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Acute Moderate and High-Intensity Endurance Exercise Suppresses *Ad-libitum* Energy Intake in Obese Males

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ABSTRACT

High-intensity exercise acutely improves suppression of appetite in populations with normal body mass index (BMI). However, whether moderate intensity exercise (MIE) and high-intensity exercise (HIE) can elicit similar (or greater) appetite suppression effects for obese populations are still relatively unknown. The main aim is to investigate the acute effects of MIE and HIE on the appetite score, eating behaviour and blood glucose regulation among the obese population. Twelve obese participants (age: 20.8 ± 1 yr, BMI: 34.1 ± 3 $kg \cdot m^{-2}$, $\dot{V}o_{2max}$: 30.7 ± 3 $ml \cdot kg \cdot min^{-1}$) were randomly allocated, in a crossover manner, with a 7-day interval in between (1) MIE (cycling at 60-75% HR_{max}), (2) HIE (cycling at 80-95% HR_{max}, 8-sec sprint x 12 sec rest) and (3) control (CON) condition after a 10-hr overnight fast. Physiological (fasting blood [glucose] and 24-hr calorie intake) and psychological responses (Three Factor Eating Questionnaire-R18, TFEQ-R18, and appetite score using Visual Analog Scale, VAS) were recorded prior to and after exercise interventions. Both MIE and HIE significantly reduced the calorie intake compared to CON ($P<0.05$), despite no changes in psychological measures were related to appetite (i.e. TFEQ-R18 and

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VAS) between the groups (*P>0.05*). A difference was found in fasting blood [glucose] level between trials in MIE (*P<0.05*), but not following the HIE condition (*P>0.05*). In response to acute intervention, both MIE and HIE improved some psychological appetite score and attenuated daily energy consumption; these positive effects could benefit obese and diabetic populations.

Keywords: Appetite, endurance, energy intake, high intensity exercise, moderate intensity exercise, obese

INTRODUCTION

The World Health Organization (WHO) cautions of the rising risk of chronic diseases since overweight and obesity rates are dramatically increasing every year. Statistics worldwide recorded a staggering population of 1.6 billion overweight and approximately 400 million obese (World Health Organization, 2017). Obesity can negatively impact the quality of life and increase the risk of cardiovascular diseases, diabetes and several types of cancers. It is currently recognised that overweight and obesity are the top 5 and top 10 risk factors for mortality and morbidity, respectively (Oreopoulos et al., 2008). It is clear that overweight and obesity impose various health-related problems, and the current global trend requires a practical strategy for weight management to expeditiously address the issue. It is recommended that weight loss intervention programs should combine the progressive reduction in energy intake (EI) with increases in energy expenditure (Alkahtani et al., 2014). Physical exercise is intimately related to energy intake and energy expenditure. It is widely believed that when exercise is performed regularly, it can elevate body energy turnover which then leads to an increase in energy expenditure, followed by a subsequent decline in body weight. Nevertheless, exercise-induced energy deficit may disturb the energy demand homeostasis, and this may affect body appetite regulation (Beaulieu et al., 2018). Accordingly, in the last few decades, there has been a surge in interest regarding the effects of exercise on appetite-related measures and hormones.

Physical activity has a prominent role in stimulating the negative energy balance by elevating energy expenditure. However, the magnitude of weight reduction is mainly dependent on calorie intake succeeding the activity; an increase in appetite to compensate the energy deficit will likely impose psychological difficulty towards any weight loss efforts. The relevance of this issue was discussed in articles from renowned media which highlighted the possibility of exercise hampering weight loss (Sailer et al., 2016) which may, in turn, promote fat accumulation (Leow et al., 2018). This is mainly a result of the increased desire for food intake following physical activity. The articles were primarily derived from evidence which indicated that energy consumption significantly increased a few minutes to hours after bouts of acute exercise (Martins et al., 2008a; Pomerleau et

al., 2004; Shorten et al., 2009). In contrast, some evidences suggested that appetite and energy consumption are relatively unchanged (Balaguera-Cortes et al., 2011; King et al., 2010). Several other studies claimed a higher rise in satiety hormone and weight reduction following acute bouts of exercise (Dorling et al., 2018; Ueda et al., 2009). These conflicting conclusions could be associated with the intensity and type of exercise used in previous studies. In support of this, clear justification is present which indicates that high-intensity exercise stimulates the circulating concentration of several appetite-related hormones, promoting weight reduction. Interestingly, the same study observed a lesser magnitude of changes in appetite-related measures and hormones with moderate intensity exercise (Bailey et al., 2015; Douglas et al., 2015; Martins et al., 2015).

Investigations on the impact of exercise concerning appetite-related measures and energy intake have mostly been restricted to limited comparisons of a single variation of type and intensity of exercise. Martins et al. (2015) reported that insulin levels and glucagon-like peptide (satiety hormones) were reduced and elevated, respectively, following isocaloric bouts of high and moderate intensity continuous cycling. However, this study mainly measured physiological data. The influence of these biomarkers on psychological indices related to appetite was not presented. Therefore, there is a notable lack of research investigating the impact of physiological and psychological measures of appetite following different exercise intensities (i.e. moderate-intensity exercise, MIE, and high-intensity exercise, HIE) and types of exercise (i.e. aerobic and resistance exercises) in the obese population. Therefore, the objective of this study is to investigate the acute effects of iso-caloric high intensity and moderate-intensity continuous exercise as well as high-intensity exercise on subsequent energy intake, appetite score and blood glucose for the obese adult population.

METHODS

Participants

Participants were screened for inclusion criteria, which includes participants that are: 1) healthy but sedentary obese individuals (body mass index of 30.0 $kg \cdot m^{-2}$ or greater) and 2) aged between 18 to 30 years old. A set of exclusion criteria were also used to determine participants' eligibility which includes: (1) current or history of cardiovascular, metabolic, gastrointestinal, renal or pulmonary disease; (2) significant change in body weight (≥5%); (3) pregnancy within the previous 12 months; (4) user of dietary supplements (excluding macronutrients) or illicit drug use; and (5) receiver of any investigational research agent within the previous 3 months. The sample size was calculated using previous studies (Maria et al., 2015). A minimum of 11 participants were required for the current study. This was on the basis of a sample size calculation of 95% confidence interval using the changes in calorie intake detected in a previous study (Sim et al., 2014). All experimental trials

were conducted at approximately the same time of day (± 1 hr). The subjects recorded the calorie intake within 24 hrs before the commencement of the first intervention protocol and were asked to perform activities as well as have similar meals in the subsequent trial. The subjects were thoroughly informed of the protocol and the possible risks and benefits of participation before a written informed consent was obtained. This study was registered, prior to its commencement, in the University hospital Medical Information Network Clinical Trials Registry (UMIN-CTR) with registration identification number: UMIN000016335. The study was approved by the Research Ethics Committee (FSR/SR243/038/2018) and conforms to the code of ethics of the Declaration of Helsinki.

Study Design

During the first visit, subjects underwent the familiarisation session and a baseline data collection. Subjects were then randomly assigned into a group of three intervention conditions in a crossover manner: (1) MIE (cycling at 60-75% HR_{max}), (2) HIE (cycling at 80-95% HR_{max}, 8-sec sprint x 12-sec rest) and (3) control (CON). MIE and HIE interventions were designed to induce approximately 250-kcal energy deficit. The trials were performed on different days with at least 7-day interval periods between each condition. Primary outcome measures included pre and post-exercises *ad-libitum* energy intake, changes in appetite score (using visual analogue scale, VAS), the food intake-behaviour analysis (using Three-Factor Eating Questionnaire-Revised 18-items, TFEQ-R18) and changes in fasting blood [glucose] level.

Pre-experimental Assessment

The preliminary examination on the subjects consisted of BMI and body composition, which were assessed using Bioelectrical Impedance Analyser (Omron BF-508, Omron Healthcare, Kyoto, Japan). Before the experimental trial, estimation of aerobic capacity was performed using a 20-m multilevel shuttle run. Results of the test were then converted to $\dot{V}o_{2max}$ based on a formula by Bammann et al. (2019). Subsequently, subjects were familiarised with the experimental protocols; on similar cycle ergometer throughout the current study and similar breakfast test meal (see *Experimental trials*).

Experimental Trials

In the 24-h before each experimental trial, subjects were required to record all food and drink consumptions in the form provided. On the morning of each experimental trial, subjects arrived at 0800 hours, having fasted for 10-hr (water was permitted during this time). Subjects then completed VAS, TFEQ-R18 and blood [glucose] for baseline (PRE) assessment. All subjects subsequently performed one of the exercise protocols (i.e. MIE

or HIE) or resting control protocol. The duration of each exercise session was individually designed (on the basis of the $\dot{V}o_{2max}$) to induce a 250-kcal (MIE and HIE) energy deficit. The exercise intensities were confirmed by monitoring HR. One hour after the exercises (i.e. MIE and HIE) or CON, each subject was provided with a standardised breakfast consisting of bread, milo drink and snacks (~442 kcal) after fasting blood [glucose] was taken. Next, subjects were instructed to fill the TFEQ-R18 and rate their Visual Analogue Scale before each trial. After each trial, VAS was immediately measured for rated hunger and followed with fasting blood [glucose]. A fingerprick-based blood glucose was conducted by trained personnel using standard protocol outlined in Rothberg et al. (2016). Additionally, subjects were provided with a standardised lunch and instructed to fill in the TFEQ-R18 immediately after consuming the lunch, 3 hours after each experimental trial. The lunch test meal consisted of fried rice, fruit drinks and snacks (~800 kcal). Subjects were then instructed to record all food and drink consumed 24-h prior to each trial.

Data Analysis Procedures

Appetite score was assessed using 100 mm Visual Analogue Scale immediately at pre and post exercise (Flint et al., 2000). The visual analogue scale took the form of five straight lines (100mm), each accompanied by a question anchored with words representing opposing extreme states of fullness, hunger, satiation, desire to eat and prospective food consumption at either end. 100 mm Visual Analogue Scale (Flint et al., 2000) was used in present study to measure the rate of hunger among the subjects, in terms of their subjective measurement such as determination to eat (DTE), hunger, fullness, and prospective food consumption (PFC) (Kawano et al., 2013; Martins et al., 2015). Scoring were calculated using the following formula: Average appetite score (mm) = [DTE + hunger + (100 - fullness) + PFC] / 4. Three factors eating questionnaire-revised 18 items (TFEQ-R18) were used to asses food intake-behaviour of subjects at (i) PRE and (ii) immediately post a standardised lunch meal. Consists of cognitive restraints (6 items), uncontrolled eating (9 items), and emotional eating (3 items). Energy intake (24-h) was assessed using a self-recorded food diary. Subjects were instructed to write everything they ate and drank (breakfast, lunch, dinner), consisted details such as time of the meals, food or beverages consumed and portion or serving of the food or beverages. The dietary macronutrient and overall energy intake were estimated (by accredited dietitians) using nutritional analysis software (Nutritics Ltd., Co. Dublin, Ireland) with calorie guidelines reference from the Ministry of Health (Malaysia) was used to insert the food information that was missing from the software database (Ministry of Health, n.d.). In each condition, fasting blood [glucose] at before and after exercise intervention were measured. The blood sample was analysed using the glucometer (One Touch model Ultra, LifeScan, Milpitas, California. Within-run precisions on the One Touch glucometer, as determined by coefficient of variation, was < 6%) .

Statistical Analysis

Descriptive statistics were used to characterise and compare the baseline characteristics of the subjects. Changes in energy intake, fasting blood [glucose], appetite score, and eating behaviour across all experimental conditions and time (i.e., pre and post) were assessed using repeated measures ANOVA. Post-hoc pairwise comparisons (Bonferroni adjustment method) were used to probe for specific differences. Where any differences were identified, 95% confidence intervals (95% CI) were used to display the likely range of the actual value in the sample population. Furthermore, effect size using partial eta squared (η^2_p) and Cohen's d (d_z) were calculated, which were defined as trivial (0–0.19), small (0.20–0.49), moderate (0.50–0.79), or large (> 0.80) (*ref; Cohen 1992). Analysis of data was conducted using the using the SPSS version 20 (SPSS Inc., Chicago, IL), with statistical significance accepted at $P<0.05$.

Results

Twelve obese male university students (mean ± SD: age 21 ± 1 yr, body weight 99 ± 1 kg, height 170 ± 6 m, BMI 34.1 ± 3 kg·m^{-2}, total body fat 33 ± 3 %, maximal oxygen uptake ($\dot{V}o_{2max}$) 30.7 ± 2.8 ml·kg·min-1) voluntarily participated in this study.

Psychological Factors of Appetite

Baseline cognitive restraint, uncontrolled eating, emotional eating and appetite score were not significantly different between conditions (P>0.05), and no significant alteration in all of these parameters between pre and post CON trial (P>0.05). Cognitive restraint post MIE was significantly lower from the post CON condition (mean difference: 32; P=0.002; 95% CI=15 to 49) and corresponding pre-trial (mean difference: 15; P=0.049; 95% CI=0.07 to 30) value. Emotional eating post CON was significantly higher post MIE (mean difference: 30; P=0.006; 95% CI=50 to 11) and post HIE (mean difference: 25; P=0.003; 95% CI=40 to 11) value, respectively. However, there were no significant changes in appetite score observed between the experimental condition (P>0.05) (Table 1).

Calorie Consumption

The group daily calorie intake in the MIE, HIE, and CON conditions are shown in Figure 1. Baseline calorie intake was not significantly different between conditions (P>0.05), and no significant alteration in calorie intake was found between pre and post CON trial (P>0.05). A trivial interaction effect ($F_{(1.8, 20)}$=7.6, P=0.004, η^2_p=0.41) and a large time effect ($F_{(1.0, 11)}$=39, P<0.001, η^2_p=0.78) of calorie intake was discovered throughout the intervention, but no effect on exercise intervention condition (P>0.05) were identified. Post hoc analysis revealed that calorie intake following CON (2163 ±478 kcal) was significantly higher than

Table 1

Appetite score (VAS) and food intake-behavior (TFEQ-R18)

	CON		MIE		HIE	
	Pre	Post	Pre	Post	Pre	Post
TFEQ-R18						
Cognitive Restraint	45.0 ± 28.4	63.6 ± 21.8	46.3 ± 28.9	31.4 ± 22.5†*	44.3 ± 23.0	45.3 ± 26.6
Uncontrolled Eating	49.4 ± 20.5	54.4 ± 12.7	52.2 ± 23.3	45.8 ± 35.8	55.8 ± 20.7	41.7 ± 18.0
Emotional Eating	51.3 ± 29.2	24.8 ± 14.9	56.3 ± 26.4	55.0 ± 23.9†	46.7 ± 24.6	50.1 ± 17.5†
Appetite score	48.8 ± 4.33	48.1 ± 3.86	46.0 ± 6.07	48.5 ± 9.38	48.8 ± 9.91	45.4 ± 7.37

Values are presented as mean ± SD

†Significantly different from the corresponding baseline (Pre) value (P<0.05);

*Significantly different from the corresponding CON value (P<0.05)

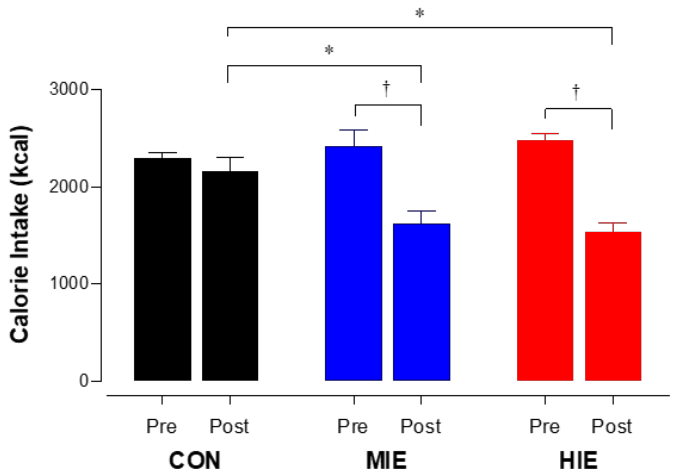

Figure 1. Mean ± SE total calorie intake at pre and post control (CON), moderate intensity exercise (MIE) and high-intensity exercise (HIE). †Significantly different from the corresponding baseline (pre) value (P<0.05); *Significantly different from CON condition (P<0.05).

post HIE (1622 ±460 kcal; P=0.005, d_z=1.01; 95% CI=233 to 1026) and post MIE (1533 ±339 kcal; P=0.004, d_z=0.74; 95% CI=206 to 875). Post calorie intake following MIE (mean difference: 795 kcal; P<0.001; 95% CI=403 to 1187) and HIE (mean difference: 943 kcal; P<0.001; 95% CI=4611 to 1276) was significantly lower from the corresponding pre-trial.

Fasting Blood [Glucose]

The group mean fasting blood [glucose] responses in the MIE, HIE, and CON conditions are illustrated in Figure 2. Baseline fasting blood [glucose] was not significantly different between conditions ($P>0.05$), and no significant change in fasting blood [glucose] was observed between pre and post CON trial ($P>0.05$). A trivial interaction effect of fasting blood [glucose] was observed throughout the intervention ($F_{(1.8, 20)}$=4.2, P=0.03, η^2_p=0.13), but no main effect of fasting blood [glucose] (P>0.05) were detected. Post hoc analysis revealed that fasting blood [glucose] following MIE (4.7 ±0.36 mmol) was significantly lower than post HIE (5.1 ±0.34 mmol; P=0.03, d_z=0.74; 95% CI=0.06 to 0.74) and post CON (5.0 ±0.36 mmol; P=0.006, d_z=0.84; 95% CI=-0.58 to -0.12).

Discussion

Reports of several previous investigations had shown that exercise (and exercise intensity) played a vital role in negative energy balance by exerting a suppression of energy intake in normal-weight adults. The present study aims to investigate the acute effects of MIE and HIE on the appetite score, eating behaviour and blood glucose regulation in obese men. Appetite score assessment was unaltered after a bout of MIE and HIE, compared to the CON condition. Our results contradict the belief that acute exercise night hamper weight loss efforts due to an acute change in hunger, fullness and satiety (King et al., 1996;

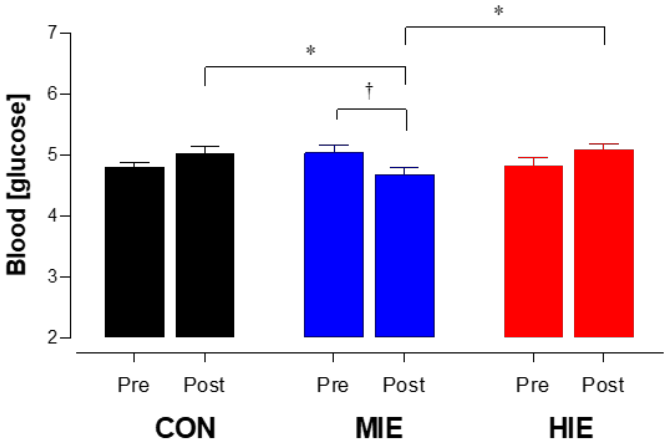

Figure 2. Mean ± SE fasting plasma [glucose] at pre and post control (CON), moderate intensity exercise (MIE) and high-intensity exercise (HIE). †Significantly different from the corresponding baseline (pre) value (P<0.05); *Significantly different from CON condition (P<0.05).

Pomerleau et al., 2004). Although the appetite score was not suppressed after an acute bout of exercise, as previously reported by Chanoine et al. (2008), we found that the total energy intake along with cognitive restraint levels were lowered in both modes of exercise intervention used in the current study. Unlike the usual dietary restriction methods which commonly result in a compensatory increase in energy intake (Taylor et al., 2018), the current investigation demonstrates that acute bout of exercise could induce energy deficit for at least ~180 min post-exercise. Overall, this finding suggests that both HIE and MIE can lower the total daily energy consumption. However, whether this improvement in energy deficit observed in obese adults could be preserved beyond the time point measured in the current study still remains unknown.

Over the past decade, the focus has been on investigating the intensity component of exercise as a way of facilitating the regulation of energy balance. Indeed, increasing exercise intensity enhances energy cost and promotes higher post-exercise energy expenditure. Fat oxidation increases the potential of skeletal muscles to utilise lipids and favours a decrease in energy intake. The central hypothesis of this study is that MIE and HIE would exert appetite suppression (as reflected by the acute decrease in energy intake), with higher intensity exercise eliciting a more significant suppression effect (Deighton et al., 2013). Although both exercise interventions attenuated the calorie intake post-exercise, the current study found no difference in the consumption of calories between both intensities of the exercises. It is worth noting that lowering of calorie intake observed in current study can be considered clinically significant, as indicated by considerable reduction in acute calories intake by 795 kcal and 943 kcal, post HIE and MIE, respectively. The US National Institutes of Health recommend that overweight or class I obese individual to reduce energy intake by 500 kcal·day^{-1}. It is estimated that overweight or class I obese to benefit a reduction of weekly body weight by 0.5 kg with daily calories deficit of 500 kcal (Fock & Khoo, 2013). Likewise, it has been suggested that, in diabetic population, a 1 mmol reduction in fasting glucose might reduce the risk of total stroke by 21% and ischemic heart disease by 23%. However, it is not known whether similar (or greater) reduction can be expected in overweight and obese population (Rodgers, 2004). Several past studies had shown that acylated ghrelin, which increases the desire to eat, was reduced and peptide YY concentration (PYY: a hormonal response which suppresses appetite desire) increased after moderate continuous exercise and high-intensity exercise (Martins et al., 2015; Ueda et al., 2009). Accordingly, Ueda et al. (2009) observed attenuation in psychological indices of appetite and total calorie intake in normal-weight subjects (Dorling et al., 2018). It was further discovered that suppression of appetite following exercise was corroborated with the finding of elevated biomarker related to the enhancement of satiety (i.e. GLP-1), which is speculated to result in reduced calorie intake of their obese subjects (Dorling et al., 2018; Ueda et al., 2009).

The food intake-behaviour was assessed using TFEQ, and the scale was selected due to its robust measure of cognitive restraint, uncontrolled eating and emotional eating (Chambers & Yeomans, 2008; Chanoine et al., 2008; Karlsson et al., 2000). Cognitive restraint is characterised by the restriction of food intake when conscious in order to influence and control body weight. As for uncontrolled eating, it represents the tendency to eat more than the regular intake because of loss of control over intake. Emotional eating characterises overeating or the inability to control eating due to a negative emotional mood, state or cues such as stress and loneliness (de Lauzon-Guillain et al., 2009). A model was proposed on how body mass, eating behaviour and physical activity could influence energy balance (Hill et al., 1995). This model proposes that one's susceptibility to physiologic cues of appetite (i.e. hunger, satiety and fullness) would be dependent on the individual's eating behaviours (i.e. cognitive restraint, uncontrolled eating and emotional eating). Using this model, it can be speculated that any changes in physical activity would individually (based on body weight and dietary behaviour) and directly affect a person's energy compensation.

Results of the current study did not reveal changes in uncontrolled eating behaviour in response to exercise. The results negate those of Hill et al. (1995) which found that being overweight with uncontrolled dietary behaviour lowered the energy intake with increased physical activity, resulting in overall negative energy balance. The current study observed higher emotional eating scores in the PRE and CON compared to the MIE and HIE conditions. Accordingly, previous studies had found a link between negative emotional eating behaviour as probed by TFEQ (Chambers & Yeomans, 2008; Chanoine et al., 2008; de Lauzon-Guillain et al., 2009). However, this is in contrast with the externality theory which asserts that the unresponsiveness of internal states found in obese individuals means that the population would unlikely be affected by emotional eating behaviour. In the current study, we observed that MIE lowered scores for cognitive dietary restraint. This study supports the evidence from previous observations in which overweight sedentary male subjects differed in terms of dietary restraints in response to an acute bout of moderate intensity exercise (Martins et al., 2008b). Contrary to expectations, this study did not find a significant change in cognitive restraint in response to HIE exercise. Logically, a more intense exercise is expected to enhance the awareness of food consumption, and thus, dietary restraint is expected for an individual who is actively monitoring to limit food intake.

Doucet et al. (2003) found no significant differences in the ratings of appetite sensations (hunger and satiety) in overweight participants before access to an *ad libitum* lunch (Doucet et al., 2003). The result of the present study revealed that there was no significant difference in VAS appetite score between and within groups. However, high-intensity exercise had shown a positive main effect. Despite no statistical differences observed within the experimental session, high-intensity exercise showed a trend of improvement in appetite suppression. According to Mattes and Cowart (1994), there is a lack of strong

influence between exercise and appetite because in some studies, VAS may not have been sensitive to detect specific minor changes after exercise due to small sample sizes. In contrast, a study on different modes and exercise intensities in healthy young men showed a correlation between appetite-regulating hormones and visual analogue scale. The study demonstrated acylated ghrelin and the ratings of VAS were suppressed while PYY increased during and immediately after the exercises (Kawano et al., 2013). Additionally, other studies further stated that ratings perceived from hunger, satiation and satiety were aligned with appetite-regulating hormones in suppressing the appetite (Martins et al., 2015; Stensel, 2011; Thackray et al., 2016; Ueda et al., 2009). The current study found significant reduction in blood [glucose] levels in response to MIE, but not for HIE and CON. However, it was noted that the reduction in blood [glucose] following MIE was clinically trivial. A possible explanation for this could be that a higher elevation of blood [lactate] (found in the previous study) may contribute to retarding the lowering of blood [glucose] during HIE (Guelfi et al., 2007; Júnior et al., 2001). However, this is only an extrapolation as this study did not measure the lactate level. The interference in the release of insulin and its response to skeletal muscle glucose uptake can be expected with vigorous activity, which may explain the higher blood [glucose] during post HIE compared to MIE. The gain in catecholamine levels in response to HIE found in the previous study may heighten renal glucose synthesis and simultaneously suppress the insulin-glucose uptake. It is suggested that portable glucometers may lack validity and reliability in comparison to laboratory glucose analysers, which provide more accurate results; similar to the industry reference standards (Salacinski et al., 2014). However, portable glucometers used in the current study seemingly produced valid and reliable results, as observed in previous studies (Chan et al., 1997; Naito et al., 1993). This demonstrates that the glucometer presently utilised was amply sensitive and appropriate for the blood glucose measurement.

CONCLUSION

In conclusion, this study has identified that MIE and HIE plays an essential role in negative energy balance by induced suppression of appetite and calories intake. These findings suggest that exercise (i.e. MIE and HIE) can reduce calories intake within 24-h post-intervention but may not be accounted for by changes in appetite score and eating behaviour.

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