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ORIGINAL ARTICLE

The acute effects of exercise-induced energy expenditure on physical activity energy expenditure

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Abstract

Exercise-induced energy expenditure (ExEE) is commonly adopted as a mean to volitional body mass loss. ExEE can also modulate physical activity energy expenditure (PAEE) and potentially limit the rate of body mass loss. The detrimental effect of ExEE on PAEE may be more prevalent in women than in men. This study was to investigate the sex differences in regulation of PAEE and body mass change following an acute exercise-induced perturbation in energy balance. In this 3-week study, 4 men and 6 women with body mass index (BMI) of 28.8 ± 2.3 kg/m2 were required to exercise on a leg ergometer, expending 15% of their total daily energy expenditure (TDEE) in each exercise session of week two (Exercise week). Participants had to complete three exercise sessions within the same week and on non-consecutive days. PAEE was assessed via wrist-worn accelerometers over the entire study period. Resting metabolic rate (RMR) and body composition were measured at the beginning of the study. Men had significantly greater stature ($p=0.021$), body mass ($p=0.035$) and resting metabolic rate (RMR) ($p=0.01$) than women. Whereas women body fat % was significantly greater than men ($p=0.004$). At the end of the study, there was no significant difference body mass % change ($p=0.409$) and ExEE ($p=0.91$) between men and women. There was no significant main effect of time for PAEE ($p=0.613$), or between sex ($p=0.470$). Three bouts of ExEE performed within the same week did not alter PAEE in men and women with overweight and obesity.

Keywords: exercise · body mass loss · physical activity · obesity · energy expenditure · adaptive thermogenesis

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Introduction

Obesity is a complex multifactorial disease, characterised by excess accumulation of body fat (Pi-Sunyer, 2009). Since 1980, prevalence of obesity has nearly doubled, which is particularly concerning as fat mass gain can promote other co-morbidities such as type 2 diabetes and certain forms of cancer (Pi-Sunyer, 2009).

Both genes and environment substantially contribute to changes in body weight across the life span (Hall 2017). Landmark studies on twins and non-related individuals showed that genetic differences can account for 50-70% of the disparity in body mass among the population (Locke et al., 2015). On the other hand, our genetic makeup has remained relatively unchanged over the past century, and yet, rates of obesity have been constantly increasing (Pi Sunyer, 2009). Consequently, several researchers suggested that urbanisation and modernisation have both contributed to the reduction of physical activity and facilitated availability of energy-dense foods, which collectively led to the creation of an obesogenic environment (Hall 2017). While both genes and environment seem to influence body mass, gains in body fatness are ultimately caused by prolonged periods of positive energy balance, a state where energy intake exceeds energy expenditure (Romieu et al., 2017). Conversely, a reduction in body fat can be induced through a state of negative energy balance over prolonged periods (Hall et al., 2012).

A state of negative energy balance can be induced via exercise, dietary restriction or by a combination of both (Romieu et al., 2017). When combined, exercise and dietary restriction seem to synergistically enhance body mass loss (Clark, 2015). By contrasts, interventions using exercise alone demonstrated a high degree of intra-individual variability and compensatory changes, with only modest reductions in body mass that are often below-expected (Barwell et al. 2009; Church et al. 2009). For example, Borer (2008) found that in absence of dietary restriction, daily exercise-induced energy expenditure (ExEE) of 400kcal yielded only 30% of the theoretically predicted body mass loss over the course of a month. Similarly, Barwell (2009) found that 7 weeks of ExEE resulted in highly divergent changes in body mass ranging between -5.3kg to +2.1kg in sedentary women. Initially, the below-predicted reductions in body mass were mainly attributed to poor adherence to exercise interventions. However, Church and colleagues (2009) compared actual to predicted body mass loss across three doses of ExEE (4, 8 and 12 kcal/kg/week) during 6 months of supervised exercise. Reductions in body mass nearly matched the predicted body mass losses in the 4 and 8 kcal/kg/week groups, however, in the 12 kcal/kg/week group, the actual body mass loss was significantly lower than the theoretically predicted. Interestingly, greater reductions in body mass were observed in the 8 kcal/kg/week group than the 12 kcal/kg/week group, despite a lower total ExEE (Church et al. 2009). These findings indicate that ExEE may modulate appetite, potentially promoting a compensatory response by increasing energy intake (EI). After 7-14 days of structured exercise, EI starts to track the disruption in energy balance and compensates for ~30% of the ExEE (Drenowatz 2015). Some individuals seem to be more susceptible to compensation via EI than others (Rocha et al. 2013). Moreover, due to ExEE, levels of PAEE can be also affected thereby reducing the energy deficit (Hall 2012). Notably, women seem to experience slower body mass loss rates than men, even when data is normalized (Donnelly and Smith 2005). Therefore, we hypothesise that ExEEwould decrease PAEE, and that this compensatory response would be more pronounced in women than in men.

Method

Ten (six women; four men) untrained participants (mean ± SD; age: 26 ± 6 years; stature: 173 ± 12 cm; body mass: 86.7 ± 13.1 kg; BMI: 28.8 ± 2.3 kg/m2) were recruited to the study. Posters were used to advertise the study to a university community and individuals who showed interest were provided with a participant information sheet, an explanation of the study requirements and answers to any questions they had. Participants were included in the study if between 18 and 40 years of age, BMI between 25 and 35kg/m2, under no medication and without any chronic disease. All participants self-reported being body mass stable and not involved in any form of structured exercise for at least 2 months prior to taking part in this study. Participants were fully informed both verbally and in writing about the study and given 7 days to decide whether to participate. A Physical Activity Readiness Questionnaire (PARQ) was completed to ensure there were no underlying health issues. The study was approved by Abertay University's School of Applied Sciences Research Ethics Committee (EMS1014), and all participants provided written informed consent. All experimental procedures were carried out in accordance with the World Medical Association Declaration of Helsinki.

To investigate the compensatory response in PAEE following ExEE, all participants completed the same experimental condition, thereby serving as their own controls. The study consisted of three sequential weeks namely Pre-exercise week (Day 1-7), Exercise week (Day 8-15), Post-exercise week (Day 15-21) with each week commencing on a Monday and ending on a Sunday (Figure 1). On day 1 of the study, anthropometrical, body composition and RMR measurements were taken for analysis. On day 22, body composition was measured again. PAEE was measured from day 1 to day 22 of the study. During the Exercise week (day 8-14), participants completed three bouts of exercise on non-consecutive days (Figure 1).

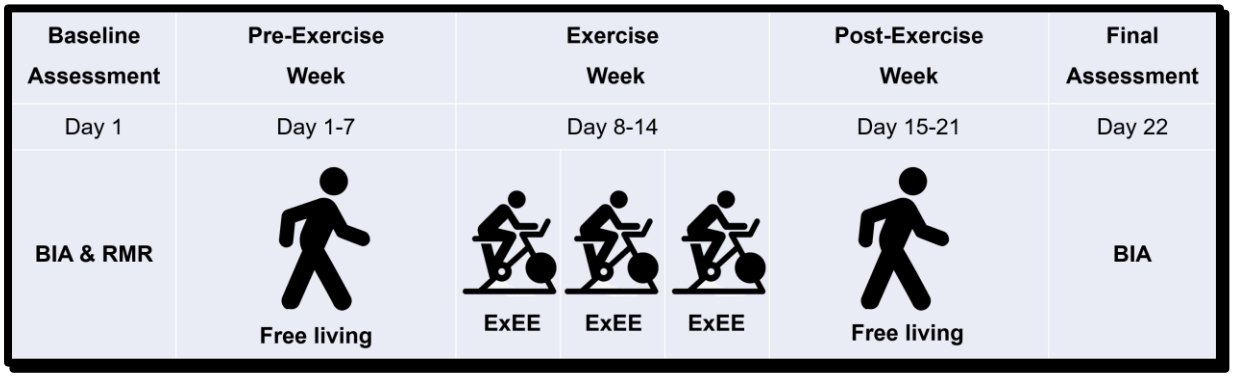

Figure 1. Schematic overview of the study design.
Note: BIA: bioelectrical impedance; RMR: resting metabolic rate; ExEE: exercise-induced energy expenditure.

To ensure consistency, participants visited the laboratory between 07:00 and 09:00 following an overnight fast from food, caffeine, nicotine and any caloric beverage in the 12 hours prior to testing but were allowed water ad libitum. Participants were instructed to avoid vigorous exercise 24 hours prior to testing to ensure accurate measurement of RMR. Upon arrival, participants were asked to void their bladder prior to commencing testing. Compliance was verified by verbal self-reporting to the investigator, to ensure accurate measurement of RMR and body composition.

On day 1, stature was measured to the nearest 0.1 cm using a stadiometer (SECA 216, SECA, Hamburg, Germany) in the Frankfort horizontal plane. The remaining anthropometric variables were measured during all laboratory visits. To ensure an accurate and consistent reading of body composition via the leg-to-leg bioelectrical impedance scale (SC-330ST, Tanita Europe, Amsterdam, the Netherlands), participants were asked to void their bladder and then remove excess clothing prior to recording body mass (kg) and body fat (%) to the nearest 0.1 respective unit.

RMR was determined by breath-by-breath analysis using an open-circuit indirect calorimetry (MetaMax 3B, Cortex Biophysik, Leipzig, Germany). The participant rested in a comfortable supine position in a quiet environment for 30 min. Full calibration of the metabolic cart was carried out in accordance with the manufacturer's guidelines using a 3-L calibration syringe, pressure calibration with a digital barometer (Barometer GA690, Castle Group, UK) and gas calibration using a 1.2 L bottle with 15% oxygen (O2), 5% and carbon dioxide (CO2), in nitrogen (N2). Next RMR was measured for 15–20 min and the average VO2 and VCO2 values from the last 10 min were used for analysis.

$$\text{RMR (kcal/day)} = (3.941 \times \text{VO2 (ml/min)} + (1.106 \times \text{VCO2 (ml/min)}) \times 1.44)$$

PAEE was measured from day 1 to day 22 using a tri-axial accelerometer (Mi Band, Xiaomi, Beijing, China) which was worn on the wrist of the dominant arm. This activity tracker has shown an accurate and valid alternative to more costly accelerometers that are validated for clinical use (El-Amrawy et al. 2015). Participants were advised to wear the accelerometers continuously except during their sleep and activities which would submerge the accelerometers in water. A day was considered valid only when the accelerometers were worn for at least 10h between 0700 and 2300 (LeCheminant et al. 2017). In addition, a week was considered valid only when it was comprised of four or more valid days.

Participant's TDEE was calculated by the gathered RMR, average PAEE from the Pre-exercise week and TEF which was assumed as 10% of TDEE:

$$\text{TEF(kcal)} = (\text{RMR} + \text{average PAEE}) \times 0.1$$

TDEE(kcal)=RMR + TEF + PAEE

Each bout of exercise consisted of steady-state cycling on a cycle ergometer (Monark Ergomedic 894E, Vansbro, Sweden) at 50 revolutions-per-minute (RPM) with resistance set at 2% of participant's body mass. The bout of exercise was performed until 15% of participant's TDEE was expended via ExEE, which was calculated per minute of exercise (kcal/min) through the ACSM leg ergometer metabolic equation (Pescatello 2014):

Statistical analyses were performed using SPSS version 24.0 (IBM Corp., Armonk, N.Y., USA. Group differences in descriptive characteristics were analysed using unpaired t-tests. PAEE from each phase were analysed using a 3 x 2 (Time x Sex) mixed model ANOVA, whereas body mass from the pre-measurement and post-measurement days were analysed through paired t-tests. Significance level was set at $p \leq 0.05$. All data are presented as means ± standard deviation.

Results

Table 1 shows descriptive characteristics of the participants. Men had significantly greater stature ($p = 0.021$), body mass ($p = 0.035$) and RMR ($p = 0.01$) than women. Whereas women body fat % was significantly greater than men ($p = 0.004$). There was no significant difference in BMI ($p = 0.694$), body mass % change ($p = 0.409$) and ExEE ($p = 0.91$) between men and women.

Table 1. Descriptive characteristics of the subjects (n= 4 men, 6 women)

Variable	Men	Women
Age (years)	30 ± 8	23 ± 2
Height (cm)	184.3 ± 9.6*	165.7 ± 6.2
Body mass (kg)	96.9 ± 12.8*	79.9 ± 8.6
Body fat (%)	24.5 ± 3.7	34.5 ± 4**
BMI (**kg/m²**)	28.4 ± 2.1	29.1 ± 2.5
RMR (Kcal/day)	1721 ± 475*	1053 ± 122
ExEE (Kcal/week)	986 ± 292	632 ± 63
Body mass change (%)	1.2 ± 1.9	0.6 ± 0.5

Note: *Significantly greater in men. **Significantly greater in women.

There was no significant main effect of time on PAEE ($p = 0.613$), or between sex ($p = 0.470$). There was also no significant interaction between sex and time ($p = 0.806$).

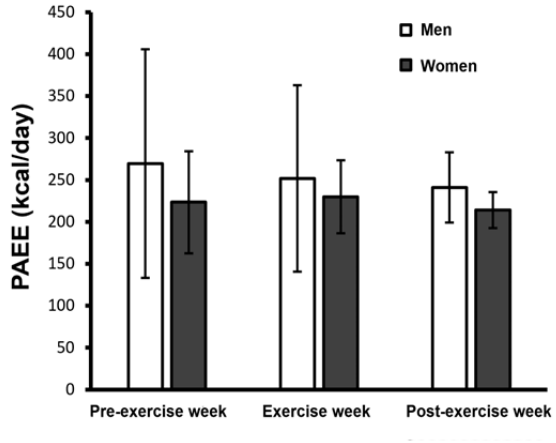

Figure 2. PAEE levels throughout the study period and the influence of ExEE

Discussion

The aim of this study was to investigate whether acute exercise-induced perturbation in energy balance would decrease levels of PAEE. The main finding of this investigation is that an acute exercise-induced disruption in energy balance does not affect PAEE neither in men nor in women. The findings are in line with previous short-term studies examining PAEE changes due to ExEE (Alahmadi et al. 2011; Stubbs et al. 2002; McLaughlin et al. 2006).

The present study indicates that a potential exercise-induced compensation in PAEE might still exist over the short-term. Although not statistically significant, when compared to the pre-exercise phase, a 7% decline in men and a 3% increase in women in levels of PAEE were observed during the exercise week. Whereas levels of PAEE declined in the post-exercise week in both men and women by 12% and 4%, respectively. Collectively, over the course this study, the average decline in PAEE in men and women accounted for 1% and 8%, respectively. Interestingly, during the exercise

phase, only men showed a mild decrease in PAEE (Hollowell et al. 2009). This could be attributed to the modality of the exercise bouts, which seems to elicit greater sense of acute fatigue in men than in women (Hunter et al. 2016). On the other hand, these compensatory changes in PAEE seem to usually diminish over time as fitness levels increase PAEE (Hollowell et al. 2009). Interestingly, however, the reduction in PAEE became more pronounced in both men and women during the post-exercise week, when participants were in free-living conditions and no self-reported exercise was performed. This delayed reduction in PAEE could be potentially explained by the two to four-day corrective lag that is often observed when energy balance is perturbed (de Castro 2004). Nevertheless, this potential delay required to adjust EE, and the reduction in PAEE that was observed in both sexes during the post-exercise phase partially supports the constraint model of energy expenditure (Pontzer 2015). In contrast, the additive model of energy expenditure suggests that an increase in ExEE results in a linear increase in TDEE, Pontzer (2015) hypothesised that EE is genetically set with increases in one component being offset by reductions in another. The same group of researchers validated this model in a large study showing that additional physical activity seems to markedly increase TDEE only in inactive individuals, whereas plateaux in highly active ones (Pontzer et al. 2016). While participants of the present study reported no involvement in any form of structured exercise, weekly physical activity levels were not assessed prior to commencing the intervention. Therefore, highly active participants at baseline might have experienced a plateau in PAEE, whereas more inactive participants might have experienced a more linear increase in TDEE via ExEE without compensating in PAEE (Pontzer et al. 2016).

Similarly to PAEE, changes in body mass were not significant in either men or women, potentially due to the magnitude of ExEE as well as duration of the study. Notably, body mass change increased in both men and women by 1.2% and 0.6%, respectively. Relatively similar body mass changes were observed in a short-term study by McLaughlin (2006) where women achieved a modestly greater reduction in body mass via exercise than men. Although the present study's results show a lesser adverse change in body mass in women, they may support the notion that a potential exercise-induced body mass gain can occur (Barwell et al. 2009). While this study involved only three exercise bouts that collectively yielded an ExEE of 986 ± 292Kcal and 632 ± 62Kcal in men and women, respectively, a marginal body mass gain was observed for both sexes. The body mass change observed in men (+1.2%) and women (+0.6%) may suggest that a compensation via energy intake might have occurred, which was potentially mediated by the exercise bouts (Rocha et al. 2013). It has been noted that when individuals are aware of the ExEE they can be more likely to compensate with an increased EI through consumption of energy dense foods (McCaig et al. 2016). Although appetite and EI responses to acute exercise-induced energy deficits seem to be similar between men and women (Thackray et al. 2016), a divergent response in eating behaviour between more active and less active participants might have been a confounding factor (Myers et al. 2016).

Our study provides a minor addition to the body of existing evidence (Alahmadi et al. 2011; Stubbs et al. 2002; McLaughlin et al. 2006). Nevertheless, it is important to extrapolate our findings as conservative due to the several limitations. A major limitation of this study were the small sample number and the lack of control for menstrual cycle phase and menstrual cycle regularity. In fact, EE and EI seem to vary between the follicular phase and luteal phase of the menstrual cycle. During the luteal phase, both EI and EE tend to be higher than in the follicular phase (Bryan et al. 2006). However, rather than both linearly increase, EI seems to transiently exceed EE hence eliciting an acute energy surfeit, which may have compromised levels of PAEE and body mass change during the study (Davidsen et al. 2007). Second, our study did not control for individuals' aerobic capacity and hence, some may have experienced greater discomfort and acute sense of fatigue during exercise bouts than others, potentially affecting both PAEE and EI (Drenowatz 2015; Hollowell et al 2009). Lastly, PAEE was objectively assessed via commercially available tri-axial accelerometers. While the device used in the present study was shown to be precise and accurate when compared to other commercially available activity trackers (El-Amrawy et al. 2015), extrapolation of movement data to estimation of energy expenditure has been often shown to introduce significant errors (Lam and Ravussin 2016). This is because they are not able to detect muscle activities toward postural efforts against gravity, fidgeting, static exercise and its intensity (Wang et al. 2017). Therefore, quality of data could be improved by adopting clinically validated tri-axial accelerometry (Lam and Ravussin 2016).

The practical applications from this study should be extrapolated conservatively due to the presented

limitations. Nevertheless, in respect to the previously published research, ExEE may have a minor impact on PAEE, particularly in individuals who are predisposed to body mass gain (Hollstein and Piaggi 2023). And moreover, ExEE seems to be paramount in promoting body mass management following body mass loss (Bourdier et al., 2023). Besides that, ExEE is still a crucial tool to improve cardio metabolic health and reduce risk of all-cause-mortality (Kraus et al., 2019).

In conclusion, this study indicated that three separate bouts of ExEE, each amounting to 15% of individuals' TDEE, did not significantly alter levels of PAEE and body mass. Moreover, the ExEE elicited changes in PAEE and body mass did not differ between men and women.

Declarations

Authors' contributions: MA conceived the study, carried out data collection and analysis. MA drafted the initial manuscript. AC proofread and edited the initial man-uscript and provided valuable insights.

Declaration of interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Funding: Not available.

Ethical approval: Participants provided written consent after being briefed about the objectives and potential risks associated with the study. The research adhered to the principles of the Helsinki Declaration and received approval from the National Medical Ethics Committee (approval number: EMS1014).

Data availability: All collected data are included in the manuscript. Raw data is available upon reasonable request to the corresponding author.

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