THE IMPACT OF HIGH INTENSITY INTERMITTENT EXERCISE ON RESTING METABOLIC RATE IN HEALTHY MALES

Benjamin Kelly. James A. King. Jonas Goerlach. Myra A. Nimmo.

School of Sport, Exercise and Health Sciences, Loughborough University, Loughborough, Leicestershire, LE11 3TU, UK

Correspondence:

Professor Myra A. Nimmo,

School of Sport, Exercise and Health Sciences,

Loughborough University,

Loughborough,

LE11 3TU.

Phone: +44 (0)1509 226311

Fax: +44 (0)1509 226301

e-mail: M.A.Nimmo@lboro.ac.uk

ABSTRACT

High intensity intermittent exercise training (HIT) may favourably alter body composition despite low training volumes and predicted energy expenditure (EE). **Purpose:** to characterise the acute impact of two common HIT protocols on EE and post-exercise oxygen consumption (11 h EPOC). **Methods:** oxygen consumption ($l \cdot min^{-1}$), respiratory exchange ratio (RER) and EE were measured in nine healthy, lean males over 12 h under three conditions: control (CON), HIT1 (10x one min high intensity cycling bouts followed by one min rest) and HIT2 (10x four min high intensity cycling bouts followed by two min rest). **Results:** total exercise period EE during HIT1 (1151 ± 205 kJ) (mean ± SD) was significantly lower than HIT2 (2788 ± 322 kJ; $p < 0.001$). EE within the 60 min after exercise was significantly albeit marginally higher after HIT1 (388 ± 44 kJ; $p = 0.02$) and HIT2 (389 ± 39 kJ; $p = 0.01$) compared with CON (329 ± 39 kJ), with no difference between exercise conditions ($p = 0.778$). RER during this period was significantly lower in HIT1 (0.78 ± 0.06; $p = 0.011$) and HIT2 (0.76 ± 0.04; $p = 0.004$) compared with CON (0.87 ± 0.06). During the 'slow phase' of EPOC (1.25 h – 9.75 h) there were no significant differences in EE ($p = 0.07$) or RER ($p = 0.173$) between trials. **Conclusions:** single HIT sessions notably increases EE during exertion however the influence on metabolic rate post-exercise is transient and relatively minor.

Keywords: Interval Training, EPOC, Energy Expenditure, Energy Balance

INTRODUCTION

Despite the common acceptance that regular physical activity reduces the risk of many chronic conditions such as obesity, type 2 diabetes and cardiovascular disease, participation in physical activity remains low (WHO 2010). Specifically, within the UK approximately two-thirds of men and women do not perform sufficient activity to benefit their health (WHO 2010). Traditionally, health oriented physical activity guidelines have centred on individuals undertaking moderate intensity aerobic, continuous forms of exercise on most days of the week (Department of Health 2011). Given that the most commonly cited reason for not participating in physical activity is lack of time, and the recent evidence that individuals preferred an intermittent exercise protocol in comparison to continuous exercise (Bartlett et al. 2012), it is now timely to consider other forms of exercise which can potentially be more readily adopted.

In recent years there has been a surge in interest concerning high intensity intermittent exercise training (HIT) due to its ability to confer notable cardio-metabolic health benefits rapidly, with great time efficiency (Gibala & Little 2010). Specifically, HIT has been shown to improve insulin sensitivity and glycaemic control (Burgomaster et al 2006, 2008; Gibala et al. 2006; Wisloff et al. 2007; Tjonna et al. 2008; Babraj et al. 2009; Haram et al. 2009; Nybo et al. 2010; Richards et al. 2010; Whyte et al. 2010; Gaesser and Angadi. 2011; Hood et al. 2011; Little et al. 2011; Shepherd et al. 2013) as well as other cardiovascular health factors (Fletcher et al. 1996) after just two weeks of training (typically six training sessions).

An intriguing body of research has also emerged documenting beneficial changes in body composition and/or mass in response to HIT (Boutcher 2010) with studies reporting favourable changes within short time-frames. Accordingly, Whyte et al. (2010) subjected overweight and obese males to six training sessions over two weeks with each session consisting of four to six 30s Wingate sprints, interspersed with 4.5 min of recovery. Along with positive changes in several metabolic health parameters, this group noted a significant 2.4 cm reduction in waist circumference. Our research group also observed a significant reduction in waist circumference (1.4 cm) in a group of overweight and obese males following the completion of two weeks of HIT (Leggate et al. 2012). In this study each training session consisted of 10 x four min bouts of cycling at ~85% $\dot{V}O_{2peak}$, separated by two min of recovery. Additionally, research has hinted that HIT may be superior to continuous moderate-intensity exercise training for the purpose of adiposity reduction (Macpherson et al. 2011). In this study six weeks of treadmill based sprint interval training induced a two-fold greater reduction in total adiposity (12.4 vs. 5.8%) compared with a much greater volume of continuous moderate-intensity running. Given the relatively short

duration of these interventions and accumulated exercise volume, the energy expended during exercise was unlikely to have been sufficient to account for the reported changes in waist circumference/adiposity. However, definitive information regarding this aspect of HIT is lacking and consequently the total impact of HIT on energy balance is unknown.

Recently, Knab et al. (2011) reported that metabolic rate is augmented for 14 h after a single bout of moderate-high intensity aerobic exercise, equating to an additional 795 kJ energy expenditure (EE) on top of that expended during exertion. The authors of this previous paper suggest that this excess post-exercise oxygen consumption (EPOC) may significantly contribute to fat loss over the course of exercise training programmes. Although research has been conducted to profile EPOC after continuous forms of exercise (Borsheim and Bahr 2003; LaForgia et al. 2006), less attention has been given to high intensity forms of exercise (McGarvey et al. 2005; Malatesta et al. 2008). Specifically, how high intensity intermittent exercise (HIIE – representing one single bout of HIT) impacts upon EE and substrate utilisation, both during and after exercise, is not well understood. Given recent reports demonstrating notable changes in body composition and/or mass with HIT (Boutcher 2010) it is necessary to understand how HIIE influences these parameters. Recent reports have provided preliminary information on this issue (Williams 2013; Chan & Burns 2013; Hazell et al. 2012) however in these studies the focus has been on sprint interval training i.e. the classical form of HIT which is characterised by bouts of all-out effort over 30s. This type of training is not feasible for many who are inactive or who have chronic conditions e.g. type 2 diabetes or cardiovascular disease. More acceptable forms of HIT have emerged which are more suitable for such individuals, however there is currently no information regarding the impact of these protocols on EE and substrate utilisation, during or after exercise.

The present investigation sought to accurately characterise the EE during exercise and EPOC (11 h post-exercise) of two commonly used HIT protocols which have been developed to be implemented with untrained individuals and/or clinical groups. The first protocol (HIT1) examined a HIT intervention which has been demonstrated as being suitable for individuals with type 2 diabetes, conferring rapid and marked glucoregulatory benefits (Little et al. 2011). The second HIT protocol (HIT2) examined was that previously implemented by our research group (Leggate et al. 2012) and others (Talanian et al. 2007), where we observed a significant decrease in the waist circumference of overweight/obese men after just six training sessions. In the present investigation we aimed to characterise the EE elicited by HIIE in terms of both the immediate and

delayed effect. In doing so, we sought to determine the potential contribution of HIT induced EE to previously documented reductions in body fat and/or waist circumference.

METHODS

Participants

Nine untrained and moderately active men (defined as doing no more than two moderate intensity exercise sessions per week with no involvement in any structured training for ≥ two years) volunteered to take part in the current study. The study inclusion criteria demanded that participants had a BMI 18.5-29.9 $kg \cdot m^{-2}$, and had been weight stable for at least three months prior to the study. Participants were free of cardiovascular, metabolic and haematological disease and were not taking any medications or supplements. The participants were fully informed of the aims, risks and discomforts associated with the investigation before providing written informed consent. This study conformed to local guidelines, the declaration of Helsinki, and was approved by the local ethics committee.

Preliminary Testing

Before main trials resting metabolic rate (RMR) was measured on two separate occasions in order to obtain reliable baseline results (Compher et al. 2006; Roffey et al. 2006). For these tests participants reported to the laboratory in the morning (~07:30) having not eaten since the previous evening. Resting metabolic rate assessments followed the guidance of Compher et al. (2006). In brief, participants were asked: not to exercise during the preceding 24 h; not to consume caffeine, alcohol or drugs during the preceding 24 h; to remain fasted for at least 10 h prior to laboratory entry; to record what they had eaten in the preceding 24 h in order to replicate this feeding pattern prior to the second RMR measurement; to eliminate uncontrolled activity by travelling to the laboratory by motorised transport, with any essential walking being kept to < 50 metres. The laboratory temperature was maintained at 23.5-24.5°C. To minimise movement participants were assisted when necessary by wheelchair.

All resting metabolic measurements were performed using an open circuit, ventilated hood indirect calorimetry system (GEM Nutrition Ltd., Cheshire, England). This technique has been described as the gold standard in indirect calorimetry and has been validated in the measurement of RMR in both adults and children (Weissman et al. 1990; Tissot et al. 1995). After a 30 min seated resting period, participants were instructed to lie in a

comfortable supine position. The clear hood canopy was placed over the head area and plastic sheeting attached to the hood was placed around the body to form a seal between the air inside and outside the hood. Participants were instructed not to talk or fidget and listened to the radio throughout the measurement to reduce boredom and to prevent sleeping. $\dot{V}O_2$ ($l \cdot min^{-1}$), $\dot{V}CO_2$ ($l \cdot min^{-1}$), RER and EE (kJ) were determined at 30s intervals (as pre-determined by software) over a 30 min period. For analysis, the first 10 min of data were discarded to account for any initial short-term respiratory artefact. Minute averages for $\dot{V}O_2$ along with RER caloric equivalents were used to estimate EE. The mean percentage difference between the two baseline measurements was 1.6%.

Before main trials participants also performed a $\dot{V}O_{2peak}$ test to volitional exhaustion using an electromagnetically braked cycle ergometer (Lode Excalibur, Groningen, The Netherlands). $\dot{V}O_2$ was measured continuously throughout the test using an online breath-by-breath gas analysis system (Cortex Metalyzer, CPX International Inc, Berlin, Germany). For this test participants first warmed up for three min against a resistance of 50 watts (W). After this, the workload was increased to a starting work rate which was subsequently increased by 30 W at intervals of three min. The initial work load for the test was individualised for each participant, being selected so that the test would last for approximately 12 min (four stages). The test was terminated when participants could no longer maintain pedal cadence at 50 revolutions per minute (RPM). $\dot{V}O_{2peak}$ was identified over the highest 30s period during the test. Heart rate (HR) was measured during the test using a telemetric heart rate monitor (Polar RS100, Polar Electro, UK Ltd., Warwick, UK).

Main Experimental Trials

All participants completed two HIIE main trials and one control trial with each study visit being separated by at least four days. Figure 1 provides a schematic overview of the main trial protocols. Prior to each main trial participants abstained from structured physical activity and standardised their dietary intake within the prior 24 h. On the morning of trials participants arrived at the laboratory via motorised transport to eliminate extraneous activity. An 11 h EPOC assessment was made across each main trial and for clarity and comparability across trials we have defined the beginning of this measurement point as time zero (0 h). Thus, exercise protocols ended at the same time (prior to the start of EPOC assessment) which meant that it was necessary for the HIIE start times to differ. Consequently, the beginning of the control trial and HIT2 is defined at -67 min which reflects the 60 min exercise period (in HIT2) before EPOC measurement, two min cool down, and an additional five min resting period necessary to allow CO_2 levels to return to values which fall within acceptable limits for transfer to the ventilated hood (essential for the exercise trials). Accordingly, the beginning of HIT1 is defined

as -27 min which reflects the duration of the 20 min exercise protocol as well as the aforementioned seven min recovery period after exercise.

Insert Figure 1 near here

For the control trial participants reported to the laboratory in a fasted state at ~07:30. Participants then relaxed in a comfortable seated position for 30 min prior to the start of the trial (-67 min). At this point breath collection via online gas analysis (Cortex Metalyzer, CPX International Inc, Berlin, Germany) began whilst participants remained in a seated position. Gas collection occurred continuously for 67 min. This period prior to post-exercise assessment is defined as the 'exercise period' (EP). Immediately after the EP, the rapid phase (RP) of EPOC was measured continuously over 60 min using the ventilated hood (0 h – 1 h). During this time, measurements were taken continuously with participants resting in a supine position. Immediately after the RP, participants were seated next to the ventilated hood where breakfast was provided (1 h – 1.25 h). Participants consumed breakfast within 15 min, after which point ventilated hood measurement recommenced. Subsequently, expired gas analysis was performed continuously for 15 of every 30 min over the next 9.75 h. This period was identified as the EPOC slow phase (SP). Standardised meals were also consumed by participants at 4 h and 8 h with each meal being consumed within 15 min.

Identical procedures were completed in the exercise trials except a bout of HIIE was completed at the beginning of trials. During exercise, gas analysis data was collected continuously with $\dot{V}O_2$, $\dot{V}CO_2$, RER and HR averaged every 15s. Within each protocol the two min cool down period was composed of cycling against no resistance. The exercise protocol within HIT1 was identical to that described by Little et al. (2011). This protocol consisted of 10 x one min high intensity intervals each separated by one min of light pedalling against a resistance of 50 W. Whilst maintaining an RPM of 80-100, workload was manipulated throughout to ensure HR was maintained at ~90% HR_{peak} during each high intensity effort. The exercise protocol within HIT2 was identical to that described previously by Leggate et al. (2012), consisting of 10 x four min intervals at an RPM of 60-80, each separated by two min of light pedalling against a set resistance of 50 W. The workload was manipulated during the trial to ensure that the average corresponding $\dot{V}O_2$ during high intensity bouts equated to ~85% $\dot{V}O_{2peak}$.

Food Intake

During main trials participants were provided with three standardised meals which were identical across trials. All meals were consumed within 15 min. The total amount of energy provided for participants at each meal was

individualised to meet daily energy requirements for an inactive day. This value was calculated by multiplying each individual's average resting metabolic rate (from preliminary assessments) by 1.375 (Harris et al. 1918). Breakfast was provided immediately after the EP (at 1 h) and consisted of toasted white bread and jam (30% of daily energy requirements). Lunch and dinner were provided at 4 and 8 h with both meals consisting of a tuna mayonnaise sandwich, salted crisps, chocolate muffin and green apple (each meal 35% of daily energy requirements). The macronutrient composition of the food provided across each trial was typical of a UK diet and consisted of 38% fat, 14% protein and 48% carbohydrate (Department for Education, Food and Rural Affairs 2010). Mean energy intake equated 12, 385 kJ. Throughout main trials, after the completion of exercise, water was available for participants to consume *ad libitum*.

Calculation of Energy Expenditure

In the present study EE was estimated during and after exercise as per the Weir equation (Weir 1949). These calculations require oxygen consumption and RER data and are valid for RER values up to 1.0. Consequently, as RER transiently increases above 1.0 during HIIE, calculations for the EP period utilised an RER of 1.0 when values were higher than this level.

Statistical Analysis

The statistical analysis for this study was performed using the statistical package for social sciences for Windows (SPSS Ver.19). All data are reported as Mean ± SD. For both the EP and RP periods, $\dot{V}O_2$, RER and EE minute averages were determined for each participant, whilst for the SP period data were collected as 30 min averages for each trial hour. The latter values were subsequently multiplied by two, to provide an estimate of hourly $\dot{V}O_2$, RER and EE. Data were also analysed for the RP and SP periods combined to give a total EPOC. Each of these trial segments were analysed separately using repeated measures ANOVA. Where appropriate, data was analysed using area under the curve (AUC) which was calculated using the trapezoid method (Matthews et al. 1990). No differences were found between mean and AUC data and therefore AUC data is not reported. Where significant differences were found a Bonferroni post-hoc test was used to identify where significant differences lay. For all analyses alpha was set at 5%.

RESULTS

Participant and Exercise Characteristics

The physical characteristics of the study participants are summarised in Table 1. The average workload undertaken by study participants to elicit 90% of peak HR during exercise within HIT1 was 204 ± 47 W. Similarly, the average workload completed to elicit 85% of $\dot{V}O_{2peak}$ during HIT2 was 211 ± 46 W.

Insert Table 1 near here

Energy Expenditure

When analysing EE for the entire trial period (combining EP, RP and SP phases) one-way ANOVA revealed significant differences between experimental conditions ($p < 0.001$). The total energy expended within HIT2 (7280 ± 703 kJ) was significantly higher than that expended in both HIT1 (5761 ± 598 kJ; $p < 0.001$) and CON (4874 ± 456 kJ; $p < 0.001$). Energy expenditure in HIT1 was also significantly higher than CON ($p < 0.001$). Figure 2a illustrates the EE during the EP independently. Within this period one-way ANOVA revealed a significant difference between trials ($p < 0.001$). Specifically, EE during the EP in HIT2 (2788 ± 322 kJ) was significantly greater than HIT1 (1151 ± 205 kJ; $p < 0.001$). Logically, the EE in both exercise conditions during the EP was higher than CON (368 ± 46 kJ; $p < 0.001$).

Insert Figure 2 near here

Figure 2b shows the EE during the EPOC RP across main trials. One-way ANOVA revealed a significant difference between trials for the EPOC RP ($p < 0.001$). Specifically, the EE for this period after HIT1 (388 ± 44 kJ) was significantly higher than that expended over the same period during CON (329 ± 39 kJ; $p = 0.02$). Similarly, EE during the RP in HIT2 (389 ± 39 kJ) was also higher than CON ($p = 0.01$). During this period there was no difference in EE between exercise conditions ($p = 0.778$). Figure 2c illustrates the EE during the EPOC SP across main trials. Within this period one-way ANOVA found no differences between experimental trials ($p = 0.07$). Energy expenditure during the SP was 4107 ± 387, 4227 ± 426, and 4268 ± 389 kJ for CON, HIT1 & HIT2, respectively. When the total EPOC period was analysed together (RP & SP combined; Figure 2d) one-way ANOVA revealed no significant differences between experimental trials with total EE of 4437 ± 419, 4615 ± 457 and 4658 ± 421 kJ for CON, HIT1 and HIT2 ($p = 0.209$).

Respiratory Exchange Ratio

One-way ANOVA showed that there was a significant difference in the RER during the RP after exercise ($p < 0.001$). In the RP RER was significantly lower in HIT1 (0.78 ± 0.06; $p = 0.011$) and HIT2 (0.76 ± 0.04; $p = 0.004$) compared with that over the same period during CON (0.87 ± 0.06). There were no significant differences between HIT1 and HIT2 with respect to RER in this period ($p = 0.411$). Within the SP one-way ANOVA showed that there were no significant differences in RER between any experimental trials ($p = 0.173$). Furthermore, this was also the case when extending the analysis to include the RP period component i.e. RP & SP combined ($p = 0.215$).

DISCUSSION

The primary aim of this study was to profile the energy expended during, and for up to 11 hours after two routinely used HIIE protocols. Using a sample of healthy, moderately active males, this study has demonstrated that both HIIE interventions substantially raise EE during exertion and additionally increase EE and reduce RER for up to one hour after. Following this period however, no differences in EE or RER are apparent. Consequently, these findings demonstrate that the impact of these HIIE protocols on EE and RER after exercise is transient, with effects on EE particularly being of relatively small magnitude. These data imply that the impact of HIT on energy balance is primarily related to the EE induced during exertion, rather than to any influence on post-exercise metabolic rate.

In the present study both HIT1 and HIT2 substantially raised EE during exertion. The EE elicited by the HIIE protocols implemented in this study were substantially higher than that induced by the classical form of HIIE i.e. Wingate based sprint interval training. Specifically, two recent studies have identified remarkably similar EE profiles during sessions of sprint interval training with values of 595 kJ (Deighton et al. 2012) and 735 kJ (Hazell et al. 2012). Comparatively, both HIIE protocols utilised in the present investigation elicited substantially higher EE values compared with these previous studies, approximating two-fold higher with HIT1 and 4-4.5 fold higher with HIT2. These findings are of practical importance with the indication that these forms of HIIE may convey dual benefit by enhancing metabolic health parameters whilst also exerting a noteworthy influence on energy balance. Such information begins to allow us to make judgements regarding the most appropriate forms of HIT suitable for various populations with differing intervention goals.

Perhaps the most notable finding from the present investigation was that both HIIE protocols increased EE within the hour immediately after exercise (EPOC rapid phase) although this response was transient and of small magnitude. Specifically, the net increase in EE during this period was approximately 60 kJ in both HIIE interventions, equating to just two (HIT2) and five (HIT1) percent of the energy expended during the actual exercise component. The similarity in response between protocols is noteworthy given the marked difference in the duration and EE induced in HIT1 and HIT2. This is likely due to the similarity of exercise intensity between protocols, as it is known that exercise intensity is the most important determinant of the EPOC magnitude, explaining five times more of the variance than exercise duration or total work completed (Gore & Withers 1990).

The EPOC magnitude and duration in the present investigation was small and brief. This finding is congruent with other studies which have examined the effects of HIT on EPOC and reported small changes in post-exercise EE (Chan & Burns 2013; Williams et al. 2013; LaForgia et al. 1997) but at odds with a recent report which described a large increase in post-exercise EE after an acute bout of continuous moderate intensity cycling (Knab et al. 2011). This latter investigation is noteworthy given the gold standard methodology employed. Specifically, post-exercise metabolic rate was measured after 45 min of moderate intensity cycling (~73% $\dot{V}O_{2max,}$) in a respiratory chamber over an extended duration and the researchers reported a 795 kJ net increase in EE over 14.2 h. At first glance, when specifically compared with HIT2 in the present study, it is unclear why such markedly different outcomes are apparent given that the exercise intensity in HIT2 was significantly greater and associated EE comparable. Closer scrutiny of the study protocol however reveals one key difference between study methodologies. Notably, in the study conducted by Knab et al (2011) participants were fed back the energy which they expended during exercise (2171 kJ) so that they consumed a much greater amount of food on the exercise trial than their control trial. This extra energy would have significantly augmented resting metabolism (thermic effect of feeding) above that on the control trial and therefore the EPOC that the researchers reported reflects the true increase with exercise plus the increase promoted by a heightened food intake. Conversely, in the present investigation participants consumed identical meals across trials and therefore the independent effect of exercise was isolated.

In the current study EE was higher only during the RP of EPOC i.e. up to one hour after the end of exercise. This outcome is consistent with other studies which have examined the impact of HIIE on post-exercise

metabolic rate (Chan & Burns 2013; Williams et al. 2013). Specifically, Chan & Burns (2013) observed an increase in oxygen consumption up to 30 min after sprint interval exercise (four, 30s Wingate sprints). Moreover, with a similar protocol, Williams et al (2013) observed an increase in post-exercise oxygen consumption that lasted for up to 45 min. These findings, and those from HIT1 in the present investigation, are consistent with the suggestion that brief intense exercise impacts up on the EPOC early response, but that more significant and prolonged exercise is necessary to influence more prolonged changes in metabolic rate (LaForgia et al. 2006). The brevity of response, particularly with regards HIT2 in the present study, is not entirely clear given the longer protocol duration and relatively large associated EE. It is possible that the intermittent nature of the protocol attenuated the associated stress and physiological burden however other studies utilising intermittent protocols have documented significantly longer EPOC durations (Bahr et al. 1992; LaForgia et al. 1997). Both of these past investigations involved supramaximal exercise however, raising the possibility that the higher exercise intensity is significant. Furthermore, the post-exercise EE assessment protocols were very different compared to the current investigation with significantly lower sampling volume and hence greater interpolation of data.

In the present study both HIIE protocols had a significant impact on the RER during exercise and within the EPOC RP. Explicitly, during exercise the RER increased in each protocol representing an increasing reliance on carbohydrate oxidation commensurate with the high exercise intensity. Subsequently, within the immediate hour after exercise, RER decreased dramatically, indicating an enhancement of fat oxidation within the immediate hour after exercise. This response was transient however in the current study as participants consumed a breakfast meal one hour after exercise and the resultant switch in metabolic fuel preference masked any more prolonged enhancement of fat oxidation which may otherwise have occurred. Specifically, other investigations have shown that lower RER values may persist for several hours after interval type exercise (McGarvey et al. 2005; Malatesta et al. 2008). The mechanisms responsible for this change in metabolism possibly include an elevated catecholamine response to HIIE which may stimulate mitochondrial respiration, lipolysis and fat oxidation (Mulla et al. 2000). An increase in CO_2 retention as a means of replenishing the bicarbonate used to buffer lactic acid (Bahr et al. 1986, 1991a, 1991b, 1992; Short and Sedlock 1997; LaForgia et al. 2006) may also be important. Nonetheless, given the relatively small absolute increase in EE the importance of this change in fuel preference is debatable.

This investigation has notable strengths and limitations which should be recognised. Firstly, the strict control of diet and physical activity, prior to and during the investigation, is a key strength of the study. Moreover, the detail of post-exercise assessment in terms of the percentage of actual sampling time, rather than data interpolation, is also notable (51% of the total 11 h EPOC assessment). Limitations of this study include the lack of generalisability afforded by the use of a homogenous sample of young, healthy males. Specifically, previous investigations which have shown improvements in body weight/composition with the HIIE protocols utilised in the current investigation have studied overweight/obese individuals. Thus, we cannot be certain that the results obtained in the present investigation would transfer directly to this different population. Consequently, there would be merit in future studies to examine the impact of HIIE on metabolic rate in individuals who are overweight/obese whereby post-exercise changes may make a greater relative contribution to the overall energy deficit imposed by exercise. Sex based differences in metabolism and energy homeostasis also demand that investigations are conducted with females.

In conclusion, the present investigation has shown that HIIE exerts a notable influence on EE and RER during exertion however after the end of exercise these changes diminish rapidly. Consequently, the impact of HIT on energy balance appears to be chiefly determined by alterations in metabolic rate that occur solely during exercise, and not after. These findings may suggest that modifications in post-exercise metabolic rate (EPOC) are unlikely to be responsible for previously documented changes in body weight/composition that have been reported in response to HIT (Boutcher 2010). Additional work is needed to confirm this however given that changes in body weight/composition have previously been reported in individuals who are overweight/obese. Nonetheless, it is pertinent to consider other potential mechanisms contributing to the documented improvements in body weight/composition. Data from a recent investigation indicate that changes in appetite and energy intake may be important (Sim et al. 2013) however a better understanding of the impact of HIT on these parameters are needed before firm conclusions can be drawn.

AKNOWLEDGEMENTS

The research was supported by the National Institute for Health Research (NIHR) Diet, Lifestyle & Physical Activity Biomedical Research Unit based at University Hospitals of Leicester and Loughborough University. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

REFERENCES

Babraj JA, Vollaard NBJ, Keast C, Guppy FM, Cottrell G, et al (2009) Extremely short duration high intensity interval training substantially improves insulin action in young healthy males. BMC Endocr Disord 9:3

Bahr R (1992) Excess post exercise oxygen consumption--magnitude, mechanisms and practical implications. Acta physiologica Scandinavica. Supplementum 605:1-70

Bahr R & Maehlum S (1986) Excess post exercise oxygen consumption. A short review. Acta physiologica Scandinavica. Supplementum 556:99-104

Bahr R, Ingnes I, Vaage O, Sejersted OM, Newsholme EA (1987) Effect of duration of exercise on excess post-exercise O2 consumption. J Appl Physiol 62:485-90

Bahr R, & Sejersted OM (1991a) Effect of feeding and fasting on excess post exercise oxygen consumption. J Appl Physiol 71:2088-93

Bahr R, & Sejersted OM (1991b) Effect of intensity of exercise on excess post exercise O_2 consumption. Metabolism 40:836-41

Bahr R. Grønnerød O. Sejersted OM (1992) Effect of supra-maximal exercise on excess post exercise O2 consumption. Med Sci Sports Exerc 24:66-71

Bangsbo J, Gollnick PD, Graham TE, Saltin B (1991) Substrates for muscle glycogen synthesis in recovery from intense exercise in man. J Physiol 434:423-40

Bartlett JD, Close GL, MacLaren DPM, Gregson W, Drust B, et al (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

Børsheim E, & Bahr R (2003) Effect of exercise intensity, duration and mode on post-exercise oxygen consumption. Sports Med 33:1037-60

Boutcher SH (2010) High-intensity intermittent exercise and fat loss. J Obes: 868305

Brockman L. Berg K. & Latin R (1993) Oxygen uptake during recovery from intense intermittent running and prolonged walking. J Sports Med Phys Fitness 33:330-6

Burgomaster KA, Howarth KR, Phillips SM, Rakobowchuk M, Macdonald MJ, et al (2008) Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. J Physiol 586:151-60

Burgomaster KA, Heigenhauser GJF, Gibala MJ (2006) Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. J Appl Physiol 100:2041-7

Chad KE, Quigley BM (1991) Exercise intensity: Effect on post exercise O_2 uptake in trained and untrained women. J Appl Physiol 70:1713-9

Chan HH, and Burns F (2013) Oxygen consumption, substrate oxidation and blood pressure following sprint interval exercise. Appl Physiol Nutr Metab 38:182-87

Compher C, Frankenfield D, Keim N, et al (2006) Best Practice Methods to Apply to Measurement of Resting Metabolic Rate in Adults: A Systematic Review. J Am Diet Assoc 106:881-903

Deighton K, Barry R, Cannon CE, Stensel, DJ (2012) Appetite, gut hormone and energy intake responses to low volume sprint interval and traditional endurance exercise. Eur J Appl Physiol 113:1147-56

Department For Environment, Food and Rural Affairs. (2010) Family Food 2010 – A National Statistics Publication. London. 24-8.

Department of Health (DOH) (2011) Physical activity guidelines in the UK: Review and recommendations. London. 1-87

Fletcher GF, Balady G, Blair SN, Blumenthal J, Caspersen C et al (1996) Statement on exercise: benefits and recommendations for physical activity programs for all Americans. A statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. Circulation 94:857-62

Gaesser GA, Angadi SS (2011) High-intensity interval training for health and fitness: can less be more? J Appl Physiol 111:1540-1

Gibala MJ, Little JP, van Essen M, Wilkin GP, Burgomaster KA, Safdar A, Raha S, & Tarnopolsky MA (2006a) Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. J Physiol 575:901-11

Gibala MJ, Little JP (2010) Just HIT it! A time-efficient exercise strategy to improve muscle insulin sensitivity. J Physiol 588:3341-2

Gleeson TT (1996) Post exercise lactate metabolism: a comparative review of sites, pathways, and regulation. Annu Rev Physiol 58:565-81

Gore CJ, Withers RT (1990) Effect of Exercise intensity and duration on post-exercise metabolism. J Appl Physiol 68:2362-8

Haram P, Kemi OJ, Lee SJ, Bendheim MØ, Al-Share QY et al (2009) Aerobic interval training vs. continuous moderate exercise in the metabolic syndrome of rats artificially selected for low aerobic capacity. J Cardiovasc. Dis. Res 81:723-32

Hargreaves M, McKenna MJ, Jenkins DG, Warmington SA, Li JL, Snow RJ, & Febbraio MA (1998) Muscle metabolites and performance during high-intensity, intermittent exercise. J Physiol 84:1687-91

Harris JA, Benedict FG (1918) A Biometric Study of Human Basal Metabolism. Proc Natl Acad Sci USA 4: 370-3

Hazell TJ, Olver TD, Hamilton CD, Lemon P WR (2012) Two minutes of sprint interval exercise elicits 24-hr oxygen consumption similar to that of 30 min of continuous endurance exercise. Int J Sport Nutr Exerc Metab 22:276-83

Hood MS, Little, JP, Tarnopolsky MA, Myslik F, Gibala, MJ (2011) Low-volume interval training improves muscle oxidative capacity in sedentary adults. Med Sci Sports Exerc 43:1849-56

Kiens B, Richter EA (1998) Utilization of skeletal muscle triacylglycerol during post exercise recovery in humans. Am J Physiol 275:E332-7

Knab AM, Shanely RA, Corbin K, Jin F, Sha W, & Nieman DC (2011) A 45-Minute Vigorous Exercise Bout Increases Metabolic Rate for 14 Hours. Med Sci Sports Exerc 43:1643-8

LaForgia J, Withers RT, & Gore CJ (2006) Effects of exercise intensity and duration on the excess post-exercise oxygen consumption. J Sports Sci 24:1247-64

Laforgia J, Withers RT, Shipp NJ, & Gore CJ (1997) Comparison of energy expenditure elevations after submaximal and supramaximal running. J Appl Physiol 82:661-6

Leggate M, Carter WG, Evans MJ, Vennard RA, Sribala-Sundaram S, & Nimmo MA (2012) Determination of inflammatory & prominent proteomic changes in plasma & adipose tissue after high intensity intermittent training in overweight & obese males. J Appl Physiol 112:1353-60

Little JP, Gillen JB, Percival M, Safdar A, Tarnopolsky MA, Punthakee Z, Jung ME, et al (2011) Low-volume high-intensity interval training reduces hyperglycemia and increases muscle mitochondrial capacity in patients with type 2 diabetes. J Appl Physiol 111:1554-60

Macpherson RE, Hazell TJ, Olver TD, Paterson DH & Lemon PW (2011) Run sprint interval training improves aerobic performance but not maximal cardiac output. Med Sci Sports Exerc 43:115-22

Maehlum S, Grandmontagne M, Newsholme EA, & Sejersted OM (1986) Magnitude and duration of excess post exercise oxygen consumption in healthy young subjects. Metabolism 35:425-9

Malatesta D, Werlen C, Bulfaro S, Chenevière X, & Borrani F (2008) Effect of high-intensity interval exercise on lipid oxidation during post exercise recovery. Med Sci Sports Exerc 41:364-74

Matthews JNS, Altman DG, Campbell MJ & Royston P (1990) Analysis of serial measurements in medical research. Br Med J 300: 230-5

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

Mulla NAL, Simonsen L, Bullow J (2000) Post exercise adipose tissue and skeletal muscle lipid metabolism in humans: The effects of exercise intensity. J Physiol 524:919-28

Nikolovski S, Faulkner DL, Palmer TN, Fournier PA (1996) Muscle glycogen repletion from endogenous carbon sources during recovery from high intensity exercise in the fasted rat. Acta physiologica Scandinavica 157:427-34

Nybo L, Sundstrup E, Jakobsen MD, Mohr M, Hornstrup T, et al (2010) High-intensity training versus traditional exercise interventions for promoting health. Med Sci Sports Exerc 42:1951-8

Palmer TN and Fournier PA (1997) Replenishment of muscle glycogen after high-intensity exercise: a role for intramuscular lactate glyconeogenesis? Biochem Soc Trans 25:25-30

Pascoe DD, Gladden LB (1996) Muscle glycogen resynthesis after short term, high intensity exercise and resistance exercise. Sports Med 21:98-118

Richards JC, Johnson TK, Kuzma JN, Lonac MC, Schweder MM, Voyles WF, Bell C (2010) Short term sprint interval training increases insulin sensitivity in healthy adults but does not effect the thermogenic response to bta-adrenergic stimulation. J Physiol 588:2961-72

Roffey D, Byrne N, Hills A (2006) Day-to-Day Variance in Measurement of Resting Metabolic Rate Using Ventilated-Hood and Mouthpiece & Nose-Clip Indirect Calorimetry Systems. JPEN 3:426-32

Sedlock DA (1991b) Effect of exercise intensity on post exercise energy expenditure in women. Br J Sports Med 25:38-40

Shepherd SO, Cocks M, Tipton, KD et al (2013) Sprint interval and traditional endurance training increase net intramuscular triglyceride breakdown and expression of perilipin 2 and 5. J Physiol 591.3:657 - 75

Short KR, & Sedlock DA (1997) Excess post exercise oxygen consumption and recovery rate in trained and untrained subjects. J Appl Physiol 83:153-9

Sim AY, Wallman KE, Fairchild TJ, Guelfi KJ (2013) High-intensity intermittent exercise attenuates ad-libitum energy intake. Int J Obes (Lond) advance online publication 9 July 2013; doi: 10.1038/ijo.2013.102

Talanian JL, Galloway SDR, Heigenhauser GJF, Bonen A, & Spriet LL (2007) Two weeks of high-intensity aerobic interval training increases the capacity for fat oxidation during exercise in women. J Appl Physiol 102:1439-47

Tissot S, Delafrosse B, Bertrand O, Bouffard Y, Viale J, Annat G (1995) Clinical validation of the Deltatrac monitoring system in mechanically ventilated patients. J Intensive Care Med 21:149-53

Tjønna AE, Lee SJ, Rognmo Ø, Stølen TO, Bye A, Haram PM, Loennechen JP, et al (2008). Aerobic interval training versus continuous moderate exercise as a treatment for the metabolic syndrome: a pilot study. Circulation 118:346-54

Weir JB (1949) New methods for calculating metabolic rate with special reference to protein metabolism. J Physiol 109: 1-9

Weissman C, Sardar A, Kemper M (1990) *In vitro* evaluation of a compact metabolic measurement instrument. JPEN 14:216-21

Whyte LJ, Gill JMR, Cathcart AJ (2010) Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. Metabolism 59:1421-8

Williams, CB et al (2013) Changes in mechanisms proposed to mediate fat loss following an acute bout of high intensity interval and endurance exercise. Appl Physiol Nutr Metab. doi 10.1139/apnm-2013-0101

Wisløff U, Støylen A, Loennechen JP, Bruvold M, Rognmo Ø, Haram M, Tjønna AE, 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

World Health Organisation (WHO), (2010) Global recommendations on physical activity for health: Geneva. WHO publications:1-60

World Health Organisation (WHO), (2010) Global status report on non-communicable diseases: Geneva. WHO publications:116-59

Abbreviations

ANOVA: Analysis of variance

Con: Control

CO_2: Carbon dioxide

EE: Energy expenditure

EP: Exercise period

HIIE: High intensity intermittent exercise

HIT: High intensity intermittent exercise training

EPOC: Excess post-exercise oxygen consumption

HR: Heart rate

RER: Respiratory exchange ratio

RMR: Resting metabolic rate

RP: Rapid phase (EPOC)

SP: Slow phase (EPOC)

$\dot{V}O_{2peak:}$ Peak oxygen uptake

TABLES

Table 1 Characteristics of the study participants.

	Mean	Minimum	Maximum
Age (yr)	21± 2	19	24
Height (cm)	181.2 ± 7.5	172.1	192
Weight (kg)	73.9 ± 7.5	61.6	88.2
Body Mass Index ($kg.m^{-2}$)	22.5 ± 1.8	20.3	25.0
$\dot{V}O_{2Peak}$ ($mL.kg.min^{-1}$)	44 ± 6	33	58
RMR ($kJ.day^{-1}$)	8629 ± 636	7478	9655

Values are mean ± SD for all participant variables. n = 9. Minimum and maximum values for each variable are also presented.

FIGURE CAPTIONS

Fig 1 Schematic representation of the main trial protocols. Grey shaded box represents a two min cool down and five min resting period (seven min total).

Fig 2 Energy expenditure (mean ± SD) during the (a) exercise period; (b) EPOC rapid phase; (c) EPOC slow phase; (d) EPOC rapid & slow phases combined (total EPOC) for the control, HIT1 and HIT 2 trials. * significantly different from control (P<0.05); ‡ significantly different from HIT1& control (P<0.05)