Appetite* **22**, 159–64.
38. Wade GN & Jones JE (2004) Neuroendocrinology of nutritional infertility. *Am J Physiol Regul Integr Comp Physiol* **287**, R1277–96.
39. Balaguera-Cortes L, Wallman KE, Fairchild TJ et al. (2011) Energy intake and appetite-related hormones following acute aerobic and resistance exercise. *Appl Physiol Nutr Metab* **36**, 958–66.
40. King NA, Burley VJ, Blundell JE (1994) Exercise-induced suppression of appetite: effects on food intake and implications for energy balance. *Eur J Clin Nutr* **48**, 715–24.
41. Maraki M, Tsofliou F, Pitsiladis YP et al. (2005) Acute effects of a single exercise class on appetite, energy intake and mood. Is there a time of day effect? *Appetite* **45**, 272–8.
42. Shorten AL, Wallman KE, Guelfi KJ (2009) Acute effect of environmental temperature during exercise on subsequent energy intake in active men. *Am J Clin Nutr* **90**, 1215–21.
43. Almada C, Cataldo LR, Smalley SV et al. (2013) Plasma levels of interleukin-6 and interleukin-18 after an acute physical exercise: relation with post-exercise energy intake in twins. *J Physiol Biochem* **69**, 85–95.
44. Westerterp-Plantenga MS, Verwegen CR, Ijedema MJ et al. (1997) Acute effects of exercise or sauna on appetite in obese and nonobese men. *Physiol Behav* **62**, 1345–54.
45. Finlayson G, Bryant E, Blundell JE et al. (2009) Acute compensatory eating following exercise is associated with implicit hedonic wanting for food. *Physiol Behav* **97**, 62–7.

46. Kissileff HR, Pi-Sunyer FX, Segal K et al. (1990) Acute effects of exercise on food intake in obese and nonobese women. *Am J Clin Nutr* **52**, 240–5.
47. Malkova D, McLaughlin R, Manthou E et al. (2008) Effect of moderate-intensity exercise session on preprandial and postprandial responses of circulating ghrelin and appetite. *Horm Metab Res* **40**, 410–5.
48. King NA, Lluch A, Stubbs RJ et al. (1997) High dose exercise does not increase hunger or energy intake in free living males. *Eur J Clin Nutr* **51**, 478–83.
49. Wasse LK, Sunderland C, King JA et al. (2012) Influence of rest and exercise at a simulated altitude of 4,000 m on appetite, energy intake, and plasma concentrations of acylated ghrelin and peptide YY. *J Appl Physiol* **112**, 552–9.
50. Wasse LK, Sunderland C, King JA et al. (2013) The influence of vigorous running and cycling exercise on hunger perceptions and plasma acylated ghrelin concentrations in lean young men. *Appl Physiol Nutr Metab* **38**, 1–6.
51. Borer KT, Wuorinen E, Chao C et al. (2005) Exercise energy expenditure is not consciously detected due to oro-gastric, not metabolic, basis of hunger sensation. *Appetite* **45**, 177–81.
52. Borer KT, Wuorinen E, Ku K et al. (2009) Appetite responds to changes in meal content, whereas ghrelin, leptin, and insulin track changes in energy availability. *J Clin Endocrinol Metab* **94**, 2290–8.
53. King JA, Wasse LK, Broom DR et al. (2010) Influence of brisk walking on appetite, energy intake, and plasma acylated ghrelin. *Med Sci Sports Exerc* **42**, 485–92.
54. Pomerleau M, Imbeault P, Parker T et al. (2004) Effects of exercise intensity on food intake and appetite in women. *Am J Clin Nutr* **80**, 1230–6.
55. Hanlon B, Larson MJ, Bailey BW et al. (2012) Neural response to pictures of food after exercise in normal-weight and obese women. *Med Sci Sports Exerc* **44**, 1864–70.
56. Deighton K, Zahra JC, Stensel DJ (2012) Appetite, energy intake and resting metabolic responses to 60 min treadmill running performed in a fasted versus a postprandial state. *Appetite* **58**, 946–54.

57. Imbeault P, Saint-Pierre S, Alméras N et al. (1997) Acute effects of exercise on energy intake and feeding behaviour. *Br J Nutr* **77**, 511–21.
58. Boutcher SH (2011) High-intensity intermittent exercise and fat loss. *J Obes* 868305.
59. Heydari M, Freund J, Boutcher SH (2012) The effect of high-intensity intermittent exercise on body composition of overweight young males. *J Obes* 480467.
60. Trapp EG, Chisholm DJ, Freund J et al. (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.
61. Tremblay A, Simoneau JA, Bouchard C (1994) Impact of exercise intensity on body fatness and skeletal muscle metabolism. *Metabolism* **43**, 814–8.
62. Naish J (2013) Is three minutes a week of exercise all you need to get fit? Scientists say ideal fitness regime involves intense bursts of activity. *The Daily Mail*. Available at: <http://www.dailymail.co.uk/health/article-2255867/Is-minutes-week-exercise-need-fit-Scientists-say-ideal-fitness-regime-involves-intense-bursts-activity.html>.
63. Bendoris M (2012) The two-minute wonder workout. *The Scottish Sun*. Available at: <http://www.thescottishsun.co.uk/scotsol/homepage/scotlandfeatures/4159373/The-two-minute-wonder-workout.html>.
64. Deighton K, Barry R, Connon CE et al. (2013) Appetite, gut hormone and energy intake responses to low volume sprint interval and traditional endurance exercise. *Eur J Appl Physiol* **113**, 1147–56.
65. Deighton K, Karra E, Batterham RL et al. (2013) Appetite, energy intake, and PYY3-36 responses to energy-matched continuous exercise and submaximal high-intensity exercise. *Appl Physiol Nutr Metab* **38**, 947–52.
66. Barwell ND, Malkova D, Leggate M et al. (2009) Individual responsiveness to exercise-induced fat loss is associated with change in resting substrate utilization. *Metabolism* **58**, 1320–8.
67. Jakicic JM, Marcus BH, Lang W et al. (2008) Effect of exercise on 24-month weight loss maintenance in overweight women. *Arch Intern Med* **168**, 1550–9.