Title: Perceived appetite and appetite-related hormones following 12 weeks of aerobic or resistance exercise training in previously sedentary overweight and obese men

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Abstract

Objective: To investigate the effect of 12 weeks of aerobic (AER) compared with resistance training (RES) on perceived hunger and fullness, together with appetite-related hormones in both the fasted state and postprandially. **Methods:** Thirty-three inactive, overweight and obese men (age 49 ± 7 yr; BMI 30.8 ± 4.2 kg/m^2) were allocated to either AER exercise (n=12), RES exercise (n=13) or a control group (CON; n=8). AER and RES completed 12 weeks of training (3 sessions per week), while CON continued their sedentary routine. Perceived hunger and fullness, together with appetite-related hormones (active ghrelin, leptin, insulin, pancreatic polypeptide (PP), and peptide tyrosine tyrosine (PYY)) were assessed pre and post-intervention in the fasted state and in response to oral glucose consumption (1284 kJ; 75 g carbohydrate). **Results:** Both AER and RES training elicited a decrease in fat mass ($p<0.05$), while CON did not. There was no difference in perceived hunger in either the fasted state ($p>0.05$) or in response to caloric consumption ($p>0.05$) following the intervention in any group. In contrast, both fasting and postprandial perceived fullness was higher following AER exercise ($p<0.05$), but not RES exercise or CON. These observations were not associated with alterations in fasting or postprandial active ghrelin, PP or PYY, although fasting and postprandial leptin was reduced following both AER and RES training ($p<0.05$). **Conclusion:** Aerobic exercise training is associated with an increase in satiety, while an equivalent period resistance training does not.

Key Words: hunger, satiety, ghrelin, pancreatic polypeptide, leptin

List of Abbreviations

AER	Aerobic exercise
ANOVA	Analysis of variance
API	Aerobic power index
AUC	Area under the curve
BMI	Body mass index
CON	Control
DEXA	Dual Energy X-ray Absorptiometry
HR_{max}	Heart rate maximum
PP	Pancreatic polypeptide
PYY	Peptide tyrosine tyrosine
RES	Resistance exercise
RM	Repetition maximum
SD	Standard deviation
VAS	Visual analogue scale
VO_2	Volume of oxygen consumption
W	Watts

Introduction

Safe levels of weight loss can be achieved by reducing energy intake from food and drink, increasing energy expenditure through physical activity, or a combination of these two approaches [1,2]. It is important to note that although exercise is beneficial for increasing energy expenditure, exercise has also been shown to influence appetite and overall energy consumption [3,4]. For example, it has been demonstrated that 6 weeks of regular aerobic exercise training improves appetite regulation in previously inactive men and women by promoting more sensitive eating behaviour in response to previous energy intake (i.e. down-regulation of energy intake after a high energy preload compared with a low energy preload) [5]. These authors suggested a potential role for the gastrointestinal hormones involved in the regulation of appetite (i.e. ghrelin, pancreatic polypeptide, peptide tyrosine tyrosine) in mediating the observed changes in appetite control; however, there is limited research on the response of these hormones to exercise training [4].

Martins and colleagues [6] were the first to examine the effect of aerobic exercise training on fasting and postprandial plasma levels of appetite-related orexigenic and anorexigenic peptides, in combination with subjective sensations of appetite in sedentary overweight individuals. In this study, 12 weeks of supervised aerobic exercise training (500 kcal of treadmill walking or running 5 days per week) was associated with increased fasting acylated ghrelin and subjective feelings of hunger, but also tended to increase postprandial release of glucagon like peptide-1 (GLP-1). Based on these observations, the authors concluded that aerobic exercise training-induced weight loss may increase the drive to eat in the fasted state, but this is balanced with an improved satiety response to meal consumption. Of interest, it is likely that the effect of exercise training on appetite regulation is influenced by the specific mode of training employed. In particular, whether similar findings would be noted with

regular resistance training is not known. This is important given that resistance exercise is an integral component of a holistic exercise program for weight control due to its role in increasing fat-free mass [7], as well as general health and well-being [8].

Although no studies have investigated the effect of regular resistance training on appetite regulation, there is evidence to suggest that acute sensations of appetite and appetite-related hormones respond differently to a single bout of resistance compared with aerobic exercise [9,10]. Balaguera-Cortes and co-workers [9] observed a transient hormonal milieu that would appear favourable for reduced energy intake in response to an acute bout of resistance exercise compared with aerobic exercise. Specifically, reduced active ghrelin and elevated pancreatic polypeptide (PP) and insulin were observed following resistance exercise compared with a resting control trial, while an aerobic exercise session of matched duration resulted in elevated PP, but reduced insulin. It is not known whether such alterations in the acute response to exercise translate to differences in longer-term appetite regulation. Consequently, the purpose of the present study was to investigate the effect of 12 weeks of aerobic or resistance training on perceived appetite, together with a range of appetite-related hormones in both the fasted state, and in response to caloric consumption, in previously sedentary overweight and obese men. It was hypothesised that both aerobic and resistance training would improve the satiety response to caloric consumption compared to a no exercise control group, and that this would be related to increased postprandial concentrations of PP.

Methods

Participants

Thirty-three previously inactive (no regular pattern of planned or incidental exercise >1d·wk^{-1} in the preceding 12 months), overweight and obese men volunteered for the study (mean ± SD age 49 ± 7 yr; body mass index 30.8 ± 4.2 kg/m^2). Overweight and obese participants (rather than lean individuals) were recruited since the effect of regular exercise training on appetite regulation may be of most relevance to this population. Participants obtained medical clearance and were not dieting to lose weight for at least 3 months prior to the study. To minimise the potential for biased responses, the participants were not informed that appetite-regulation was being assessed. Instead, they were informed that the blood glucose and insulin response to a glucose drink would be monitored to determine their risk of diabetes. Following completion of the study, all participants were personally debriefed, with the true aims of the study revealed and explained in full. The study was approved by the Charles Sturt University Research Human Ethics Committee and written and verbal consent was attained from all participants.

Experimental design

Participants were allocated to either an aerobic-based exercise training program (AER; n = 12), a resistance-based training program (RES; n = 13) or a control group (CON; n = 8). Those allocated to the AER and RES training completed 3 sessions of supervised exercise each week (with at least one day of rest separating consecutive sessions) for a total of 12 weeks. The CON group continued with their normal sedentary routine. Outcome measures were assessed at baseline (before the commencement of training) and after completion of the intervention period.

Exercise training

The session duration and intensity were matched between AER and RES groups and progressively increased from 40 min at the beginning of the intervention, up to 60 min by the end of the 12 week period. The AER group participated in aerobic-based activities consisting primarily of stationary cycling (Monark 828E, Monark Exercise AB, Varburg, Sweden), with elliptical cross training (Victory XE125, Spirit, China) incorporated periodically mid-session to enhance variety. Participants exercised at 70-75% of heart rate maximum (HR_{max}; Polar Electro Oy, Kempele, Finland) [11] for weeks 1-4, with exercise intensity progressed to 75-80% HR_{max} for weeks 5-12. In contrast, RES completed a range of resistance-based (weight training) exercises for each major muscle group of the body. Exercises incorporated a combination of pulley-weight machines and free-weights (chest or bench press, shoulder or military press, seated or bent-over-row, lat pulldown, leg press, leg curls, lunges, machine squats and deadlifts depending on past experience). Participants progressed from 3 sets of 10 repetitions of each exercise at 75% of 1 repetition maximum (1RM; estimated from pre-intervention 5RM strength testing as described later), to 4 sets of 8 repetitions at 85% 1RM by the end of the intervention. Each session was commenced with a 5 min warm-up on a rowing ergometer, followed by completion of the prescribed exercises in an alternating manner from upper- to lower-body. A standard 90 s of recovery was enforced between sets. A rating of perceived exertion was obtained following each session for both AER and RES groups using the category-ratio (CR0-10) Borg scale [12]. Of note, it was not the intention of this study to match the energy expenditure of AER and RES exercise. Instead, this study aimed for ecological validity, with the duration of the exercise matched to reflect the common situation in which an individual has a set time available to exercise, in order to determine the expected responses depending on whether an individual chooses to perform aerobic versus resistance exercise within this set timeframe.

Outcome measures

Outcome measures were assessed during two separate testing sessions conducted both pre- and post-intervention. The initial session was conducted ~1 week prior to the commencement of the training intervention and involved the assessment of perceived hunger and fullness, together with appetite-related hormones, in both the fasted state and in response to caloric consumption. The second session was conducted ~3 days later and involved the assessment of body composition using Dual Energy X-Ray Absorptiometry (DEXA), as well as the assessment of cardiorespiratory fitness and muscular strength. The same outcome measures were assessed post-intervention, with participants abstaining from exercise for 3 days before completing the post-intervention assessment of appetite and appetite-related hormones to minimise any potential acute effects from the last training session.

Session A - Assessment of appetite

In the 3 days leading up to the assessment of appetite, each participant was required to avoid vigorous physical activity and to document all food and drink intake in a diary. This dietary information was overviewed by the research team upon arrival to the laboratory, and participants were required to replicate their food and drink intake (standardisation within each participant) in the 3 days leading up to the post-intervention assessment of appetite. A reminder to replicate dietary intake was given the week prior to post-intervention testing and compliance was confirmed verbally and following inspection of the records by the investigators upon arrival to the laboratory. On the day of testing, each participant arrived at the laboratory at 0600-0800 h (with the time matched within participants) following a 10 h overnight fast and was required to rate their perceived levels of hunger and fullness, respectively, using a validated 100-mm visual analogue scale (VAS) [13]. Following this, a

cannula was inserted into an antecubital vein to allow for venous blood to be sampled for the determination of the fasting levels of a range of appetite-related hormones (insulin, leptin, active ghrelin, pancreatic polypeptide and peptide YY). Participants were then provided with a standardised caloric stimulus (Carbotest; 1284 kJ; 75 g carbohydrate in 300 mL; Lomb Scientific, Australia) to consume within 5 min. Liquid calories were preferred to a solid meal in order to remove any possible influence of perception of food intake or the act of mastication itself on the observed responses [14]. The use of an oral glucose load as the caloric stimulus was intended to assist with the deception of the participants about the true aims of the study. At regular time intervals following ingestion (30, 60, 90 and 120 min post-consumption), participants were required to rate their perceived hunger and fullness using the VAS, while further blood samples were obtained to monitor the acute hormonal response. For the VAS, only the questions for each individual time-point could be viewed at any one time, to ensure that participants could not refer to their previous ratings [13].

Session B - Assessment of body composition, cardiorespiratory fitness and muscular strength

Body composition was assessed pre- and post-intervention using DEXA, with participants scanned in light clothing (Norland XR800, Cooper Surgical Company, Turnbull, CT, USA). Scan images were analysed for total body mass, fat mass and lean body mass (Illuminatus DXA, version 4.2.0, Turnbull, CT, USA). Cardiorespiratory fitness was assessed using a submaximal graded exercise test which was performed on an electronically-braked cycle ergometer (LODE Excalibur Sport, LODE BV, Groningen, The Netherlands). The test commenced at a power output of 25 W, with increments of 25 $W \cdot min^{-1}$ until 80% of HR_{max} (estimated using the equations of Inbar and colleagues [11]) was achieved. During the test, heart rate was recorded each min (Vantage NV, Polar, Finland) and oxygen consumption was measured using a calibrated metabolic gas analysis system (TrueOne 2400 metabolic

measurement system; Parvomedics; Sandy, Utah, USA). From this, the workload at which 80% HR_{max} was achieved was used to calculate an aerobic power index (API), which is the principal outcome measure of the tri-level fitness profile, and a highly reliable sub-maximal exercise protocol in sedentary participants [15,16]. Muscular strength was assessed using 5RM testing for chest press (for the upper body) and leg press (for the lower body). This protocol was preferred to 1RM testing due to the sedentary overweight nature of the participants and to minimise muscular soreness.

Blood analysis

Venous blood was aliquotted into fluoride oxalate tubes for measurement of glucose, into lithium heparin tubes for measurement of insulin, and treated with EDTA and serine protease inhibitor (Pefabloc SC, Roche Diagnostics, NSW, Australia) for measurement of active ghrelin, leptin, PP, and peptide tyrosine tyrosine (PYY). All samples were then centrifuged at 1000 *g* for 15 min at 4°C with the plasma obtained stored at -80°C until later analysis. Active ghrelin, leptin, PP, and PYY were measured in duplicate using a commercially available assay kit (Milliplex Human Gut Hormone Panel, Millipore Corporation, Billerica, MA, USA) according to the manufacturer's instructions on a Luminex 200 system (Luminex Corporation, Austin, TX, USA). Fluorescence data were analysed using Luminex xPONENT software (Luminex Corporation, Austin, TX, USA). Glucose was assayed using an enzymatic method and bi-chromatic endpoint technique measurement (Dimension Xpand Plus, Siemens Healthcare Diagnostics, Deerfield, IL). Insulin was measured using a solid-phase, two-site chemiluminescent immunometric assay (IMMULITE 2000 analyzer, Diagnostic Products Corporation, Los Angeles, CA, USA).

Data analysis

One-way ANOVA was used to compare baseline (pre-intervention) age, body mass and cardiorespiratory fitness between the three groups. One-way ANOVA were used to compare baseline (pre-intervention) age, body mass and cardiorespiratory fitness between the three groups. One way repeated measures ANOVA (pre to post intervention) were used to compare the effect of each intervention on body composition, cardiorespiratory fitness, muscular strength, fasting appetite and appetite-related hormones. Two-way repeated measures ANOVA (pre to post intervention x 5 time points of caloric loading) were used to assess the effect of each intervention on the responses of perceived appetite and appetite-related hormones to caloric consumption. Post-hoc paired samples t-tests were used to determine where any differences lay pre- to post-intervention within each group. Area under the curve (AUC) was calculated using the trapezoidal rule for both the hormonal and perceptual responses to caloric consumption, with one-way repeated measures ANOVA used to compare pre and post-intervention within each group. Insulin sensitivity was calculated using the method of Matsuda and DeFronzo [17]. Statistical significance was accepted as $p \leq 0.05$ (SPSS 18.0 for windows computer software package, Chicago, USA). All data are reported as mean ± SD unless otherwise indicated.

Results

Training compliance, cardiorespiratory fitness and muscular strength

All participants attended and completed no fewer than 30 of the 36 supervised training sessions. Mean session attendance and completion rates were 33 of 36 (92 ± 7%) for the AER group, and 33 of 36 (92 ± 6%) for the RES group ($p = 0.755$). There was no difference in mean ratings of perceived exertion between the exercise groups with the AER group (4 ± 1) rating the training similar to the RES group (4 ± 1; equivalent to “somewhat hard”) ($p = 0.228$). Following the intervention period, cardiorespiratory fitness (based on the API) was

improved in the AER group ($p < 0.001$), but remained unchanged in the RES ($p = 0.226$) and CON groups ($p = 0.81$; Table 1). Likewise, the rate of oxygen consumption at 80% HR_{max} was significantly higher post-intervention in the AER group ($p < 0.001$), while there was no change following the intervention period in RES or CON groups ($p > 0.05$). Lower body strength (based on leg press 5RM) improved in both AER ($p < 0.001$) and RES groups ($p < 0.001$), but the improvement was greater in RES, while there was no change in the CON group ($p = 0.567$; Table 1). Upper body strength (based on chest press 5RM) improved in the RES group only ($p < 0.001$).

Body composition

There was no difference in age ($p = 0.114$), total body mass ($p = 0.478$), or BMI ($p = 0.605$) at baseline between groups. Following the intervention period, there was a significant decline in body mass, BMI and fat mass ($p < 0.05$), but not lean body mass ($p = 0.432$) in the AER group (Table 2). In contrast, RES training did not alter total body mass or BMI, but resulted in increased lean body mass and reduced fat mass ($p < 0.05$). For the CON group, there was no change in body mass, BMI, or fat mass, while lean body mass declined slightly following the intervention period ($p = 0.034$).

Perceived hunger

There was no change in perceived hunger in the fasted state pre- to post-intervention in any group (Figure 1A; $p > 0.05$). Likewise, there was no change in perceived hunger in response to caloric consumption (Figure 1A; $p > 0.05$). This was further supported by similar AUC results for perceived hunger following caloric consumption pre- to post-intervention in all groups ($p > 0.05$; Table 3).

Perceived fullness

There was a significant increase in perceived fullness in the fasted state following the AER training intervention ($p = 0.001$), but no change in the CON ($p = 0.551$) or RES groups ($p = 0.113$; Figure 1B). Likewise, in response to caloric consumption, perceived fullness was higher following the AER training intervention ($p = 0.006$), while there was no change in the CON or RES groups ($p > 0.05$). AUC for perceived fullness in response to caloric consumption followed a similar temporal pattern with an increase post-intervention in the AER group ($p = 0.013$), but no difference pre to post-intervention in CON or RES groups (Table 3).

Hormone responses

There was no change in fasting concentrations of active ghrelin, PP, PYY or insulin in any group post-intervention (Figure 2; $p > 0.05$). With respect to leptin, there was no change in fasting concentrations as a result of the intervention period in CON ($p = 0.584$), while concentrations decreased following both AER ($p = 0.001$) and RES training ($p = 0.028$). On the other hand, fasting glucose was higher in the CON group post-intervention ($p = 0.009$), but remained unchanged in AER and RES groups ($p > 0.05$).

There was no change in the response of active ghrelin, PP or PYY to caloric consumption pre- to post-intervention (Figure 2; $p > 0.05$), although there was a main effect for time for all groups with a decrease in active ghrelin and increased PP and PYY in response to acute caloric consumption ($p < 0.05$). With respect to circulating leptin, there was no change in the response to caloric consumption pre- to post-intervention in CON ($p > 0.05$), but lower concentrations were observed post-intervention in the AER ($p = 0.005$) and RES groups ($p = 0.002$). This observation was supported by lower AUC for leptin following caloric

consumption in AER ($p = 0.005$) and RES ($p = 0.002$) post-intervention, but not in the CON group ($p = 0.543$; Table 3). The response of insulin and glucose to caloric consumption, together with insulin sensitivity, was not altered pre- to post-intervention in CON ($p > 0.05$). Following AER training, the response of glucose and insulin to caloric consumption was attenuated compared with pre-training ($p < 0.05$; Figure 2; Table 3). For the RES group, there was no difference in glucose pre- to post-intervention, while there was a tendency for lower insulin levels following the training period based on AUC ($p = 0.066$). Estimated insulin sensitivity improved in response to both AER and RES training ($p < 0.05$).

Discussion

We have found that 12 weeks of resistance training does not influence perceived hunger or fullness in either the fasted state or in response to a glucose caloric stimulus. In contrast, an equivalent period of aerobic exercise training increased both fasting and postprandial fullness, but did not affect perceived hunger. The role of appetite-related hormones in mediating this response is not clear. Neither AER or RES training significantly altered the fasting or postprandial levels of active ghrelin, PP or PYY, although leptin was reduced following both AER and RES training, likely in association with the fat loss observed in these two groups. The postprandial concentrations of both glucose and insulin were attenuated following AER training, while for the RES group there was only a tendency for lower postprandial insulin. Regardless, the finding that aerobic exercise training is associated with an increase in satiety, while an equivalent period resistance training is not may have important implications for exercise prescription for the overweight individual.

This is the first study to examine the responses of perceived appetite and appetite-related hormones to a period of regular resistance training. In contrast, 12 weeks of supervised

aerobic exercise training has previously been shown to result in increased fasting acylated ghrelin and perceived hunger, but also tended to increase postprandial GLP-1 [6]. These researchers suggested that the latter hormonal change was evidence for improved satiation in response to meal ingestion following aerobic training. Our study provides further support for this notion, with increased feelings of fullness reported following the AER intervention in both the fasted state and in response to a caloric stimulus. However, unlike Martins and colleagues [6] we did not observe a concurrent increase in perceived hunger or active ghrelin following AER training. Possible reasons for this discrepancy between findings may be related to differences in the precise training protocol (5 days versus 3 days per week in the present study), as well as the degree of fat loss resulting from training (2.9 kg versus 1.4 kg in the present study). The inclusion of women in the study of Martins and colleagues may also explain their observation of increased hunger given that the initiation of exercise training has been shown to stimulate appetite-related hormones in a direction expected to increase energy intake (i.e. higher acylated ghrelin) in women, but not men [18]. Regardless, both studies support a role for aerobic exercise training in improving satiety, although it is important to note that it is not possible to determine whether these findings can be attributed to the exercise training alone, or to exercise-induced weight loss. This could only be determined in a study whereby exercise training was performed in energy balance (with the energy cost of each exercise session matched by an equivalent increase in energy intake), compared with exercise training performed without a concomitant increase in energy intake, rather than the free-living conditions of the present study.

The mechanism through which aerobic exercise-induced weight loss stimulates satiety is unclear. Martins and colleagues [6] suggested that aerobic exercise training may benefit satiety based on their observation of a tendency for increased late postprandial release of

GLP-1. The hormones measured in the present study were based on the previous comparison of the acute effects of a one-off bout of aerobic and resistance exercise on appetite-related hormones [9]. We observed an attenuated response of glucose and insulin to caloric consumption, and improved insulin sensitivity, following AER training. It is possible that these changes may have contributed to the increase in postprandial satiety observed following AER training given that insulin sensitivity has been shown to be negatively correlated with ad libitum energy intake in an overweight population [19]. However, it is important to note that insulin sensitivity also improved in RES, although to a lesser degree (non-significant). Furthermore, it is difficult to attribute the change in satiety following AER training to the associated reduction in leptin, since reduced leptin was also observed following RES training, albeit again to a lesser degree (non-significant). Likewise, PP and PYY do not appear to play a role in mediating these changes in satiety following AER training. Whether GLP-1 or other peptides such as cholecystokinin played a specific role in the present findings is not known.

Other potential mechanisms through which aerobic exercise may stimulate satiety which were not investigated in the present study include an enhanced rate of gastric emptying [20], allowing for faster release of satiety signals [21]. Others have hypothesised that training-induced changes in substrate metabolism (i.e. increased fatty acid oxidation) may play an important role in appetite regulation by influencing hepatic energy status [22,23]. It is also possible that myokines may play a role in mediating the effect of exercise training on appetite-regulation. The transient rise in IL-6 observed in response to an acute bout of exercise has been reported to suppress post-exercise energy intake [24]. It is possible that repeated episodic signalling following acute exercise sessions may influence long-term regulation of appetite. Clearly, the mechanisms through which exercise training affects appetite represents a major gap in the research and further study is required in this area.

As outlined in international guidelines on exercise prescription, the holistic exercise program should include both aerobic and resistance training for optimal health and fitness benefits [7,25]. Our findings support this notion, given the distinct benefits of AER exercise for improving cardiorespiratory fitness, while RES training was more effective in improving muscular strength. With respect to weight management, aerobic modes of exercise are often promoted over resistance exercise based on the higher energy expenditure per unit of time [26]. The present study may add further support for the role of aerobic exercise in assisting weight management, given the observed increase in perceived fullness in both the fasted state and postprandial following AER but not RES training. Yet it is important to acknowledge that the effect of such changes in the perception of appetite on actual energy intake remains to be determined. The participants in the present study were instructed not to alter their food intake throughout the intervention period to allow for isolation of the effect of exercise on outcome variables. The use of self-reported food diaries was not enforced during the intervention period to confirm consistency of food intake since we did not want to bring increased attention to monitoring of energy intake as this may have altered food consumption in itself and alerted participants to the true aims of the study. It must also be noted that daily activity levels outside of the intervention were not monitored throughout the 12 week intervention period. Given the findings of the present study, future research should compare the effect of aerobic and resistance exercise training on ad libitum energy intake or energy intake following pre-load consumption [5].

The present study did not attempt to match the energy expenditure of AER and RES exercise. For ecological validity, the duration of the exercise was matched to reflect the common real-life situation in which an individual has a set time available to exercise. The alternative of

attempting to match the energy expenditure of AER and RES exercise was not preferred since this would likely increase the duration of the resistance-based sessions to almost double the duration of the aerobic sessions [9]. Employing such a design would have limited practical application since most individuals would not spend up to 2 h completing such a resistance training session. Finally, the caloric stimulus used in the present study consisted of a liquid glucose drink, rather than a mixed solid meal. Although this would not influence the fasting measures of appetite and hormone concentrations, the use of liquid calories has been shown to result in higher ratings of postprandial hunger compared with a solid meal of similar energy content [27] and does not reflect typical daily caloric consumption. The liquid mode of energy consumption was preferred in the present study to remove any possible influence of perception of food intake or the act of mastication itself on the observed responses [14]; i.e. we wanted perception of appetite to reflect the physiological signals within the body, not learned psychological responses to food intake. In support of the use of a liquid caloric load, the hormonal responses observed following ingestion clearly show that the drink was successful in triggering the intended physiological response, with a postprandial reduction in active ghrelin and increased PP, PYY, glucose and insulin. However, it is interesting to note a lack of sensitivity to these internal physiological cues, with a failure of feelings of hunger to decrease 30 min following consumption of the caloric load in the overweight and obese men. Whether this lack of sensitivity is related to the nature of the caloric stimulus (an oral glucose drink), learned psychological responses to food intake overriding physiological feedback, or to disturbed perception of physiological signals within the body is not known. In support of the latter, obese individuals have been shown to have lower sensitivity to hormonal cues of appetite in response to a standard nutrient challenge compared with normal weight participants, suggesting a disturbance in appropriate perception of physiological cues [28]. Furthermore, overweight individuals tend to automatically direct their attention to food-

related stimuli to a greater extent than normal-weight individuals, particularly when food-deprived [29]. This lack of sensitivity to internal cues was not improved by 12 weeks of exercise training in the present study. Future studies should seek to examine the effect of other modes of caloric consumption that more closely reflect typical intake (i.e. a mixed solid meal) on feelings of appetite in overweight and obese individuals to further examine this issue.

In summary, 12 weeks of regular aerobic exercise training stimulates satiety in both the fasting state and for up to 2 hours postprandial, while an equivalent period of resistance training does not. In contrast, neither mode of exercise appears to influence perceived hunger in either the fasted state or in response to consumption of an oral glucose caloric stimulus. These findings may have implications for weight control, although the role of appetite-related hormones in these changes is unclear and requires further investigation. Regardless, aerobic modes of exercise may be promoted over resistance exercise for weight control based on the higher energy expenditure per unit of time, together with the increase in satiety observed in the present study.

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Disclosure statement

No conflicts of interest to declare.

Author contributions

KJG contributed to developing the experimental design, conducted the pre- and post-intervention assessment of appetite measures, assay of appetite-related hormones, analysis and interpretation of the data collected, and drafting the manuscript. CED contributed to developing the experimental design, coordinated the pre- and post-intervention testing of outcome measures, conducted the exercise training, and contributed to the interpretation of results and drafting of the manuscript. RD contributed to developing the experimental design, pre- and post-intervention assessment of outcome measures, interpretation of results and drafting of the manuscript.

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Table 1. Cardiorespiratory fitness (based on the Aerobic Power index) and muscular strength (based 5 repetition maximum; 5RM) before and after 12 weeks of aerobic-based exercise training (AER; n = 12), resistance-based exercise training (RES; n = 13) or a no exercise control (CON; n = 8).

		CON	AER	RES
Cardiorespiratory fitness ($W \cdot kg^{-1}$ @ 80% HR_{max})	Pre	1.63 ± 0.59	1.86 ± 0.29	1.63 ± 0.37
	Post	1.56 ± 0.86	2.34 ± 0.38*	1.75 ± 0.43
Cardiorespiratory fitness (VO_2 $L.min^{-1}$ @ 80% HR_{max})	Pre	2.07 ± 0.56	2.25 ± 0.51	1.94 ± 0.39
	Post	2.06 ± 0.55	2.82 ± 0.60*	2.17 ± 0.54
Lower body strength (5RM leg press kg)	Pre	190 ± 37	145 ± 48	130 ± 32
	Post	183 ± 46	180 ± 54*	259 ± 54*
Upper body strength (5RM chest press kg)	Pre	62 ± 15	65 ± 12	54 ± 13
	Post	64 ± 21	71 ± 12	87 ± 13*

* Indicates significant difference from pre-intervention values ($p \leq 0.05$).

Table 2. Body composition before and after 12 weeks of aerobic-based exercise training (AER; n = 12), resistance-based exercise training (RES; n = 13) or a no exercise control (CON; n = 8).

		CON	AER	RES
Body Mass (kg)	Pre	93.9 ± 20.4	102.0 ± 12.6	98.4 ± 12.1
	Post	94.0 ± 21.3	100.0 ± 11.9*	98.6 ± 12.5
Body Mass index (kg/m^2)	Pre	30.1 ± 6.1	31.7 ± 3.5	30.3 ± 3.5
	Post	30.1 ± 6.3	31.1 ± 3.3*	30.3 ± 3.7
Fat Mass (kg)	Pre	23.2 ± 10.8	28.0 ± 6.7	27.5 ± 7.3
	Post	23.9 ± 11.5	26.6 ± 6.2*	26.8 ± 7.3*
Lean Body Mass (kg)	Pre	67.4 ± 10.4	70.5 ± 7.7	67.5 ± 6.6
	Post	66.9 ± 10.5*	70.1 ± 7.5	68.5 ± 6.9*

* Indicates significant difference from pre-intervention values ($p \leq 0.05$).

Table 3. Area under the curve for perceived hunger and fullness, as well as a range of appetite-related hormones before and after 12 weeks of aerobic-based exercise training (AER; n = 12), resistance-based exercise training (RES; n = 13) or a no-exercise control (CON; n = 8).

		CON	AER	RES
Perceived Hunger	Pre	94 ± 32	77 ± 42	73 ± 34
(mm over 2 h)	Post	87 ± 36	66 ± 40	83 ± 32
Perceived Fullness	Pre	67 ± 31	45 ± 25	73 ± 30
(mm over 2 h)	Post	72 ± 20	59 ± 28*	88 ± 34
Active Ghrelin	Pre	190 ± 178	105 ± 54	136 ± 66
(pg/ml over 2 h)	Post	207 ± 169	126 ± 100	124 ± 63
PP	Pre	206 ± 147	358 ± 314	384 ± 253
(pg/ml over 2 h)	Post	200 ± 137	322 ± 314	285 ± 123
PYY	Pre	188 ± 56	162 ± 60	140 ± 40
(pg/ml over 2 h)	Post	192 ± 59	156 ± 59	141 ± 30
Leptin	Pre	22987 ± 23538	19663 ± 7795	20013 ± 10483
(pg/ml over 2 h)	Post	21142 ± 18071	15439 ± 7331*	16616 ± 8618*
Insulin	Pre	110 ± 63	187 ± 85	155 ± 106
(μIU/ml over 2 h)	Post	124 ± 80	106 ± 49*	122 ± 73
Glucose	Pre	14.6 ± 2.7	14.9 ± 3.0	14.6 ± 3.4
(mmol/l over 2 h)	Post	14.9 ± 4.7	13.0 ± 2.2*	13.8 ± 2.5

* Indicates significant difference from pre-intervention values ($p \leq 0.05$).

Figure Captions

Figure 1. Perceived hunger (A) and fullness (B) in the fasted state (0 min) and in response to caloric consumption (represented by ↓) before (●) and after (○) 12 weeks of aerobic-based exercise training (AER; n = 12), resistance-based exercise training (RES; n = 13) or a no-exercise control (CON; n = 8; mean ± SEM). *Indicates significant difference from pre-intervention values in the fasted state (i.e. at 0 min; $p \leq 0.05$). †Indicates a significant main effect of the intervention on responses to caloric consumption ($p \leq 0.05$).

Figure 2. Plasma concentrations of (A) active ghrelin, (B) peptide tyrosine-tyrosine (PYY), (C) pancreatic polypeptide (PP), (D) leptin (E) glucose and (F) insulin in the fasted state (0 min) and in response to caloric consumption (represented by ↓) before (●) and after (○) 12 weeks of aerobic-based exercise training (AER; n = 12), resistance-based exercise training (RES; n = 13) or a no-exercise control (CON; n = 8; mean ± SEM). *Indicates a significant difference from pre-intervention values in the fasted state (i.e. at 0 min; $p \leq 0.05$). †Indicates a significant main effect of the intervention on responses to caloric consumption ($p \leq 0.05$). ‡Indicates a significant main effect of time in response to caloric consumption ($p \leq 0.05$).

Figure 1.

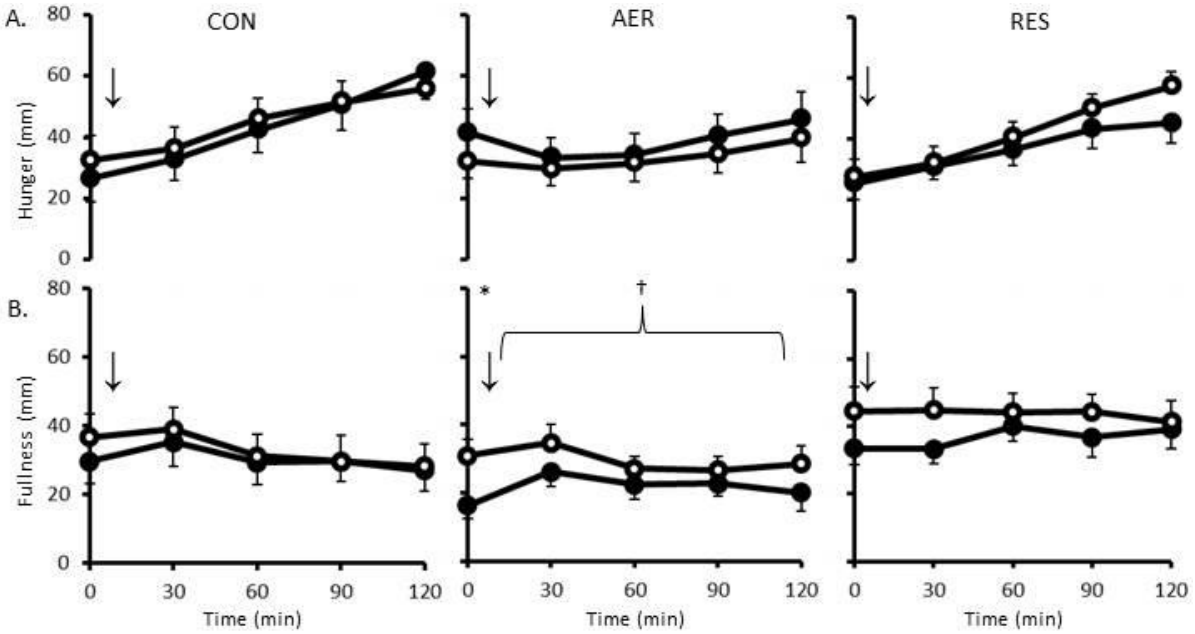
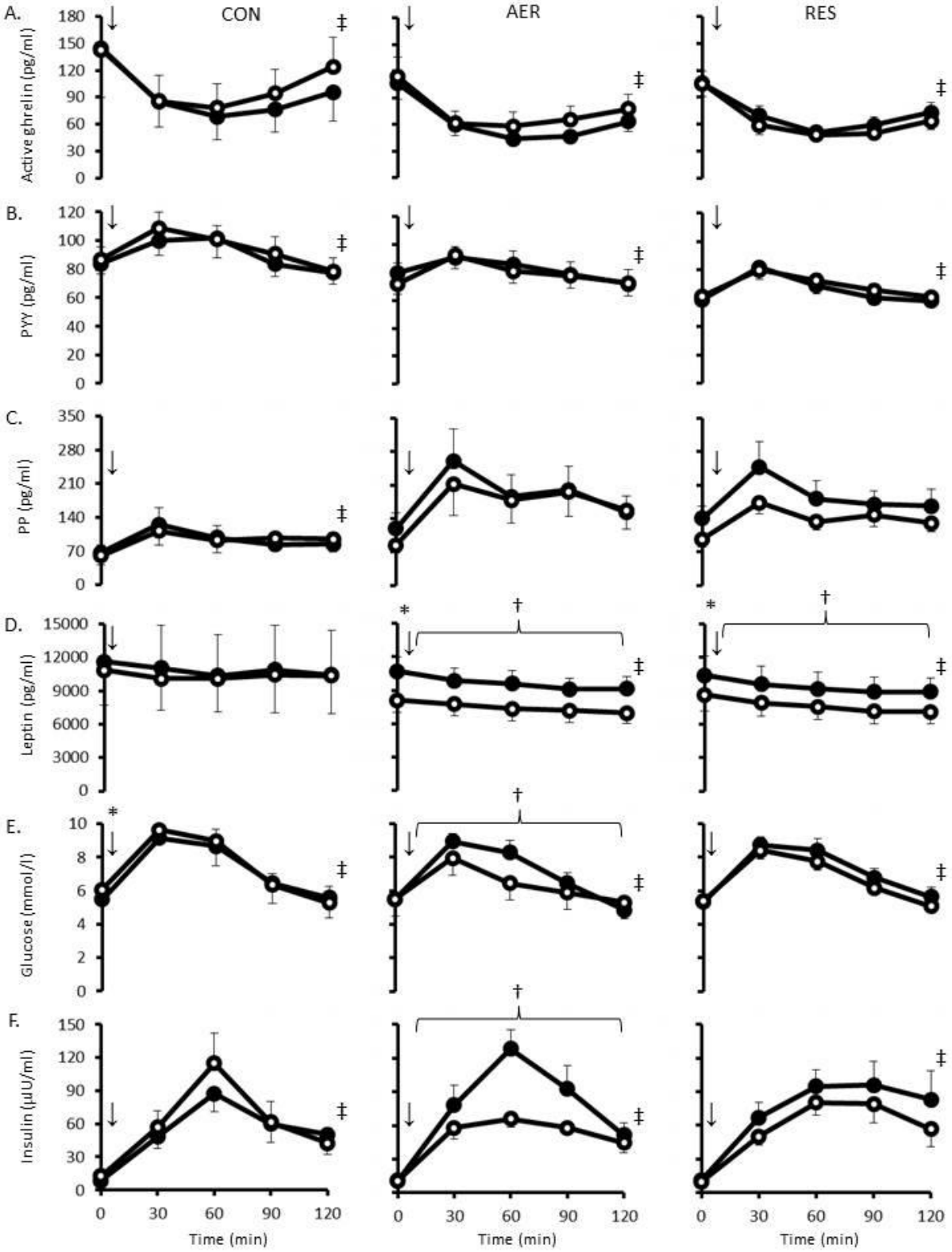

A.
CON
AER
RES
Hunger (mm)
80
60
40
20
0
B.
Fullness (mm)
*
†
0
30
60
90
120
Time (min)

Figure 2.


A.
CON
AER
RES
Active ghrelin (pg/ml)
B.
PYY (pg/ml)
C.
PP (pg/ml)
D.
Leptin (pg/ml)
E.
Glucose (mmol/l)
F.
Insulin (μU/ml)
Time (min)
Time (min)
Time (min)