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ROCHA, Joel, PAXMAN, Jenny <http://orcid.org/0000-0003-3596-489X>, DALTON, Caroline <http://orcid.org/0000-0002-1404-873X>, WINTER, Edward and BROOM, David <http://orcid.org/0000-0002-0305-937X>

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Effects of a 12-week aerobic exercise intervention on eating behaviour, food cravings and 7-day energy intake and energy expenditure in inactive men

Joel Rocha¹*, Jenny Paxman², Caroline Dalton³, Edward Winter⁴ and David Broom⁵

¹Division of Sport and Exercise Sciences, School of Social & Health Sciences, Abertay University, DD1 1HG
²Food and Nutrition Group, Sheffield Business School, Sheffield Hallam University, S1 1WB
³Biomolecular Sciences Research Centre, Faculty of Health and Wellbeing, Sheffield Hallam University, S1 1WB
⁴Centre for Sport and Exercise Sciences, Faculty of Health and Wellbeing, Sheffield Hallam University, S10 2BP
⁵Academy of Sport and Physical Activity, Faculty of Health and Wellbeing, Sheffield Hallam University, S10 2BP

*Corresponding author

Address for correspondence: Division of Sport and Exercise Sciences, School of Social & Health Sciences, Abertay University, DD1 1HG, UK

E-mail: J.Rocha@abertay.ac.uk
Telephone: +44 (0)1382 308529

E-mail addresses:

JP: J.R.Paxman@shu.ac.uk
CD: C.F.Dalton@shu.ac.uk
EW: E.M.Winter@shu.ac.uk
DB: D.R.Broom@shu.ac.uk
Abstract

This study examined effects of 12 weeks of moderate-intensity aerobic exercise on eating behaviour, food cravings and weekly energy intake and expenditure in inactive men. Eleven healthy men (mean ± SD: age, 26 ± 5 years; body mass index, 24.6 ± 3.8 kg/m²; maximum oxygen uptake, 43.1 ± 7.4 mL/kg/min) completed the 12-week supervised exercise programme. Body composition, health markers (e.g. lipid profile), eating behaviour, food cravings and weekly energy intake and expenditure were assessed before and after the exercise intervention. There were no intervention effects on weekly free-living energy intake \((p=0.326, d=-0.12)\) and expenditure \((p=0.799, d=0.04)\), or uncontrolled eating and emotional eating scores \((p>0.05)\). However, there was a trend with a medium effect size \((p=0.058, d=0.68)\) for cognitive restraint to be greater after the exercise intervention. Total food cravings \((p=0.009, d=-1.19)\) and specific cravings of high-fat foods \((p=0.023, d=-0.90)\), fast-food fats \((p=0.009, d=-0.71)\) and carbohydrates/starches \((p=0.009, d=-0.56)\) decreased from baseline to 12 weeks. Moreover, there was a trend with a large effect size for cravings of sweets \((p=0.052, d=-0.86)\) to be lower after the exercise intervention. In summary, 12 weeks of moderate-intensity aerobic exercise reduced food cravings and increased cognitive restraint, however, these were not accompanied by changes in other eating behaviours and weekly energy intake and expenditure. The results indicate the importance of exercising for health improvements even when reductions in body mass are modest.
Key words: exercise, food cravings, eating behaviour, weekly energy intake and expenditure.

Introduction

Exercise is an important strategy to reduce or maintain body mass because it can create a negative energy balance while improving several health-related outcomes (Jakicic and Otto 2006). Nevertheless, exercise interventions elicit marked individual variability in changes of body mass (Donnelly and Smith 2005). Differences in participants' adherence to exercise interventions could explain part of this variability (Byrne et al. 2006; Colley et al. 2008).

However, differences in body mass and fat loss still occur when adherence is accounted for (Caudwell et al. 2013b; King et al. 2009; King et al. 2008). Compensatory changes in energy intake and non-exercise energy-expenditure have been proposed as an explanation for variability (King et al. 2007) but the extent to which they contribute to individual differences in body mass or fat loss is unclear.

Studies into effects of one to two weeks of exercise training on energy intake have indicated that men partially compensate for the exercise-induced energy expenditure by increasing energy intake (Stubbs et al. 2004; Whybrow et al. 2008). Hence, an increase in energy intake towards a full energy compensation could occur during subsequent weeks. Notably, increases in energy intake have not been observed after short- (Martins et al. 2007) medium- (Caudwell et al. 2013a; Caudwell et al. 2013b) and long-term exercise interventions (Donnelly et al. 2003; Van Etten et al. 1997; Westerterp et al. 1992). However, these results could be explained by unclear definitions of participants' activity status (Caudwell et al. 2013b; Stubbs et al. 2002; Whybrow et al. 2008), unsupervised exercise interventions (Martins et al. 2007)
and assessment of energy intake through a food-frequency questionnaire (Drenowatz et al. 2012).

In contrast, effects of exercise on physical activity energy expenditure are confusing. Some studies report exercise-induced decreases in non-exercise energy expenditure (Colley et al. 2010; Manthou et al. 2010; Meijer et al. 1999), others no change (Church et al. 2009; Hollowell et al. 2009; Keytel et al. 2001; Turner et al. 2010; Van Etten et al. 1997), an increase (Hunter et al. 2000) and even mixed findings among groups (Manthou et al. 2010; Rosenkilde et al. 2012). Differences could be explained by participants’ characteristics (e.g. age), exercise characteristics (e.g. intensity, duration and volume), and different measurement techniques (e.g. accelerometers vs. diaries). For instance, in some studies physical activity has been assessed from recall (McLaughlin et al. 2006; Manthou et al. 2010) that has questionable validity and reliability in the assessment of energy expenditure (Andre and Wolf 2007).

There is a need to investigate long-term effects of exercise on energy intake and physical activity energy expenditure using improved measures. In an attempt to overcome limitations of previous research, this study examined effects of a supervised moderate-intensity 12-week exercise intervention on 7-day energy intake and expenditure using weighed-food diaries and a device that combines heart rate monitoring and accelerometry (Actiheart), respectively. Additionally, this study examined only inactive men because: this group compensates differently than active counterparts in response to an acute bout of exercise (Rocha et al. 2013); low physical activity is associated with dysregulation of energy intake over a year (Shook et al. 2015); physical inactivity is a major risk factor for the development of metabolic diseases and increased mortality from all causes (Haskell et al. 2009); and
difficulties studying women because of influences from menstrual cycle, premenstrual symptoms and use of hormonal contraceptives (Rocha et al. 2015).

**Materials and methods**

**Participants and study procedures**

The study was approved by the Faculty of Health and Wellbeing Research Ethics Committee, Sheffield Hallam University. From advertisements placed in the University and local community forums, seventy-eight participants requested information about the study from which sixty-nine maintained interest. These participants were contacted by telephone or e-mail and invited for preliminary screening. During this visit, participants were given a tour of the university facilities, an explanation of study requirements and any questions were answered. Informed consent was given followed by completion of a health screen, physical-activity, eating-behaviour and food-cravings questionnaires.

Participants were recruited if they were healthy (no known chronic diseases) men aged between 18 and 40 years, normal, overweight or obese (body mass index between 18.5 and 35 kg/m²), non-smokers, not dieting and had a stable body mass (less than 2 kg variation) in the six months before the study. Additionally, participants were excluded if deemed highly restrained eaters (i.e. having a score of more than 18 and a percentage above 66% on the Three-Factor Eating Questionnaire (TFEQ) (Karlsson et al. 2000), undertook more than 150 minutes of moderate-intensity physical activity per week (self-reported - modified version of Godin Leisure-Time Exercise Questionnaire, Godin and Shepard 1985) or took medications that could affect food intake or metabolism.
From the sixty-nine interested participants, only twelve met the inclusion criteria and were invited for a second visit that occurred in the morning after a 10-hour overnight fast with only water consumption permitted. This visit involved the measurement of resting blood pressure and heart rate, collection of finger-prick capillary-blood samples, anthropometry and completion of the Åstrand-Rhyming cycle ergometer test (Åstrand and Rhyming, 1954). An explanation of dietary recording and the use of the Actiheart was also provided. Participants then wore the Actiheart and recorded their food consumption for a week before starting the supervised 12-week exercise programme. At this point, the veracity of self-reported measures of physical activity was confirmed with the Actiheart data (all participants were in the sedentary to light activity lifestyle range (1.40-1.69) - WHO, 2004) and one participant withdrew from the study because he could not adhere to the protocol. Hence, a total of 11 inactive men undertook the exercise intervention.

After completing the intervention, to minimise circadian and other similar influences on measures, participants visited the laboratory for assessment at the same time of day and under the same conditions as the pre-intervention occasion. During this post-intervention visit participants completed the eating-behaviour and food-cravings questionnaires. Measures initially made in the second preliminary visit were also repeated. The seven-day assessment of post-intervention free-living energy intake and expenditure then began on the same day of the week as the pre-intervention assessment. At the end of the study participants were offered three months free gym membership. A schematic representation of the study is presented in Figure 1.
Eating behaviour and food cravings

Participants completed the 18-item revised version of the TFEQ (Karlsson et al. 2000) at the preliminary and post-intervention visits to assess changes in three aspects of eating behaviour: cognitive restraint (conscious restriction of food intake to control body mass); uncontrolled eating (tendency to eat more than usual because of a loss of control over intake accompanied by subjective feelings of hunger); and emotional eating (inability to resist emotional cues relating to consumption). Cronbach’s Alpha for the subscales in this study was 0.71, 0.80 and 0.85, respectively. Participants’ food cravings for the previous month were also assessed at the preliminary and post-intervention visits using the Food Craving Inventory (FCI) (White et al. 2002). The FCI is a 28-item self-report measure of general and specific food cravings scored on a 1-5 Likert scale (where 1 = Never, 5 = Almost every day). It consists of four subscales: carbohydrates/starches (eight items), fast-food fats (four items), high-fat foods (eight items), and sweets (eight items) and defines food craving as an intense desire to consume a particular food (or food type) that is difficult to resist (White et al. 2002).

According to the same authors, this definition recognises that food craving is an internal experience that has cognitive and emotional (drive or motivational) properties. For the present study, the FCI was modified slightly by substitution of typically American foods with English equivalents (e.g., replacing "hot dogs" with "sausage rolls") with similar macronutrient profiles. This ensured that the composition of each subscale was unaffected.

Total craving score was calculated as the mean of all food items scores, and the specific craving score as the mean score for the food items included in that subscale. In this study, the Cronbach’s Alpha for the FCI subscales was Carbohydrates/starches = 0.80; fast food fats = 0.62; high-fat foods = 0.63; sweets = 0.72.
Resting heart rate and arterial blood pressure

Resting heart rate and arterial blood pressure were assessed during the health screening by oscillometric blood pressure monitoring (Dash 2500, GE Healthcare, Finland) calibrated according to the manufacturer’s requirements. Before the assessment, participants lay supine for 10 minutes, relaxed and not moving or speaking. The arm measured was supported at the same height as the heart and was not constricted by tight clothing. Measurements were taken in duplicate with at least 1-min rest between and the mean of these values recorded.

Blood analyses

Finger-prick capillary-blood samples were taken after a 10-hour overnight fast and analysed with an enzymatic peroxidase dry chemistry method (Cholestech LDX System) to determine plasma total cholesterol (TC), high density lipoproteins (HDL), low density lipoproteins (LDL), non-high density lipoproteins (Non-HDL), triglycerides and glucose concentrations. The Cholestech LDX analyser has been validated with comparisons made against laboratory analysis (Parikh et al. 2009). This method has a range of detection for TC (2.59-12.9 mmol/L), HDL-C (0.39-2.59 mmol/L), triglycerides (range 0.51-7.34 mmol/L), and glucose (range 2.78-27.8 mmol/L). According to manufacturer’s instructions LDL and Non-HDL are calculated from previous values as follows:

\[ \text{LDL} = (\text{TC} - (\text{HDL}) - (\text{triglycerides} \div 5) \]
\[ \text{Non-HDL} = \text{TC} - \text{HDL} \]

Anthropometry
Procedures adhered to recommendations of the International Society for the Advancement of Kinanthropometry (ISAK). Stature, body mass, waist and hip circumference were recorded as previously described (Rocha et al. 2013). Body Mass Index (BMI) was calculated as body mass in kilograms divided by the square of stature in metres. Percentage of body fat, skeletal muscle mass and visceral fat area were estimated via a bioelectrical impedance body composition analyser InBody720 (Derwent Healthcare Ltd, UK) according to manufacturer’s instructions. Measurements were performed without shoes and socks with participants instructed to slightly abduct their arms and remain still in the upright position. All bioelectrical impedance measurements were performed with the participants having fasted for at least two hours, voided and having refrained from exercise during that day.

Åstrand-Rhyming cycle-ergometer test

The Åstrand-Rhyming cycle ergometer test (Åstrand & Rhyming, 1954) estimated participant's maximum oxygen consumption. The test comprised 6 min of continuous cycling aiming for a heart rate between 125 and 170 bpm. The pedalling rate was initially set at 50 or 60 rpm depending on the participant’s ability to maintain it. Exercise intensity was adjusted to the individual as indicated by the test protocol. After completion of the test, the intensity of cycling was decreased and participants cooled down. The mean of the last two heart rates and the final exercise intensity were used to estimate maximum oxygen consumption from the adjusted nomogram (Åstrand 1960). Being submaximal, this test minimises risk in men of various ages who are unaccustomed to exercise.

7-day free-living energy intake
All participants received guidance on how to complete the dietary record and weigh food portions and were instructed to contact the experimenter if they were unsure or had any questions about how to record foods accurately. When weighing was not possible, participants estimated portion sizes using standard household measures. Upon receipt, food diaries were reviewed in the presence of the participant to ensure completeness and legibility, with any missing or unclear items being corrected. Food diaries were analysed to estimate energy and macronutrient intake using the dietary analysis software NetWisp (version 3.0; Tinuviel, UK) with unlisted foods being inputted according to information provided on the packaging.

7-day free-living energy expenditure

Free-living energy expenditure for the seven days was estimated from Actiheart data (Cambridge Neurotechnology, UK). This single-piece, light-weight water-proof device has a heart rate monitor and an accelerometer (Brage et al. 2005). The device was attached to the participant’s chest using two electrocardiogram (ECG) electrodes (E4 T815 Tele electrode, UK): a medial electrode placed at the level below the apex of the sternum and a lateral electrode placed on the same horizontal level as lateral as possible. This positioning of the monitor at the level below the apex of the sternum produces clearer heart rate data (Brage et al. 2006). Participants were told to wear the monitor at all times, awake or asleep, and were also asked to record in an activity log the times (if any) where they did not wear the device. The epoch (i.e. interval of time between recordings) was set for one minute.

At the end of the free-living period, participants returned the Actihearts and the data were downloaded using a docking station and analysed using commercial software. Heart rate and accelerometer data were converted to energy expenditure according to the revised group
calibration branched equation (Brage et al. 2007). Total daily energy expenditure was calculated as the sum of physical activity energy expenditure (PAEE), diet-induced thermogenesis, and resting energy expenditure. Resting energy expenditure was estimated from the Schofield equations (Schofield 1985) while diet-induced thermogenesis was assumed to equal 10% of total energy expenditure.

**Exercise intervention**

All supervised sessions were led by one researcher and undertaken at the exercise suite on University grounds. During the exercise intervention, participants exercised for one hour a minimum of three times per week. Each session involved a 5-10 min warm-up period, a 40 min main exercise period at approximately 50-60% of heart rate reserve and a 5-10 min cool down. Energy expended by each participant during the exercise sessions was estimated at week 1 and week 12 of the intervention according to a heart rate prediction equation (Keytel et al. 2005). This equation improves on previous heart rate predictive equations (Hiiloskorpi et al. 1999; Rennie et al. 2001) by allowing adjustment for age, sex, body mass and fitness.

Each participant's exercise intensity and mode were altered according to their rating of perceived exertion and general feedback. Participants chose to exercise on a treadmill, cycle ergometer, rower or elliptical ergometer. For the first three weeks, participants completed three sessions per week, which could increase to a maximum of four supervised sessions per week. Unsupervised sessions were undertaken when work or other commitments did not allow participants to attend the three sessions at the exercise suite. For these sessions the researcher gave participants a heart rate monitor and a session plan to undertake in their own
time. This required participants to record the day, time, type, duration and intensity (i.e. mean heart rate during each bout of exercise) of all exercise undertaken in the session sheet.

262 **Statistical analyses**

263 Data were analysed using the Statistical Package for the Social Sciences software for windows (SPSS 19.0, U.S.A.). Paired t-tests compared estimated exercise energy expenditure, mean exercise heart rate and rating of perceived exertion, body composition, resting heart rate, arterial blood pressure, estimated maximum oxygen consumption, metabolic profile (TC, HDL, non-HDL, triglycerides, LDL, and fasting glucose), cognitive restraint, uncontrolled eating, emotional eating, food cravings, 7-day mean energy intake, macronutrient intake and energy expenditure before and after the exercise intervention. Fully within-groups factorial ANOVAs (Intervention x Day of the week) compared energy intake, macronutrient intake and energy expenditure before and after the exercise intervention (Intervention effect) over the 7 days (Day of the week effect). In addition, Cohen’s $d$ (standardised mean difference) effect sizes evaluated outcomes. The $d$ was determined by dividing the difference between means with the pooled standard deviation thus reflecting differences expressed in standard deviation units. According to Cohen’s (1988) guidelines, effect sizes were interpreted as small ($d=0.2$), medium ($d=0.5$), and large ($d=0.8$).

278 **Results**

279 **Participants’ baseline characteristics**
Participants’ mean age at baseline was 25.5 years (SD = 4.8) and physical characteristics are presented in Table 1. Participants’ BMIs ranged from 19.7 to 33.8 kg·m\(^{-2}\) with participants being classified as lean (n=6), overweight (n=4) and obese (n=1).

Compliance and exercise energy expenditure

Individual compliance with the exercise sessions (supervised and unsupervised) was eight participants having 100% attendance, two participants 97% and one participant 81%. In total, 68% of exercise sessions were supervised, 29.8% were unsupervised and 2.2% were missed. However, half of the unsupervised sessions were undertaken by two participants who changed residences in the first two weeks of their exercise intervention. The new residences were an hour away from the exercise suite by motorised transport. This compromised attendance. Contrary to most participants (n=9) that had sporadic unsupervised sessions, these two participants had a high percentage of unsupervised sessions (75% and 92%, respectively).

Mean exercise heart rate ($p=0.031$, $d=0.41$) and RPE ($p=0.036$, $d=0.29$) were greater in week 12 (146 ± 6 bpm; 13.4 ± 0.9) than in week 1 (144 ± 6 bpm; 13.1 ± 1.0) but there were no differences between the mean individual ($p=0.646$, $d=0.42$) or total weekly ($p=0.370$, $d=0.42$) exercise energy expenditure between the first (2478 ± 220 kJ; 7433 ± 660 kJ) and last week (2527 ± 463 kJ; 7881 ± 2061 kJ) of the exercise intervention.

Anthropometry

The 12-week exercise intervention reduced body mass, BMI, waist circumference, hip circumference, body fat and percentage of body fat (Table 1). No changes were observed in
skeletal muscle mass (SMM), percentage of SMM or visceral fat area. At the individual level, changes in body mass, body fat and SMM, varied considerably (Figure 2).

**Resting heart rate, arterial blood pressure and cardiopulmonary fitness**

Resting diastolic blood pressure decreased (70 ± 7 mmHg vs. 66 ± 6 mmHg, \( p=0.021, d=-0.64 \)) with the exercise intervention whereas \( \dot{V}O_{2\text{max}} \) increased (43.1 ± 7.4 ml/kg/min vs. 51.1 ± 8.4 ml/kg/min, \( p=0.001, d=1.05 \)). There were no differences between baseline and post-intervention values for resting heart rate (58 ± 10 bpm vs. 57 ± 8 bpm, \( p=0.657, d=-0.16 \)) and systolic blood pressure (120 ± 8 mmHg vs. 118 ± 8 mmHg, \( p=0.186, d=-0.27 \)).

**Metabolic profile**

Because some participants had values outside the range of detection of the Cholestech LDX, sample size was different for TC and glucose (n=11), HDL and Non-HDL (n=10), triglycerides (n=8), and LDL (n=7). Fasting total cholesterol and glucose were greater before than after the 12-week exercise intervention (Table 2). There were no differences between baseline and post-intervention values for HDL, non-HDL, LDL and triglycerides.

**Eating behaviour and food cravings**

There were no differences between baseline and post-exercise intervention scores for uncontrolled eating and emotional eating scores (Table 3). However, there was a trend with a medium effect size for cognitive restraint to be greater after the exercise intervention than at baseline. Total food cravings and specific cravings of high-fat foods, fast-food fats and
carbohydrates/starches decreased from baseline to 12 weeks. There was also a trend with a large effect size for cravings of sweets to be lower after the exercise intervention than at baseline.

Energy and macronutrient intake

There were no main effects or interaction for energy and macronutrients intake ($p>0.05$). There were also no differences between the 7-day mean energy and macronutrients intake before and after the exercise intervention (Table 4).

Energy expenditure

Two participants did not complete 7-day Actiheart data for at least one of the two measurement periods, therefore analyses were made for 9 participants. There were no main effects or interaction for energy expenditure and physical activity energy expenditure ($p>0.05$). Likewise, there were no differences between the 7-day mean energy expenditure and physical activity energy expenditure before and after exercise intervention (Table 4).

Discussion

The main finding arising from this study is that 12 weeks of moderate-intensity aerobic exercise decreases food cravings and increases cognitive restraint without changing other eating behaviours, 7-day energy intake or expenditure in inactive men. Moreover, the lack of difference between pre- and post-exercise intervention energy expenditure suggests that
participants in this study reduced their activity to values similar to baseline immediately after
direct supervision and support ended.

Twelve weeks of moderate aerobic exercise did not change uncontrolled eating and emotional
eating but there was a trend with a medium effect size for participants to have a greater
cognitive restraint score after the exercise intervention. This finding is unsurprising since
increases in cognitive restraint have been associated with reductions in body mass (Foster et
al. 1998) in exercise (King et al. 2009) and dietary (Westerterp-Platenga et al. 1998)
interventions. However, it is unclear if this increase in cognitive restraint arose from the
treatments or reductions in body mass. A possible explanation is that participants’ become
more aware of their lifestyle, which in turn could increase their control over it. Total food
cravings and specific cravings of high-fats, fast-food fats and carbohydrates/starches
decreased from baseline after the exercise intervention. To the authors’ knowledge no study
has assessed general and specific food cravings before and after an exercise intervention
alone. However, these findings are in agreement with Cornier et al. (2012) which suggests
that chronic exercise training is associated with an attenuated response to visual food cues in
brain regions known to be important in food intake regulation. Additionally, food cravings
are closely associated with mood, which can act as an antecedent and as a consequence of the
food cravings (Hill et al. 1991). Therefore, it is possible that the decrease in food cravings is
related to the exercise-induced improvement in mood (Hoffman & Hoffman, 2008).

Reductions in food cravings have also occurred with low- and very-low energy diets (Harvey
et al. 1993; Martin et al. 2006) but these findings are not universal as Wadden et al. (1997)
reported no differences on food cravings after 48 weeks of diet alone, diet plus aerobic
training, diet plus strength training, or diet combined with aerobic and strength training.
Moreover, Foster et al. (1992) did not find any effects of 24 weeks of three very-low-energy
diets on food cravings, however these two later studies did not use a validated measure of food cravings.

Consistent with previous studies (Caudwell et al. 2013a; Caudwell et al. 2013b), the exercise intervention did not change energy intake. These findings are surprising considering the large inter-individual variability in changes of body and fat mass. In the present study, nine participants had varied reductions in body and fat mass, one participants’ body composition did not change and one participants’ body and fat mass increased. These changes suggest that compensatory responses are highly individual. However, findings for the two latter participants could be explained by differences in the exercise intervention as the participant that did not have any changes in body composition consistently reduced the intensity of the prescribed exercise for the unsupervised sessions (estimated exercise mean energy expenditure of supervised sessions = 2316 kJ and unsupervised sessions = 1527 kJ) while the participant with increases in body and fat mass had the highest percentage of unsupervised sessions (92%) that were reliant on self-report. The variability of responses in the remaining nine participants could not be explained by differences in the amount of supervised and unsupervised sessions or estimated exercise energy expenditure. Therefore, the use of only two time points (pre- and post-intervention) might lack sensitivity to detect compensatory responses.

Similar to previous studies, there were no changes in energy expenditure after the exercise intervention (Church et al. 2009; Hollowell et al. 2009; Turner et al. 2010). These results suggest that most participants did not continue exercising for the same frequency and intensity after the end of the study exercise intervention. This is surprising since participants were offered three months' free gym membership and encouraged to continue their exercise
programme independently so complementary strategies are needed to encourage continuation of exercise. Moreover, this could explain why exercise training studies have not shown a consistent effect on long-term maintenance of body mass (Fogelholm and Kukkonen-Harjula 2000; Franz et al. 2007).

Despite the lack of effect on energy intake and expenditure, the 12-week exercise intervention produced beneficial changes in body composition and health markers. Reductions in body mass (-1.6 ± 1.7 kg) and body fat (-1.1 ± 1.4 kg) were modest and did not reach the minimum change (5% of body mass, i.e. mean change of 4 kg in this sample) necessary to reduce metabolic and cardiovascular disease risk (Blackburn 1995; Wing et al. 2011). However, this finding could be because of the moderate-intensity and frequency of the exercise sessions, the length of the exercise intervention, and only five participants being classified as overweight or obese at baseline. Participants mean value for waist circumference at baseline did not increase health risk (> 94 cm) (Han et al. 1995) even when considering the specific optimal waist circumference cut-point of 87 cm for a body mass index between 18.5 and 24.9 kg/m² in men (Ardern et al. 2004). Nevertheless, the reduction in waist circumference (-3.8 ± 2.8 cm) is important because of the association between excess abdominal adiposity and increased risk of mortality, cardiovascular disease, diabetes, insulin resistance and metabolic syndrome (Katzmarzyk et al. 2006; Klein et al. 2007). Moreover, the exercise intervention made positive changes in health markers such as the increased estimated maximum oxygen consumption and decreased resting diastolic blood pressure, total cholesterol and fasting glucose. Together, these changes reinforce the health benefits of exercising even if reductions in body mass are modest.
The strengths of the study were its longitudinal design, control over day and time of measurements, the use of weighed-food diaries and Actihearts over a 7-day period and strict inclusion criteria to control for confounding factors. Limitations are that participants were young healthy inactive men, therefore findings might not apply to active or older adults. Moreover, the small sample size that reflects the demanding nature of the study and the strict inclusion criteria, did not allow inclusion of a control group. Finally, energy intake and expenditure data collected in the free-living should be interpreted cautiously because they are highly dependent on participants’ compliance with methods and instructions and hence, prone to error. Nevertheless, to identify possible underreporting, the mean ratio of energy intake to basal metabolic rate was calculated for each individual with no participants having a ratio lower than Goldberg et al. (1991) cut-off of 1.1.

In summary, this study demonstrated that 12 weeks of moderate-intensity aerobic exercise decreased food cravings and increased cognitive restraint without inducing changes in other eating behaviours and weekly energy intake and expenditure in inactive men. Findings support the importance of exercise for health improvements even when reductions in body mass are modest. Moreover, inactive men might not maintain the same volume of exercise without direct supervision, even when they have free access to specialist, well-equipped facilities. This suggests a need for complementary strategies to help inactive men maintain exercise after the end of the exercise intervention.

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associated with dysregulation of energy intake and fat mass gain over 1 year. The American journal of clinical nutrition, 102(6), 1332-1338. PMID:26561620


Tables
Table 1 Participants' physical characteristics

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>After intervention</th>
<th>t</th>
<th>p</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stature (m)</td>
<td>1.80 ± 0.05</td>
<td>1.80 ± 0.05</td>
<td>1.00</td>
<td>0.341</td>
<td>-0.04</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>79.9 ± 15.4</td>
<td>78.3 ± 14.6</td>
<td>3.12</td>
<td>0.011</td>
<td>-0.11</td>
</tr>
<tr>
<td>BMI (kg·m⁻²)</td>
<td>24.6 ± 3.8</td>
<td>24.1 ± 3.6</td>
<td>3.31</td>
<td>0.008</td>
<td>-0.12</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>82.9 ± 10.3</td>
<td>79.1 ± 8.6</td>
<td>4.49</td>
<td>0.001</td>
<td>-0.42</td>
</tr>
<tr>
<td>Hip circumference (cm)</td>
<td>99.6 ± 10.7</td>
<td>97.4 ± 10.1</td>
<td>4.17</td>
<td>0.002</td>
<td>-0.22</td>
</tr>
<tr>
<td>Skeletal muscle mass (kg)</td>
<td>37.4 ± 5.6</td>
<td>37.1 ± 5.2</td>
<td>1.16</td>
<td>0.274</td>
<td>-0.07</td>
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<tr>
<td>Skeletal muscle mass (%)</td>
<td>47.3 ± 4.3</td>
<td>47.8 ± 4.1</td>
<td>-1.53</td>
<td>0.157</td>
<td>0.12</td>
</tr>
<tr>
<td>Body fat (kg)</td>
<td>14.6 ± 8.5</td>
<td>13.5 ± 8.0</td>
<td>2.64</td>
<td>0.025</td>
<td>-0.14</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>17.4 ±7.3</td>
<td>16.3 ± 7.1</td>
<td>2.39</td>
<td>0.038</td>
<td>-0.16</td>
</tr>
<tr>
<td>Visceral fat area (%)</td>
<td>71.9 ± 39.3</td>
<td>71.4 ± 36.3</td>
<td>0.23</td>
<td>0.823</td>
<td>-0.01</td>
</tr>
</tbody>
</table>

N=11; values presented as mean ± SD; BMI= body mass index.

Table 2 Participants' metabolic profile

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>After intervention</th>
<th>t</th>
<th>p</th>
<th>d</th>
</tr>
</thead>
</table>

N=11; values presented as mean ± SD; BMI= body mass index.
<table>
<thead>
<tr>
<th></th>
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<th>After intervention</th>
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<th>p</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total cholesterol (mmol/L)</strong></td>
<td>4.30 ± 0.94</td>
<td>4.10 ± 1.02</td>
<td>2.46</td>
<td>0.034</td>
<td>-0.22</td>
</tr>
<tr>
<td><strong>HDL (mmol/L)</strong></td>
<td>1.18 ± 0.29</td>
<td>1.08 ± 0.25</td>
<td>1.59</td>
<td>0.146</td>
<td>-0.40</td>
</tr>
<tr>
<td><strong>Non-HDL (mmol/L)</strong></td>
<td>3.19 ± 0.89</td>
<td>3.17 ± 0.93</td>
<td>0.19</td>
<td>0.856</td>
<td>-0.02</td>
</tr>
<tr>
<td><strong>LDL (mmol/L)</strong></td>
<td>2.98 ± 0.71</td>
<td>3.05 ± 0.69</td>
<td>-0.48</td>
<td>0.652</td>
<td>0.11</td>
</tr>
<tr>
<td><strong>Triglycerides (mmol/L)</strong></td>
<td>1.01 ± 0.46</td>
<td>0.97 ± 0.38</td>
<td>0.30</td>
<td>0.776</td>
<td>-0.09</td>
</tr>
<tr>
<td><strong>Glucose (mmol/L)</strong></td>
<td>5.01 ± 0.35</td>
<td>4.58 ± 0.38</td>
<td>4.75</td>
<td>0.001</td>
<td>-1.22</td>
</tr>
</tbody>
</table>

**N=11 for Total cholesterol and glucose, N=10 for HDL and Non-HDL, N=8 for triglycerides and N=7 for LDL; HDL = high density lipoproteins; values presented as mean ± SD; Non-HDL = non-high density lipoproteins; LDL = low density lipoproteins.**

**Table 3** Eating behaviour and food cravings scores

<table>
<thead>
<tr>
<th>TFEQ-R18 scores (%)</th>
<th>Baseline</th>
<th>After intervention</th>
<th>t</th>
<th>p</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cognitive restraint</strong></td>
<td>24.7 ± 11.5</td>
<td>33.8 ± 16.0</td>
<td>-2.14</td>
<td>0.058</td>
<td>0.68</td>
</tr>
<tr>
<td><strong>Uncontrolled eating</strong></td>
<td>41.3 ± 17.0</td>
<td>36.0 ± 15.4</td>
<td>1.57</td>
<td>0.147</td>
<td>-0.34</td>
</tr>
<tr>
<td><strong>Emotional eating</strong></td>
<td>25.3 ± 23.4</td>
<td>26.3 ± 29.5</td>
<td>-0.22</td>
<td>0.831</td>
<td>0.04</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>FCI scores (1-5 Likert scale)</th>
<th>Baseline</th>
<th>After intervention</th>
<th>t</th>
<th>p</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total food cravings</strong></td>
<td>2.3 ± 0.4</td>
<td>1.9 ± 0.4</td>
<td>3.24</td>
<td>0.009</td>
<td>-1.19</td>
</tr>
<tr>
<td><strong>High-fats</strong></td>
<td>1.9 ± 0.5</td>
<td>1.5 ± 0.4</td>
<td>2.69</td>
<td>0.023</td>
<td>-0.90</td>
</tr>
<tr>
<td><strong>Fast-food fats</strong></td>
<td>2.9 ± 0.7</td>
<td>2.4 ± 0.8</td>
<td>3.24</td>
<td>0.009</td>
<td>-0.71</td>
</tr>
<tr>
<td><strong>Carbohydrate/starches</strong></td>
<td>2.3 ± 0.8</td>
<td>1.9 ± 0.7</td>
<td>3.22</td>
<td>0.009</td>
<td>-0.56</td>
</tr>
</tbody>
</table>
Table 4 Participants' 7-day mean energy intake, macronutrient intake and energy expenditure

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>After intervention</th>
<th>t</th>
<th>p</th>
<th>d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy intake (kJ)</td>
<td>11742 ± 4043</td>
<td>11294 ± 3952</td>
<td>1.03</td>
<td>0.326</td>
<td>-0.12</td>
</tr>
<tr>
<td>Protein intake (%)</td>
<td>15.9 ± 2.3</td>
<td>17.1 ± 3.0</td>
<td>-1.68</td>
<td>0.123</td>
<td>0.48</td>
</tr>
<tr>
<td>CHO intake (%)</td>
<td>51.8 ± 5.2</td>
<td>49.8 ± 6.7</td>
<td>0.91</td>
<td>0.384</td>
<td>-0.35</td>
</tr>
<tr>
<td>Fat intake (%)</td>
<td>32.3 ± 4.8</td>
<td>33.1 ± 6.2</td>
<td>-0.43</td>
<td>0.678</td>
<td>0.14</td>
</tr>
<tr>
<td>Energy expenditure (kJ)</td>
<td>11644 ± 1347</td>
<td>11729 ± 1902</td>
<td>-0.26</td>
<td>0.799</td>
<td>0.05</td>
</tr>
<tr>
<td>PAEE (kJ)</td>
<td>2856 ± 831</td>
<td>3013 ± 1299</td>
<td>-0.56</td>
<td>0.594</td>
<td>0.15</td>
</tr>
</tbody>
</table>

N=11 for 7-day energy and macronutrient intake, N=9 for 7-day physical activity energy expenditure (PAEE); CHO = carbohydrates; values presented as means ± SD.

Figure captions

Figure 1 Schematic representation of the study.

Figure 2 Individual changes in body mass, fat and skeletal muscle mass (SMM) in response to the 12 weeks exercise intervention. Each grouped three histograms represents values for one participant.