Title: Effects of Aerobic, Strength or Combined Exercise on Perceived Appetite and Appetite-Related Hormones in Inactive Middle-Aged Men

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Running Head: Exercise Mode and Appetite

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Abstract:

Aerobic exercise (AE) and strength exercise (SE) are reported to induce discrete and specific appetite-related responses; however, the effect of combining AE and SE (i.e. combined exercise; CE) remains relatively unknown. Twelve inactive overweight men (age: 48 ± 5 y; BMI: 29.9 ± 1.9 kg·m^2) completed four conditions in a random order: 1) non-exercise control (CON) (50 min seated rest); 2) AE (50 min cycling; 75% VO_{2peak}); 3) SE (10 × 8 leg extensions; 75% 1RM); and 4) CE (50% SE + 50% AE). Perceived appetite and appetite-related peptides and metabolites were assessed prior to and up to 2 h post-condition (0P, 30P, 60P, 90P, 120P). Perceived appetite did not differ between trials ($p < 0.05$). Acylated ghrelin was lower at 0P in AE compared to CON ($p = 0.039$), while pancreatic polypeptide (PP) was elevated during recovery following AE compared to CON and CE. Glucose-dependent insulinotropic peptide (GIP_{total}) was greater for all exercise conditions compared to CON during recovery, as was glucagon, although concentrations were generally highest in AE ($p < 0.05$). Glucose was acutely increased with SE and AE ($p < 0.05$), while insulin and C-peptide were higher after SE compared to all conditions in early recovery ($p < 0.05$). In inactive, middle-aged men AE, SE and CE each have their own distinct effects on circulating appetite-related peptides and metabolites. Despite these differential exercise-induced hormone responses, exercise mode appears to have little effect on perceived appetite compared with a resting control in this population.

Keywords: concurrent exercise, sedentary, hunger

Introduction

The World Health Organization (2014) has reported that there are in excess of 1.9 billion overweight adults worldwide. Such extent of obesity is of concern given the numerous health implications resulting from excess adipose tissue. Furthermore, there is some preliminary evidence to suggest that large volumes of adiposity may alter appetite-related hormone concentration, function and signalling (Batterham et al. 2003a), which could potentially make weight (fat) loss difficult and lead to further gains in adiposity. There is a growing body of evidence to suggest that both an acute bout of exercise, as well as regular exercise training, may be beneficial in achieving a negative energy balance by inducing perceptions of reduced appetite (i.e. hunger, desire to eat, prospective food consumption), the total amount of energy consumed, and/or the circulating concentrations of a number of appetite-related peptides (Broom et al. 2009; Guelfi et al. 2013; Rosenkilde et al. 2013; Sim et al. 2015). More specifically, an acute bout of exercise is associated with reduced concentrations of circulating ghrelin (Balaguera-Cortes et al. 2011; Heden et al. 2013; Sim et al. 2015), which is the only known gastrointestinal hormone to stimulate increased appetite (orexigenic properties) (Druce et al. 2005; Levin et al. 2006); whilst increasing concentrations of gastrointestinal hormones such as peptide tyrosine-tyrosine (PYY_{3-36}) and glucagon-like peptide-1 (GLP-1) (Chanoine et al. 2008; Sim et al. 2015) that suppress appetite (Degen et al. 2005, 2006). However, it is important to note that these changes in appetite-related peptides following acute exercise are often transient and do not necessarily always translate into changes in perceived appetite or a detectable reduction in energy intake (Balaguera-Cortes et al. 2011; Deighton et al. 2013; Holt et al. 2016). Furthermore, the precise effect of exercise on appetite and energy intake appears to depend on the specific characteristics of the exercise itself, with varying effects of exercise intensity, duration and mode reported in the literature (Broom et al. 2009; Laan et al. 2010; Sim et al. 2015).

With specific respect to the mode of exercise, studies comparing a single bout of aerobic exercise (AE) or strength exercise (SE) have reported mode-specific responses on appetite-related hormones (Broom et al. 2009; Balaguera-Cortes et al. 2011). However, the issue of which exercise modality suppresses appetite to a greater extent is less clear, with reports of greater suppression of perceived hunger with AE compared with SE (Broom et al. 2009), no difference in post-exercise energy intake between modalities (Balaguera-Cortes et al. 2011; Cadieux et al. 2014) and possible alterations in food preferences (McNeil et al. 2015). In addition, previous research has focused on young active populations, with no studies examining these issues in an inactive overweight group for whom the potential effects may be most relevant. Furthermore, no studies have examined the effect of a combined session of aerobic and strength exercise on appetite responses. This is important given

that current exercise guidelines encourage adults to participate in combined exercise (CE; i.e. combined AE and SE) (Donnelly et al. 2009). Previous studies suggest that CE can provide positive physiological adaptations that are similar, if not equivalent to both AE and SE when performed in isolation (Donges et al. 2012), though the effects on appetite-related hormones remains relatively unknown. Therefore, the primary aim of this study was to investigate the effect of an acute bout of AE, SE and CE on appetite-related hormones and perceived appetite in a population of untrained, overweight men.

Materials and Methods

Participants

Twelve inactive, overweight, middle-aged men (no regular pattern of planned or incidental exercise > 1 $d \cdot wk^{-1}$ in the preceding 12 months) volunteered for the study (data is presented in Table 1). Participants were required to obtain medical clearance, and to complete an oral glucose tolerance test (OGTT) and maximal graded exercise stress test (GXT) to exclude diabetes and symptomatic cardiovascular disease, respectively. Participants were not taking medications or on any special diet that may have influenced their perceived appetite responses. The study was approved by the Human Ethics Committee of Charles Sturt University and written informed consent was attained from all participants.

Study Overview

All participants attended 2 baseline laboratory visits for familiarisation and assessment of baseline characteristics, followed by 4 trials involving experimental conditions (i.e. exercise or no exercise) and post-condition measures administered in a randomised and counterbalanced order based on a Latin Square design. The 4 experimental conditions involved a non-exercise control (CON), aerobic exercise (AE), strength exercise (SE), and combined exercise (CE).

Baseline Testing

Initial assessment involved measurement of blood pressure, stature, body mass, waist and hip girths, and a dual-energy X-ray absorptiometry (DXA) scan (Norland XR800 with Illuminatus DXA, version 4.2.0, Cooper Surgical Company, Turnbull, CT, USA) to determine percentage of total body fat. This was followed by a fasted 75 g OGTT (Lomb Scientific, Thermo Fischer Scientific, NSW). Venous blood samples (10 mL) were drawn from a medial antecubital vein and aliquoted into serum separator tubes pre-ingestion, and at 30 min intervals for 2 h post-ingestion (5 × samples).

At the second baseline laboratory visit, a GXT was completed using an electromagnetically braked cycle ergometer (Velotron, RacerMate Inc., Seattle, Washington, USA). The test commenced at 25 W, and increased by 25 $W \cdot min^{-1}$ until volitional exhaustion, which typically coincided with a pedalling cadence < 40 rpm. During the test, heart rate (HR) was monitored with a 12-lead electrocardiogram (ECG) and participants breathed through a mouthpiece connected to a calibrated metabolic gas oxygen analysis system (TrueOne 2400 metabolic system, Parvomedics, Sandy, Utah, USA) to allow for the determination of VO_{2peak}. Following ~30 min of recovery, participants underwent one repetition maximum (1RM) strength testing of the quadriceps musculature on a leg extension machine (Leg Extension Basic; Panatta Sport, Apiro, Italy). To obtain 1RM strength, participants first completed a set with light resistance for familiarisation, followed by ascending resistances until two repetitions were unable to be completed. 1RM testing typically required 3-4 attempts, separated by ~2 min recovery. The VO_{2peak} and 1RM test results were used to determine the workload for the respective experimental conditions.

Trials

The 4 trials were completed by each participant in a counterbalanced order with at least 7 days between visits. Participants were instructed to record their food intake in the 24 h prior to the first trial and replicate this diet prior to the subsequent trials. In addition, participants were required to refrain from alcohol consumption and vigorous physical activity during the preceding 24 h and to fast for 10 h prior to arrival at the laboratory.

Each trial commenced between 0600-0800 h, with the exact time standardised for each participant. Upon arrival, a 12-lead ECG was applied to the participant and a mouthpiece was fitted which was connected to a calibrated metabolic gas oxygen analysis system (TrueOne 2400 metabolic system, Parvomedics, Sandy, Utah, USA) to monitor HR and VO_2, respectively. The CON condition involved quiet sitting for 50 min. For the AE condition, participants cycled for 50 min (Velotron, RacerMate Inc., Seattle, Washington, USA) at a pedalling resistance of 50% of the peak workload reached in the GXT, which equated to 78 ± 3.89% of VO_{2peak}. The SE condition involved 10 sets of 8 repetitions of bilateral leg extension exercise at a resistance of 75% of 1RM with 150 s recovery between sets. The CE condition consisted of 50% of both the SE (5 × 8 repetitions) and AE (25 min) conditions at a matched intensity. The AE and SE components were intended to align with exercise intensity and volume recommendations for adults (Pollock et al. 2000; Donnelly et al. 2009); however, rather than utilising a strength training protocol covering all the major muscle groups of the body as per typical guidelines, we employed a protocol of leg extension exercise to provide a more consistent stimulus throughout the session and keep the exercise restricted to the lower limbs to match the aerobic exercise stimulus. This

protocol was based on previous research comparing metabolic responses to aerobic, resistance and combined exercise (Donges et al. 2012). The AE, SE and CE protocols were not matched for energy expenditure given the inherent difficulties in doing so (Bloomer 2005). Rating of perceived exertion (RPE) was assessed following each strength set and every 10 min during the cycling bouts.

Assessment of Perceived Appetite

Perceived hunger, fullness, desire to eat and prospective food consumption were assessed using a validated 100 mm visual analogue scale (VAS) (Flint et al. 2000). Perceived appetite was recorded at baseline (pre), immediately post (0P), 30 min post (30P), 60 min post (60P), 90 min post (90P) and 120 min post (120P) condition.

Blood Sampling

Venous blood samples (10 mL) were drawn from a medial antecubital vein at pre, 0P, 30P, 60P, 90P and 120P during each trial. All samples were assayed for glucose and lactate concentrations from a syringe in duplicate using a blood-gas analyser (ABL800, Radiometer, Copenhagen, Denmark). The remaining blood was immediately aliquoted into pre-chilled tubes treated with ethylenediaminetetraacetic acid (Becton Dickinson, Sydney, Australia) and serine protease inhibitor (AEBSF; Pefabloc® SC, Sigma-Aldrich, St. Louis, USA) according to the manufacturer's instructions. Tubes were immediately centrifuged at 3500 rpm for 15 min at 4°C and separated plasma was stored at -80°C. A commercially available human metabolic hormone analyte panel (Cat. No# HMHMAG-34K; Milliplex, Millipore Corporation, MA, USA) was used according to the manufacturer's instructions (Luminex Corporation, Austin, TX, USA) to determine concentrations of: insulin, C-peptide, glucagon, acylated ghrelin, glucose-dependent insulinotropic peptide (GIP_{total}), $GLP\text{-}1_{active}$ (both the $GLP\text{-}1_{7\text{-}36}$ and $GLP\text{-}1_{7\text{-}37}$ isoforms), leptin, pancreatic polypeptide (PP) and PYY_{total}. Intra-assay coefficient of variation was < 7% for the abovementioned analytes. Additionally, total cholesterol, high-density lipoprotein cholesterol, and triglycerides were measured in the fasting blood sample according to manufacturer's instructions via a high-throughput automated blood analyser (EXL, Dimension®, Siemens Healthcare Diagnostics, Sydney, Australia). The Friedwald equation was used to estimate low-density lipoprotein cholesterol.

Statistical Analysis

Data are reported as mean ± standard deviation (SD) unless otherwise indicated. Two-factor (condition × time) repeated-measures analysis of variance (ANOVA) were conducted to assess the effect of each condition on

appetite-related hormones and perceived appetite in response to both exercise and recovery. If an interaction or main effect was observed, Tukey's post-hoc tests were applied to identify the source of significance, which was accepted at $p < 0.05$. Statistical analyses were performed with PASW Statistics, version 20.0 (SPSS Inc., Chicago, Ill., USA) and GraphPad Prism version 7 (San Diego, CA, USA).

Results

Characteristics of Exercise

In response to exercise, there was a significant increase in HR, VO_2, lactate and RPE compared to CON (Table 2). When comparing between exercise modes, mean VO_2 was significantly higher during AE compared to SE ($p < 0.001$) and CE ($p = 0.035$), while mean VO_2 in CE was greater than SE ($p < 0.001$). Also, estimated mean energy expenditure was significantly greater following all exercise conditions compared to CON ($p < 0.0001$). Estimated mean energy expenditure was greater during AE compared to SE ($p < 0.0001$) and CE ($p = 0.0038$), and CE was higher than SE ($p < 0.001$). Likewise, the HR response to AE was greater than CE ($p = 0.017$). However, when comparing the VO_2 and HR responses of AE alone to the AE component of CE, there was no difference between conditions ($p > 0.05$). Likewise, during the SE component of CE, there was no difference in HR or VO_2 ($p > 0.05$) compared to the SE condition alone. There was no significant difference in VO_2 during recovery from exercise between conditions ($p > 0.05$) (Table 2). Lactate was significantly higher following all exercise conditions compared with CON, with the greatest increase evident in SE ($p = 0.001$; Table 2). There was no significant difference in mean session RPE between exercise modes ($p > 0.05$).

Perceived Appetite

There was no significant interaction effect of condition and time for perceived hunger, fullness, desire to eat or prospective food consumption ($p > 0.05$; Fig. 1). However, there was a main effect of time for all conditions, with increases in hunger, desire to eat and prospective food consumption; and decreased fullness over time throughout each condition ($p < 0.001$).

Hormone and Metabolite Responses

There was a significant interaction effect of condition and time on ghrelin in response to exercise ($p = 0.019$), with significantly lower ghrelin immediately after AE compared with CON ($p = 0.039$). In contrast, there was no significant interaction of condition and time for PYY_{total}, leptin, or $GLP\text{-}1_{active}$ ($p > 0.05$; fig. 2), although there was a main effect for time for each with decreased PYY_{total}, leptin, and $GLP\text{-}1_{active}$ throughout recovery ($p < 0.05$). There was no immediate effect of exercise on PP; however, there was a main effect of condition during recovery, with higher PP following the AE condition compared with CON and CE. Likewise, there was no immediate effect of exercise on GIP_{total}, while in recovery greater concentrations were evident after the three exercise conditions compared to CON at 30P (SE and CE > CON), 60P and 90P (AE, SE and CE > CON) (fig. 2F). Regarding glucose and related hormones, there were significant interaction effects for condition and time for glucose, insulin, glucagon and C-peptide ($p < 0.05$). Immediately-post trial, there was an increase in glucose concentration following AE and SE compared to CON ($p < 0.05$; fig. 2G), with SE remaining greater than CON ($p = 0.020$) and CE ($p = 0.033$) at 30P. Glucagon was elevated in response to all exercise conditions at various timepoints in recovery compared with CON, although levels were generally higher after AE and CE compared with SE (0P, 30P, 90P and 120P) ($p < 0.05$; fig. 2I). Insulin concentrations were elevated following SE compared to CON, AE and CE at both 0P and 30P ($p < 0.05$; fig. 2H). In addition, C-peptide concentrations were significantly higher in SE than CON, AE and CE at 0P and 30P, and continued to remain higher than CON and AE at 60P ($p < 0.05$; fig. 2J).

Discussion

Previous research has elucidated that AE and SE have distinct effects on appetite in young, active men (Broom et al. 2009; Balaguera-Cortes et al. 2011). However, the effect in inactive, overweight individuals is not known, and the effect of combining these modes of exercise (e.g. CE), as is commonly performed in an exercise setting, has not been examined. As such, the aim of this study was to investigate the response of perceived appetite and appetite-related hormones and metabolites following CE, AE and SE in an inactive middle-aged cohort. The present study revealed that each exercise mode induced specific effects on the concentrations of several appetite-related peptides such as acylated ghrelin, PP, GIP_{total}, insulin and C-peptide. However, these differences were generally transient and did not translate into differences in perceived appetite between the exercise modes or compared with the resting control. Thus, it appears that an acute exercise stimulus (irrespective of mode) does not alter perceived appetite responses among middle-aged men.

With respect to the effect of each mode of exercise on the circulating concentrations of the appetite-related peptides and metabolites measured here, our study supports previous observations that AE and SE have distinct effects. More specifically, we found that AE transiently reduced acylated ghrelin and increased PP post-exercise, while SE increased insulin and C-peptide. The decreased concentration of acylated ghrelin and increase in PP in response to AE is consistent with previous research (Broom et al. 2009; Balaguera-Cortes et al. 2011). However, Balaguera-Cortes et al. (2011) also noted reduced ghrelin and elevated PP concentration in response to resistance exercise, likely due to the type of resistance exercise utilised. More specifically, Balaguera-Cortes and colleagues (2011) utilised a whole body resistance session that was 45 min in duration, while the present study used an isolated leg extension exercise for a duration of 30 min. While the resistance protocol used here is not necessarily reflective of common practise, it was intended to provide a more consistent stimulus throughout the session and keep the exercise restricted to the lower limbs to match the aerobic exercise stimulus. Hence, future research is needed to determine how varying the resistance training session itself may alter the subsequent metabolic responses. Regarding the other appetite-related peptides measured in the present study, we saw no change in PYY_{total}, leptin or GLP-1 during or following exercise compared with control. However, all modes of exercise increased GIP and glucagon during recovery, with the increase in glucagon being greater in AE followed by CE and SE. Of importance, despite the variation in the specific response of the appetite-related factors to each mode of exercise, each of the changes were in a direction that would appear favourable for reduced appetite with exercise compared with the resting CON. For instance, a reduction in ghrelin and an increase in PP, as seen in response to AE, would be expected to reduce appetite (Cummings 2006; Batterham et al. 2003b), while previous research has indicated that GIP and glucagon have anorexigenic properties, leading to increased satiety and meal termination (Habegger et al. 2010; Kelly et al. 2009).

Interestingly, CE appeared to have its own distinct effect on appetite-related factors compared with AE and SE, despite involving a combination of both modes of exercise. More specifically, CE did not alter ghrelin or PP as was observed with AE, nor did it increase insulin or C-peptide to the extent noted in SE, though the increase in glucagon after CE was between the concentrations achieved with AE and SE. This may be related, at least in part, to an effect of the order of AE and SE within the CE condition, with the SE component always completed first. Previous human studies have shown that the order of AE and SE, performed in succession, can alter the secretion of hormones, such as testosterone and cortisol (Cadore et al. 2012). It is also possible that the lesser total volume of AE and SE incorporated into the CE session, compared with AE and SE alone, may have dampened the hormonal response. Due to the untrained state of the current cohort, it was deemed inappropriate

to combine a full-dose SE and AE condition into one trial, hence the 50% SE and AE protocol was adopted. However, based on the results of this study, it appears that the volume of CE to induce a similar change to appetite-related peptides seen with AE and SE alone may need to be increased.

Despite the above-mentioned exercise-induced changes for several appetite-related peptides in a direction that would appear favourable for reducing appetite, we observed no changes in perceived appetite compared with the resting control or between exercise modes. The lack of effect of SE on appetite is consistent with results reported by Laan et al. (2010) who reported no change in hunger in response to a 45 min protocol involving 5 different strength exercises performed by young, active men and women. Likewise, Balaguera-Cortes and colleagues (2011) found no difference in post-exercise energy intake after a 45 min session of resistance exercise compared with a resting control. Whereas, an earlier study which recruited young, active men reported decreased hunger following a 90 minute SE protocol of 10 exercises at 80% of 12RM (Broom et al. 2009). Collectively, these data suggest that the volume of SE may be critical to induce changes in perceived appetite. Meanwhile, there was no significant effect of AE on appetite in the current study. The lack of significant effect of AE on appetite contrasts with previous reports that AE transiently reduces hunger (Broom et al. 2009; Laan et al. 2010; Imbeault et al. 1997; Westerterp-Plantenga et al. 1997; Lluch et al. 2000; Pomerleau et al. 2004; Maraki et al. 2005). However, one important distinction between the current study and most previous studies in this field is a focus on inactive, overweight, middle-aged participants as opposed to the young, healthy participants recruited by others. Indeed, there is some preliminary evidence in the literature to suggest that individuals carrying excess body fat may have lower sensitivity to hormonal cues of appetite or reduced concentrations of appetite-related peptides compared to normal weight controls (Adam and Westerterp-Plantenga 2005; Druce et al. 2005, Sloth et al. 2006). Accordingly, it is possible that the nature of our participants may have blunted or diminished appetite-related peptide responses which translated to no perceived appetite changes. Future research is needed in this area to confirm the effects of aging, a lack of physical activity and carrying excess body fat on appetite-regulation and sensitivity to intrinsic appetite cues. Alternatively, it may simply be that the magnitude of the changes in the circulating concentrations of appetite-related peptides observed here were not large enough to elicit changes in perceived appetite, or were too transient in nature.

The strength of the present study is that we recruited an inactive population to compare the effects of three modes of exercise on a wide array of blood markers. However, there were several limitations which need to be acknowledged and may assist the direction for future research. First, given that many of the observed changes in appetite-related peptides were relatively small and transient in nature, future studies should employ

larger samples. Indeed, large individual variation has been noted in previous research regarding energy balance, feeding and appetite (Blundell et al. 2015), but the present study represents a first foray into this population Also, the design of the SE protocol only incorporated one strength exercise rather than a holistic body program, as we wanted to target comparable muscle groups used in AE. The limited alterations in appetite-related factors and lack of changes to perceived appetite following SE may be related to the singular exercise used and as such, future research may like to utilise strength training programs covering all the major muscle groups of the body. Furthermore, it is likely that different results would have been obtained had the exercise protocols been matched for energy expenditure,. This was not attempted in this study since the duration of the resistance-based sessions would need to almost double the duration spent in aerobic exercise to match the expected energy expenditure thereby limiting ecological validity; however, future studies should examine the interactions between exercise mode, total energy expenditure and exercise duration. Also, this study focused on appetite responses to exercise in the fasted state and varied responses may be observed postprandially. Indeed, future studies may investigate the effect of different exercise modes on ad libitum energy intake, given that a recent systematic review has indicated that alterations to perceived appetite may not necessarily reflect actual energy intake (Holt et al. 2016). Balaguera-Cortes et al. (2011) observed no differences in energy intake following SE or AE exercise, despite a favourable hormonal milieu, especially after SE. However, the energy intake responses of an untrained population may be different compared to the active, healthy participants recruited by Balaguera-Cortes et al. (2011). Finally, it should be acknowledged that measurement of the concentration of appetite-related peptides in circulation does not take into account the potential for central effects on appetite mediated by direct stimulation of sensory neurons in the GI tract, before some level of metabolism. Hence, measures of some peptides in circulation, such as the active isoforms of GLP-1 (GLP-$1_{7\text{-}36}$ and GLP-$1_{7\text{-}37}$), may not directly represent the potential for alterations in appetite (Holst 2007).

In conclusion, we have shown for the first time in an inactive overweight population that AE and SE have varied effects on the circulating concentrations of appetite-related peptides, and that the combination of AE and SE dampens these effects, with no change in ghrelin or PP as per AE alone; and no change in insulin and C-peptide as seen with SE alone. However, the exercise-induced changes in the circulating concentrations of appetite-related peptides and metabolites that were seen here do not translate into alterations in perceived appetite. As such, future research should aim to better understand the effect of aging or carrying excess body fat on appetite regulation, and the mode, volume, intensity, and duration of exercise which may best negate these effects.

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Table 1. Participant Characteristics

Measure	Data
Age (y)	48 ± 5
Body mass (kg)	93.1 ± 7.74
BMI ($kg \cdot m^{-2}$)	29.9 ± 1.9
Waist girth (cm)	98 ± 5.8
WHR	0.9 ± 0.06
Body fat (kg)	25.1 ± 5.9
Body fat (%)	26.2 ± 4.7
Fasting glucose ($mmol \cdot L^{-1}$)	5.6 ± 0.7
Glucose AUC ($mmol \cdot L^{-1} \cdot 2h$)	26.9 ± 4.6
Fasting insulin ($\mu U \cdot mL^{-1}$)	10.8 ± 2.8
Total cholesterol ($mmol \cdot L^{-1}$)	5.6 ± 0.9
HDL cholesterol ($mmol \cdot L^{-1}$)	1.4 ± 0.4
LDL cholesterol ($mmol \cdot L^{-1}$)	3.6 ± 0.8
Triglycerides ($mmol \cdot L^{-1}$)	1.4 ± 0.6
Systolic BP (mmHg)	127 ± 9
Diastolic BP (mmHg)	83 ± 11
W_{peak} (W)	284 ± 56
$\dot{V}O_{2peak}$ ($L \cdot min^{-1}$)	2.9 ± 0.7
$\dot{V}O_{2peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	31.0 ± 8.0
Leg extension^ 1RM (kg)	90 ± 12

Data are mean ± SD (n = 12). BMI, body mass index; WHR, waist-to-hip ratio; AUC, area under the curve; HDL, high-density lipoprotein; LDL, low-density lipoprotein; BP, blood pressure; $W_{peak,}$ peak graded exercise test workload; $\dot{V}O_{2peak}$, peak oxygen consumption; ^bilateral assessment; 1RM, one-repetition maximum.

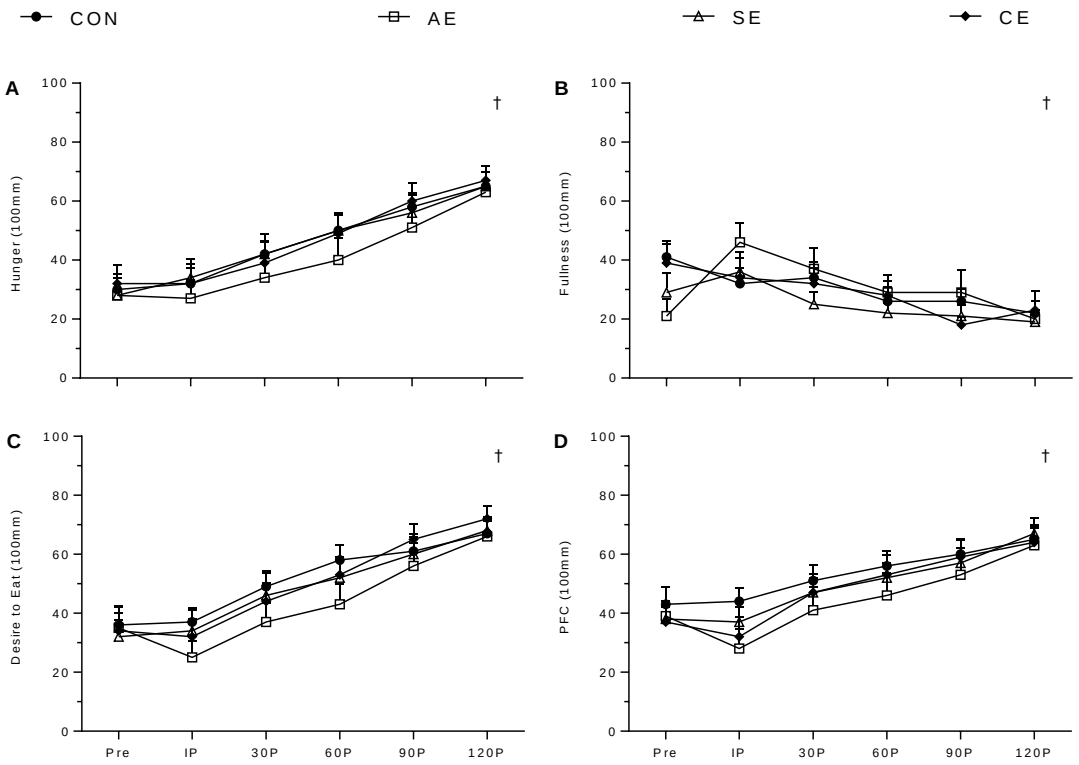
Table 2. Cardiorespiratory, estimated mean energy expenditure, lactate and perceived exertion responses during and following a non-exercise control (CON), aerobic exercise (AE), strength exercise (SE) and combined exercise (CE).

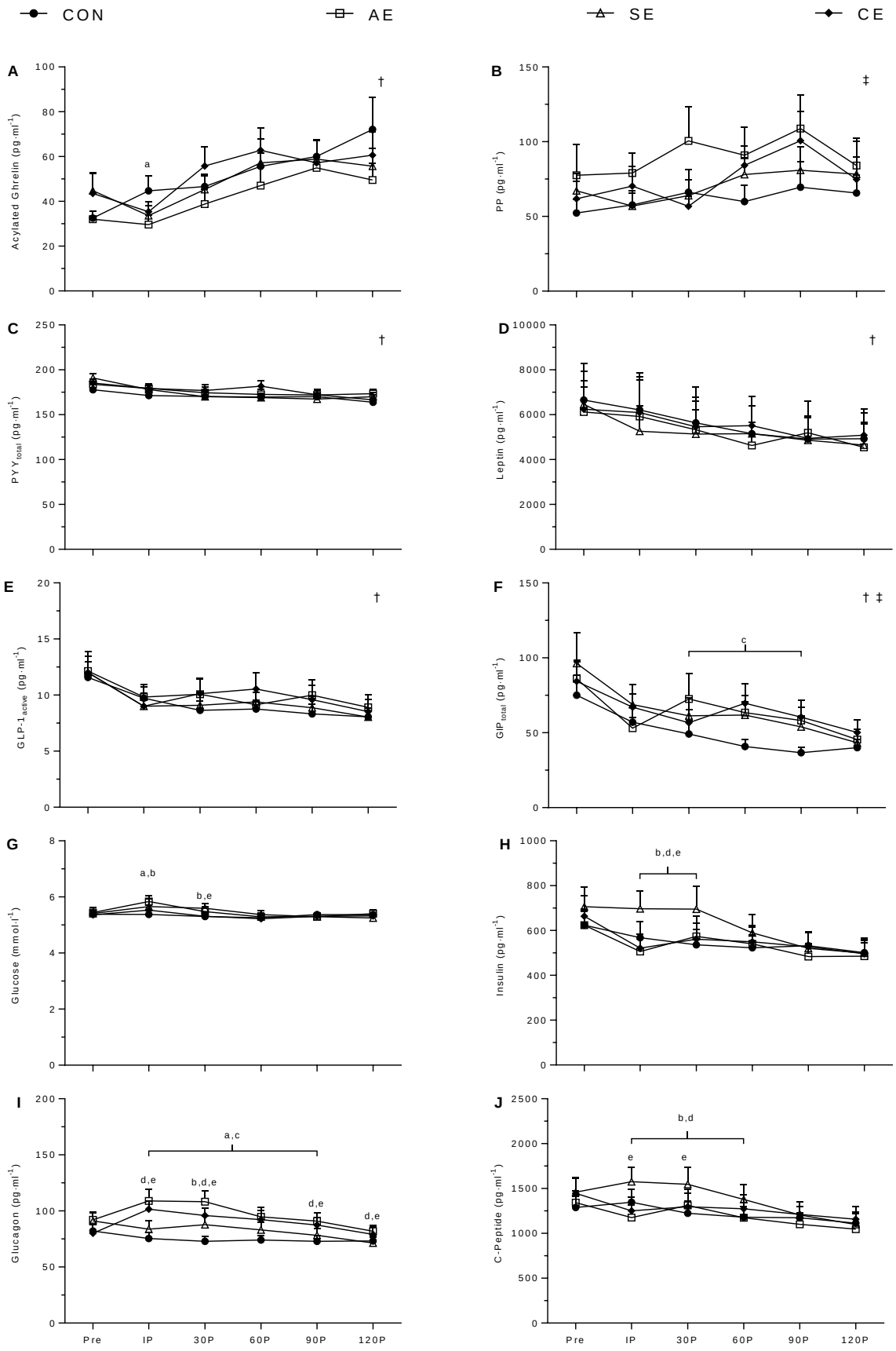
	CON	**AE**	**SE**	**CE**
Heart Rate (bpm)	67 ± 1*	140 ± 15[ac]	98 ± 17	117 ± 28
Heart rate (% maximum)	39 ± 1*	87 ± 6[ac]	54 ± 4	SE: 52 ± 4 AE: 80 ± 5
VO_2 ($L \cdot min^{-1}$)	0.31 ± 0.01*	2.26 ± 0.12[ac]	0.84 ± 0.09[b]	1.86 ± 0.68
VO_2 (% maximum)	11 ± 1*	75 ± 1[ac]	27 ± 1[b]	SE: 29 ± 1 AE: 74 ± 1
Recovery VO_2 ($L.min^{-1}$)	0.34 ± 0.01	0.36 ± 0.02	0.34 ± 0.01	0.34 ± 0.01
Pre-exercise lactate ($mmol \cdot l^{-1}$)	1.1 ± 0.4	1.2 ± 0.4	1.4 ± 0.8	1.2 ± 0.4
Post-exercise lactate ($mmol \cdot l^{-1}$)	1.3 ± 1.0*	4.7 ± 1.7	7.3 ± 3.8	4.5 ± 1.6
RPE (AU)	0.0 ± 0.1*	5.3 ± 1.3	4.9 ± 1.2	4.5 ± 1.1

*Indicates significance between CON and all exercise conditions. The following symbols indicate significance between exercise conditions [a]AE and SE, [b]SE and CE, [c]AE and CE ($p < 0.05$).

Fig. 1 Perceived hunger (A), fullness (B), desire to eat (C), and prospective food consumption (PFC) (D) in the fasted state and following a no-exercise control (CON; •), aerobic exercise (AE; □), strength exercise (SE; Δ) and combined exercise (CE; ♦) condition. † Indicates a main effect of time following all conditions ($p < 0.05$).

Fig. 2 Plasma concentrations of (A) acylated ghrelin, (B) pancreatic polypeptide (PP), (C) peptide tyrosine-tyrosine (PYY_{total}), (D) leptin, (E) glucagon-like peptide-1 ($GLP\text{-}1_{active}$), (F) glucose-dependent insulinotropic peptide (GIP_{total}), (G) glucose, (H) insulin, (I) glucagon, and (J) C-peptide following a no-exercise control (CON; •), aerobic exercise (AE; □), strength exercise (SE; Δ) and combined exercise (CE; ♦) condition. The following symbols indicate significance between conditions: [a]CON and AE, [b]CON and SE, [c]CON and CE, [d]AE and SE, [e]SE and CE ($p < 0.05$). † Indicates a main effect of time following all conditions ($p < 0.05$). ‡ Indicates a main effect of condition ($p < 0.05$)


CON
AE
SE
CE
A
B
C
D
Hunger (100mm)
Fullness (100mm)
Desire to Eat (100mm)
PFC (100mm)
†
0
20
40
60
80
100
Pre
IP
30P
60P
90P
120P


CON
AE
SE
CE
A
Acylated Ghrelin (pg·ml-1)
a
†
B
PP (pg·ml-1)
‡
C
PYYtotal (pg·ml-1)
†
D
Leptin (pg·ml-1)
†
E
GLP-1active (pg·ml-1)
†
F
GIPtotal (pg·ml-1)
c
† ‡
G
Glucose (mmol·l-1)
a,b
b,e
H
Insulin (pg·ml-1)
b,d,e
I
Glucagon (pg·ml-1)
a,c
d,e
b,d,e
d,e
d,e
J
C-Peptide (pg·ml-1)
b,d
e
e
Pre
IP
30P
60P
90P
120P