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1 **TITLE:** Exercise-induced changes in central adiposity during a RCT: Effect of exercise dose  
2 and associations with compensation

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13 **ABSTRACT**

14 Context: Exercise can decrease central adiposity, but the effect of exercise dose and the  
15 relationship between central adiposity and exercise-induced compensation is unclear.

16 Objective: Test the effect of exercise dose on central adiposity change and the association  
17 between central adiposity and exercise-induced weight compensation.

18 Methods: In this ancillary analysis of a 6-month randomized controlled trial, 170 participants  
19 with overweight or obesity (mean±SD BMI: 31.5±4.7 kg/m<sup>2</sup>) were randomized to a control  
20 group or exercise groups that reflected exercise recommendations for health (8 kcal/kg/week  
21 [KKW]) or weight loss and weight maintenance (20 KKW). Waist circumference was measured,

1 and dual-energy X-ray absorptiometry assessed central adiposity. Predicted weight change was  
2 estimated and weight compensation (weight change minus predicted weight change) was  
3 calculated.

4 Results: Between-group change in waist circumference (control: 0.0 cm [95% CI: -1.0,1.0], 8  
5 KKW: -0.7 cm [95% CI: -1.7,0.4], 20 KKW: -1.3 cm [95% CI: -2.4,-0.2]) and visceral adipose  
6 tissue (VAT; control: -0.02 kg [95% CI: -0.07,0.04], 8 KKW: -0.01 kg [95% CI: -0.07,0.04], 20  
7 KKW: -0.04 kg [95% CI: -0.10,0.02]) was similar ( $P \geq 0.23$ ). Most exercisers (82.6%)  
8 compensated (predicted weight change lower than actual weight change). Exercisers who  
9 compensated exhibited a 2.5 cm (95% CI: 0.8,4.2) and 0.23 kg (95% CI: 0.14,0.31) increase in  
10 waist circumference and VAT, respectively, versus those who did not ( $P < 0.01$ ). Desire to eat  
11 predicted VAT change during exercise ( $\beta = 0.21$ ;  $P = 0.03$ ).

12 Conclusions: In the presence of significant weight compensation, exercise at doses recommended  
13 for health and weight loss and weight maintenance leads to negligible changes in central  
14 adiposity.

## 15 **INTRODUCTION**

16 Over two-thirds of the US population lives with overweight or obesity, which are characterised  
17 by an elevated BMI (1). Overweight and obesity are key risk factors for cardiometabolic disease  
18 development (2), and an established link between BMI and cardiometabolic disease risk exists  
19 (3). However, central adiposity, primarily visceral adipose tissue (VAT), is strongly associated  
20 with metabolic disease risk factors (e.g., high blood glucose (4) and dyslipidaemia (5)),  
21 cardiometabolic conditions (e.g., metabolic syndrome, type 2 diabetes (6) and cardiovascular  
22 disease (5)), and mortality (7). Additionally, some have shown central adiposity is more strongly

1 related to metabolic diseases such as type 2 diabetes than fat stored in other regions (3,6).

2 Interventions that reduce central adiposity are therefore needed to prevent and treat metabolic  
3 disease and improve health span.

4 Aerobic exercise training can decrease central adiposity in individuals with overweight or  
5 obesity, regardless of age, sex, and ethnicity (8). However, the influence of aerobic exercise dose  
6 on central adiposity is equivocal, with some showing that greater doses do not decrease markers  
7 of central adiposity (9,10) and others suggesting that reductions in VAT are improved at higher  
8 exercise doses (11,12). Understanding the influence of exercise dose on central adiposity is  
9 crucial to help design optimal aerobic exercise regimens that enhance central adiposity  
10 outcomes. Thus, considering the conflicting findings, large randomized controlled trials are  
11 needed to test the effect of exercise dose on change in central adiposity markers, including VAT,  
12 in individuals with overweight and obesity.

13 Exercise-induced weight compensation, which is actual weight loss lower than weight loss  
14 predicted based on the energy expenditure of exercise, is common (13,14). Weight compensation  
15 can occur due to multiple behavioral and physiological factors, although substantial weight  
16 compensation during exercise is primarily caused by elevations in energy intake (13). The  
17 relationship between compensation and central adiposity change during aerobic exercise is  
18 poorly understood. Some have demonstrated that reductions in VAT during high volumes of  
19 exercise are attenuated when compensation occurs through increased energy intake (15,16), but  
20 the association between compensation and changes in central adiposity during exercise at doses  
21 similar to that recommended for health (700 to 1,000 kcal/week) and weight loss and weight loss  
22 maintenance (~2000 kcal/week) (17) has not been thoroughly studied. Moreover, the  
23 associations between central adiposity change and the mechanisms related to compensation—

1 namely increased energy intake, reduced energy expenditure and physical activity, and  
2 maladaptive eating attitudes and behaviors—during exercise training are not well understood.  
3 Assessing the compensation-related predictors of central adiposity change during exercise could  
4 pinpoint factors and/or constructs that may be targeted to enhance improvements in central  
5 adiposity.

6 This ancillary analysis had two primary aims. First, we tested the effect of aerobic exercise dose  
7 on central adiposity in individuals with overweight or obesity. Second, we examined the  
8 association between central adiposity change and compensation during exercise training at  
9 guidelines akin to those recommended for health and weight loss and weight loss maintenance.  
10 As an exploratory aim, we assessed if mechanisms related to exercise-induced compensation  
11 predict VAT change during exercise.

## 12 **METHODS**

### 13 *Study Design*

14 The methods of the Examination of Mechanisms of Exercise-Induced Weight Compensation (E-  
15 MECHANIC) study (ClinicalTrials.gov: NCT01264406) have been detailed elsewhere (13,18).  
16 Briefly, the study was a 6-month randomized controlled trial that took place at Pennington  
17 Biomedical Research Center following Institutional Review Board approval. After the provision  
18 of written informed consent, participants recruited to the study were randomized (N = 198) to  
19 one of three groups: a no-exercise control group, an exercise group that aimed to expend 8  
20 kcal/kg/week (KKW) through exercise, or an exercise group that aimed to expend 20 KKW  
21 through exercise. The 8 KKW group reflected recommendations for general health (~700  
22 kcal/week), while the 20 KKW group reflected recommendations for weight loss and weight

1 loss maintenance (~1760 kcals/week) (13,17). A biostatistician devised a 1:1:1 randomization  
2 ratio, and sex was stratified so that an equal number of males and females were randomized to  
3 each group. Randomization was concealed in an envelope until an interventionist or the study  
4 manager opened it with the participant. The participants and interventionists supervising exercise  
5 sessions were not blinded to group allocation, but the study investigators and the assessment  
6 team were because group allocation was not disclosed by the study manager or interventionists.  
7 Recruitment and data collection occurred from November 2010 to March 2015 (first participant  
8 enrolled January 2011), finishing when the target sample size was recruited (13,18).

### 9 *Participants*

10 Sedentary (not exercising >20 min on  $\geq 3$  d/week) individuals living with overweight or obesity  
11 (body mass index [BMI]  $\geq 25$  kg/m<sup>2</sup> to  $\leq 45$  kg/m<sup>2</sup>) who were otherwise healthy were recruited  
12 for the trial. Further details on the participant exclusion criteria have been reported (13).

### 13 *Intervention*

14 Aerobic exercise training was conducted on a treadmill at an intensity that maintained  
15 participants within a heart rate range equivalent to 65%-85% of baseline peak oxygen uptake  
16 ( $\dot{V}O_2$ ). Participants in the 8 KKW group performed their complete exercise dose from the start.  
17 To acclimatize participants in the 20 KKW group, participants expended 8 KKW through  
18 exercise in week 1 and 14 KKW through exercise in week 2 before completing their complete  
19 dose (20 KKW of energy expenditure through exercise) from week 3 until the cessation of the  
20 study.

21 Exercise training was fully supervised and monitored. Participants were weighed weekly with a  
22 Tanita scale (Tanita Corporation, Arlington Heights, IL) and selected their exercise frequency

1 (three, four, or five sessions per week) to aid compliance. The energy expenditure target of each  
2 session was calculated by dividing the prescribed exercise dose (8 KKW or 20 KKW) by the  
3 exercise frequency. To meet the energy expenditure targets, the length of the exercise sessions  
4 varied. Real-time estimations of energy expenditure were calculated based on intensity and  
5 participant weight, and energy expenditure was measured periodically via a metabolic cart.  
6 Participants' adherence to their exercise regimen was calculated as attained exercise energy  
7 expenditure divided by prescribed exercise energy expenditure.

8 The control group received health information (e.g., stress management and benefits of healthy  
9 foods), although they were instructed to maintain their baseline physical activity.

#### 10 *Outcome measures*

11 Body weight and waist circumference were measured at baseline and follow-up. Assessments of  
12 body composition were performed by dual-energy X-ray absorptiometry (DXA) at baseline and  
13 follow-up using Lunar iDXA with Encore software version 13.60 (GE Healthcare, Madison, WI,  
14 USA). DXA and Encore software quantified fat mass and body fat percentage for the whole  
15 body, trunk, arms, and legs, as well as VAT. The trunk-fat-to-limb-fat ratio (19) and VAT-to-  
16 total-fat ratio (20) were calculated as further assessments of central adiposity.

17 Compensation was calculated as actual weight change minus predicted weight change. Predicted  
18 weight at the end of the intervention was estimated using a validated dynamic energy balance  
19 model, which is a differential equation based on the first law of thermodynamics and accounts  
20 for metabolic adaptation and body composition changes during aerobic exercise training,  
21 overcoming the drawbacks of traditional predictions of weight (21,22). Predicted change in body  
22 weight and body composition are in response to the change in energy expenditure resulting from



1 an increase in physical activity expenditure, as derived from the literature (22). Compensation  
2 was not included in the dynamic energy balance model. Indeed, the predicted body weight and  
3 body composition changes represent changes without compensation, and hence the difference  
4 between model body weight predictions and observed body weight reflects the degree of weight  
5 compensation.

6 Several measures were conducted at baseline and follow-up to assess mechanisms related to  
7 compensation (13). Energy intake was determined through doubly labelled water (DLW). In the  
8 primary outcome manuscript, change in energy intake with DLW was adjusted for resting  
9 metabolic rate, although estimates were made without adjustment and with adjustments for body  
10 composition (13,23). Results in the present analysis were similar with all estimates of energy  
11 intake; thus, change in energy intake with adjustment for resting metabolic rate is reported.  
12 Resting metabolic rate was examined via Max II metabolic carts (AEI Technologies), and steps  
13 per day were measured with SenseWear armbands (Body Media). The Eating Inventory assessed  
14 dietary restraint, disinhibition, and hunger (24). The Food Craving Inventory assessed intense  
15 desires to consume certain foods irrespective of hunger (only total score was used in the present  
16 analysis) (25). The Food Preference Questionnaire determined food preferences for certain food  
17 groups, as well as a fat preference score (only fat preference score was used in the current  
18 analysis) (26). Further, retrospective visual analogue scales (VAS) assessed perceptions of  
19 appetite (27), the Compensatory Health Beliefs Scale measured compensatory health-related  
20 beliefs (e.g., justifying eating because of exercise) (28), and the Activity Temperament  
21 Questionnaire examined participants' tendency to move (29).

22

## 1 *Statistical analysis*

2 The current analysis assesses secondary endpoints of the E-MECHANIC trial. Since our  
3 secondary endpoints analysis requires follow-up data and adherence to the exercise intervention,  
4 participants assessed in the main analysis of the primary manuscript (i.e., individuals with  
5 baseline and follow up data and  $\geq 75\%$  adherence to their exercise regimen) were considered  
6 (13). Including individuals with  $\geq 75\%$  adherence negated the influence of adherence as a  
7 possible confounder during between-group comparisons. In total, 171 participants satisfied the  
8 follow-up and adherence criteria, but one of the 171 participants did not have a baseline DXA  
9 measurement. As a result, our reference dataset for this study was restricted to 170 participants  
10 (Supplemental Figure. 1 (30)).

11 All statistical analyses were performed in SPSS version 28, with the significance level set to  
12  $\alpha=0.05$ . Differences in change scores between the three study groups were examined by one-way  
13 ANCOVAs, with adjustments for sex and age. Subgroup analyses were performed to examine  
14 variations in study group differences between: (a) those with high waist circumference ( $\geq 102$  cm  
15 for males,  $\geq 88$  cm for females) (31) and healthy waist circumference ( $< 102$  cm for males,  $< 88$   
16 cm for females) at baseline; (b) males and females; and (c) Black participants and participants of  
17 other races. These subgroup analyses were conducted via two-way ANCOVAs adjusted for sex  
18 (except for the male vs. female subgroup analysis), age, and baseline values. Adjusted post-hoc  
19 comparisons (Holm-Bonferroni) were performed when ANCOVA omnibus tests were significant  
20 to ascertain where differences lay. In exercisers, percent compensation (percentage compensation  
21 = [actual weight loss – predicted weight loss] / predicted weight loss) (13). Differences in waist  
22 circumference, VAT, VAT-to-total-fat ratio, and trunk-fat-to-limb-fat ratio change were  
23 examined between those who showed positive compensation (percent compensation  $> 0\%$ ) and

1 those with zero or negative compensation (percent compensation  $\leq 0\%$ ) via one-way ANCOVAs  
2 adjusted for age, sex, and baseline values. Multiple linear regression models adjusted for sex,  
3 age, and baseline values also assessed the association between change in central adiposity indices  
4 and percent compensation. Pearson correlations tested the relationship between change in VAT  
5 and change in mechanisms related to compensation, and significant correlates were then entered  
6 into multiple linear regression models along with age, sex, and VAT at baseline to assess the  
7 predictors of VAT change. We calculated absolute Cohen's d effect size (ES) values to  
8 supplement between-group comparisons (32). Comparisons were considered negligible, small,  
9 medium, and large when ES values were  $<0.20$ ,  $0.20-0.49$ ,  $0.50-0.79$ , and  $\geq 0.80$ , respectively,  
10 based on previous literature (32). Unless noted otherwise, values from inferential tests are  
11 estimated marginal mean (95% CI) while descriptive data are mean (SD).

## 12 **RESULTS**

### 13 *Descriptive*

14 Descriptive characteristics of the participants included in the present analysis are shown in Table  
15 1. Characteristics were similar when the full recruited sample (N = 198) was observed (data not  
16 shown). Most of the participants were female (N = 123; 72.4%) and white (N = 113; 66.5%). The  
17 mean age, weight, and BMI of the participants was 48.8 ( $\pm 11.4$ ) years, 88.6 ( $\pm 15.4$ ) kg, and 31.5  
18 ( $\pm 4.7$ ) kg/m<sup>2</sup>, respectively.

### 19 *Intervention data*

20 The 8 KKW group completed 101.0% ( $\pm 6.3\%$ ) of prescribed exercise energy expenditure and the  
21 20 KKW group completed 98.1% ( $\pm 6.0\%$ ) of prescribed exercise energy expenditure,  
22 demonstrating the high adherence in both groups. Total energy expended by the 8 KKW group

1 and 20 KKW group during exercise was 17,114 ( $\pm 3,175$ ) kcal and 38,992 ( $\pm 7,308$ ) kcal,  
2 respectively, corresponding to 680 ( $\pm 123$ ) kcals/week for the 8 KKW group and 1521 ( $\pm 263$ )  
3 kcals/week for the 20 KKW group. Additional training data are shown in Supplemental Table 1  
4 (30).

5 The average percent compensation shown by the 8 KKW group and the 20 KKW group was  
6 70.0% ( $\pm 129.2\%$ ) and 58.0% ( $\pm 61.7\%$ ), respectively, and overall, 90 exercise participants  
7 (82.6%) showed positive compensation (i.e., lost less weight than expected). Those who  
8 displayed positive compensation and those who displayed zero or negative compensation showed  
9 similar baseline characteristics ( $P \geq 0.17$ ; Supplemental Table 2 (30)).

#### 10 *Weight and total body composition change*

11 A difference between groups was identified for weight and BMI change ( $P = 0.02$ ), with the 20  
12 KKW group exhibiting a decrease compared to control ( $P = 0.03$ ;  $ES \geq 0.51$ ; Table 2). Akin to  
13 results reported in the primary outcomes manuscript (13), total fat mass and body fat percent was  
14 reduced in the 20 KKW group compared to other groups ( $P < 0.05$ ;  $ES \geq 0.44$ ), while no  
15 differences were seen for total lean body mass ( $P = 0.51$ ;  $ES \leq 0.22$ ).

#### 16 *Regional adiposity change*

17 No significant between-group difference was observed for change in waist circumference ( $P =$   
18 0.23), despite confidence interval data indicating a within group reduction in waist circumference  
19 in the 20 KKW group (Table 2). Change in VAT, the VAT-to-total-fat ratio, and the trunk-fat-to-  
20 limb-fat ratio was similar in all groups, with negligible ES values seen (all  $P \geq 0.65$ ; all  $ES \leq$   
21 0.17). In spite of confidence interval data showing reductions in trunk fat mass and trunk percent  
22 fat in the 20 KKW group, between-group differences were not significantly different and small

1 to negligible ES values were revealed (all  $P \geq 0.06$ ; all ES  $\leq 0.43$ ). Arm percent fat, leg fat mass,  
2 and leg percent fat were statistically different between groups (all  $P \leq 0.04$ ). Post-hoc tests for  
3 leg fat mass and leg percent fat indicated a greater reduction in leg fat in the 20 KKW group  
4 compared to other groups ( $P \leq 0.04$ ), although no significant between-group variations were  
5 observed for arm percent fat following adjustment ( $P > 0.08$ ; Table 2). Results from subgroup  
6 analyses are shown in Supplemental Tables 3-5 (30). The effect of study group on changes in  
7 weight, BMI, waist circumference, and DXA endpoints was not modified by waist circumference  
8 at baseline or race (all  $P$  for interaction  $\geq 0.07$ ). There was a two-way interaction between study  
9 group and sex for VAT-to-total-fat ratio and trunk-fat-to-limb-fat ratio ( $P$  for interaction  $< 0.05$ ),  
10 but following adjustments for multiple comparisons, no significant between-group differences  
11 were seen when males and females were analyzed separately ( $P > 0.05$ ).

12 Individuals with positive weight compensation displayed a 2.5 cm (95% CI: 0.8, 4.2; ES = 0.76),  
13 0.23 kg (95% CI: 0.14, 0.31; ES = 1.31), 0.0033 (95% CI: 0.0017, 0.0050; ES = 1.02), and 0.06  
14 (95% CI: 0.01, 0.10; ES = 0.60) increase in waist circumference, VAT, the VAT-to-total-fat  
15 ratio, and the trunk-fat-to-limb-fat ratio, respectively, compared to individuals with zero or  
16 negative weight compensation (all  $P \leq 0.02$ ; Fig. 1). Multiple linear regression analyses showed  
17 greater weight compensation during exercise training was associated with increases in waist  
18 circumference, VAT, the VAT-to-total-fat ratio, and the trunk-fat-to-limb-fat ratio (all  $\beta \geq 0.24$ ;  
19  $P \leq 0.01$ ; Supplemental Table 6 (30)).

20 In Pearson correlation analyses, change in compensatory health beliefs ( $r = 0.20$ ;  $P = 0.04$ ) and  
21 retrospective desire to eat ( $r = 0.23$ ;  $P = 0.02$ ) were related with VAT change during exercise  
22 (Supplemental Table 7 (30)); hence, these variables were entered into the multiple linear  
23 regression analysis. This regression analysis revealed that retrospective desire to eat was a

1 positive predictor of VAT change ( $\beta = 0.21$ ;  $P = 0.03$ ; Table 3). This model also suggested that  
2 compensatory health beliefs was not a significant predictor of VAT change, although a similar  
3 standardized beta was observed ( $\beta = 0.16$ ;  $P = 0.09$ ).

#### 4 **DISCUSSION**

5 Overall, in this ancillary analysis of a large, 6-month randomized controlled trial in individuals  
6 with overweight and obesity, we showed negligible differences in central adiposity change  
7 between a no-exercise control group and two aerobic exercise groups – one similar to guidelines  
8 recommended for health and one similar to guidelines recommended for weight loss and weight  
9 loss maintenance. We also showed that exercisers who displayed positive weight compensation  
10 (i.e., lost less weight than predicted) showed reduced improvements in central adiposity relative  
11 to those who did not compensate. These results indicate that exercise dose has no significant  
12 impact on central adiposity, and that compensation is likely to negate central adiposity  
13 improvements during exercise at guidelines for health and weight loss and weight loss  
14 maintenance.

15 Greater energy expenditure during aerobic exercise training leads to increased weight loss due to  
16 a higher energy deficit (33), but the influence of aerobic exercise dose on central adiposity is  
17 equivocal over 6 months or more. While Recchia and colleagues demonstrated that greater  
18 energy expenditure through aerobic exercise leads to small yet significant improvements in  
19 central adiposity (12), a smaller trial showed that increasing exercise dose does not lead to  
20 improvements in VAT (9), and others have demonstrated no differences in waist circumference  
21 between individuals performing exercise at 50%, 100%, and 150% of guidelines (10). Results  
22 from our trial displayed no significant differences in central adiposity changes over 6 months  
23 between a control group and two groups exercising at doses resembling that recommended for

1 health (680 kcal/week) and for weight loss and weight loss maintenance (1521 kcal/week). We  
2 did observe significant reductions in total fat and adiposity in other non-central regions in the 20  
3 KKW group, which could provide metabolic benefits for individuals with overweight and  
4 obesity (34). Confidence intervals also indicated that the 20 KKW group demonstrated a  
5 reduction in some central adiposity indices (e.g., waist circumference and trunk fat), and  
6 between-group significance levels for trunk fat mass and trunk percent fat were close to the  
7 significance threshold. However, estimated marginal mean and effect size data show variations  
8 in central adiposity between groups are negligible or small at best. The 20 KKW group, for  
9 example, exhibited a 1.3 cm and 0.7 cm decrease in waist circumference compared to the control  
10 group and the 8 KKW group, respectively, and these differences are considered clinically  
11 unimportant (35,36) based on associations between waist circumference change and metabolic  
12 disease (37) and mortality (38). Thus, on balance, though relatively little levels of physical  
13 activity can improve central adiposity (10,39), we feel exercise doses that expended ~700  
14 kcal/week and ~1500 kcal/week induced clinically trivial changes in central adiposity during this  
15 trial. Doses with even greater differences in exercise-induced energy expenditure may be  
16 required to detect clinically meaningful improvements in central adiposity.

17 A reason why exercise groups exhibited negligible changes in central adiposity compared to  
18 control in our study could be the compensation displayed by exercise groups. The majority  
19 (82.6%) of exercisers exhibited positive weight compensation (i.e., lost less weight than  
20 expected) and these participants displayed a 2.5 cm and 0.23 kg increase in waist circumference  
21 and VAT, respectively, compared to those who did not show positive weight compensation.  
22 Although few have examined the link between compensation and changes in central adiposity,  
23 two studies in individuals with high waist circumference showed that aerobic exercise without

1 weight loss (i.e., with compensation) led to attenuated reductions in central adiposity relative to  
2 exercise with weight loss (i.e., without compensation) in males (15) and females (16), supporting  
3 our findings. Nonetheless, contrary to our results, these studies still found significant central  
4 adiposity improvements in exercisers who compensated (15,16). The fact that these earlier  
5 studies solely recruited individuals with high waist circumference is unlikely to explain why the  
6 previous studies saw improvements in central adiposity in individuals who compensated and we  
7 did not, as we found no exercise-induced differences in central adiposity change between those  
8 with high and healthy waist circumference at baseline. Rather, the discrepancies could occur  
9 because the previous studies were only 3 months and/or they implemented far greater exercise  
10 energy expenditures of 3,500 kcal/week in women (16) and 4,900 kcal/week in men (15). These  
11 findings could collectively indicate that exercise in the presence of significant weight  
12 compensation improves central adiposity during short interventions where exercise volumes are  
13 high, but not during medium-to-long term regimens where exercise performed is similar to that  
14 recommended for health and weight loss and weight loss maintenance.

15 By highlighting the compensatory mechanisms which predict central adiposity change during  
16 exercise, effective strategies can be developed to improve central adiposity outcomes during  
17 exercise. Several mechanisms could drive positive weight compensation during exercise training:  
18 an increase in energy intake, maladaptive changes in eating behaviors and physical activity  
19 patterns, and reductions in exercise and non-exercise energy expenditure (13). In this analysis, an  
20 increase in desire to eat positively predicted change in VAT. A similar association between  
21 change in VAT and compensatory health beliefs (e.g., justifying an eating episode because of  
22 exercise) was also observed, albeit the coefficient was smaller and tended to be significant.  
23 Along with findings from the primary outcome manuscript that showed exercise-induced



1 elevations in energy intake (13), these results may indicate that changes in eating habits  
2 attenuated reductions in central adiposity during exercise. This is in line with previous studies  
3 (15,16) and implies that strategies targeting desire to eat and compensatory behaviors during  
4 exercise could enhance central adiposity outcomes. Akin to other regimens (40), such strategies  
5 could include behavioral sessions that help manage appetite by encouraging participants to  
6 increase consumption of foods with low energy density (41). Additional strategies and sessions  
7 focussing on meal planning and portion and stimulus control could also decrease compensatory  
8 meals and/or snacking during exercise training (40). Nevertheless, it should be acknowledged  
9 that more work is needed to elucidate the role of compensatory behaviors in modifying central  
10 adiposity changes during exercise, since most compensatory behaviors (including energy intake)  
11 were not related to VAT and our models explained a small proportion of VAT variance.

12 A strength of the present analysis is that it comprises energy intake, energy expenditure, and  
13 physical activity outcomes assessed with gold-standard techniques, as well as questionnaires  
14 examining eating attitudes and behaviors. One limitation is that we did not use computed  
15 tomography or magnetic resonance imaging, which are considered gold standard tools for VAT  
16 assessment. Although VAT assessments via DXA are linked to computed tomography-derived  
17 measurements (42) and our primary findings were consistent amongst several indices related to  
18 VAT, future studies utilising computed tomography or magnetic resonance imaging are  
19 warranted. Additionally, since we did not assess potential physiological mediators, further  
20 studies are needed to examine the mechanisms underpinning our findings. Another limitation is  
21 that most of the sample were female and white, so it is possible we were underpowered to detect  
22 consistent and significant interactions in our subgroup analyses. It is also noteworthy that this  
23 manuscript reports an ancillary project, though it should be acknowledged that it utilized data

1 from a large randomized control trial where exercise adherence was excellent and exercise dose  
2 was fastidiously supervised and monitored.

3 Taken together, the present study indicates that in the presence of significant weight  
4 compensation, there are negligible differences in central adiposity change during aerobic  
5 exercise at doses similar to that recommended for health and weight loss and weight loss  
6 maintenance in individuals with overweight or obesity. Moreover, higher weight compensation  
7 was associated with reduced improvements in central adiposity, and exercisers with increased  
8 subjective desire to eat exhibited poorer change in central adiposity. During exercise at  
9 guidelines for health and weight loss and weight loss maintenance, exercise-induced  
10 compensation should be treated and reduced in individuals with overweight or obesity to  
11 enhance central adiposity reductions, potentially through strategies that manage appetite and  
12 compensatory food behaviors.

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### 18 **DATA AVAILABILITY**

19 Some or all datasets generated during and/or analyzed during the current study are not publicly  
20 available but are available from the corresponding author on reasonable request.

21

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8  
9 **Table 1.** Descriptive characteristics of participants included in the per protocol analysis at  
10 baseline.

	Control (N = 61)	8 KKW (N = 59)	20 KKW (N = 50)	All (N = 170)
Age (year)	49.5 (10.8)	48.3 (11.1)	48.5 (12.5)	48.8 (11.4)
Sex				
Male	16 (26.2)	16 (27.1)	15 (30.0)	47 (27.6)
Female	45 (73.8)	43 (72.9)	35 (70.0)	123 (72.4)
Race				
White	38 (62.3)	39 (66.1)	36 (72.0)	113 (66.5)
Black	21 (34.4)	20 (33.9)	12 (24.0)	53 (31.2)
Other	2 (3.3)	0 (0.0)	2 (4.0)	4 (2.4)
Income				
<\$30,000	10 (16.4)	8 (13.6)	3 (6.0)	21 (12.4)
\$30,000- \$49,999	9 (14.8)	7 (11.9)	7 (14.0)	23 (13.5)
\$50,000- \$79,999	15 (24.6)	13 (22.0)	15 (30.0)	43 (25.3)
\$80,000- \$99,999	12 (19.7)	10 (16.9)	9 (18.0)	31 (18.2)
≥\$100,000	14 (23.0)	20 (33.9)	15 (30.0)	49 (28.8)
Don't know or missing	1 (1.6)	1 (1.7)	1 (2.0)	3 (1.8)

11 Abbreviations: KKW, kcal/kg/week.

12 Continuous data are mean (SD); categorical data are number (%).

1 **Table 2.** Baseline values and 6-month change in weight, waist circumference, total adiposity, total lean mass, and regional adiposity in  
 2 the control group, 8 KKW group, and 20 KKW group.

		Control (N = 61)	8 KKW (N = 59)	20 KKW (N = 50)	<i>P</i>	Between-group ES		
						Control vs. 8 KKW	Control vs. 20 KKW	8 KKW vs. 20 KKW
Weight (kg)								
	Baseline	90.1 (86.2, 94.0)	88.7 (84.8, 92.7)	86.7 (82.4, 91.0)				
	Change	-0.3 (-1.0, 0.4)	-0.5 (-1.3, 0.2)	-1.8 (-2.6, -0.9) <sup>†</sup>	0.02*	0.07	0.51	0.43
BMI (kg/m <sup>2</sup> )								
	Baseline	32.3 (31.1, 33.5)	31.4 (30.2, 32.6)	30.6 (29.3, 31.9)				
	Change	-0.1 (-0.4, 0.2)	-0.2 (-0.5, 0.1)	-0.6 (-0.9, -0.4) <sup>†</sup>	0.02*	0.10	0.53	0.43
Waist circumference (cm)								
	Baseline	101.1 (98.0, 104.3)	98.5 (95.3, 101.7)	97.0 (93.6, 100.5)				
	Change	0.0 (-1.0, 1.0)	-0.7 (-1.7, 0.4)	-1.3 (-2.4, -0.2)	0.23	0.15	0.33	0.17
Total fat mass (kg)								
	Baseline	38.9 (36.3, 41.4)	37.0 (34.5, 39.6)	36.3 (33.5, 39.0)				
	Change	0.0 (-0.6, 0.7)	-0.3 (-1.0, 0.4)	-1.4 (-2.2, -0.7) <sup>‡</sup>	0.01*	0.12	0.57	0.44
Total lean mass (kg)								
	Baseline	48.2 (45.7, 50.8)	48.8 (46.2, 51.4)	47.4 (44.6, 50.3)				
	Change	-0.4 (-0.7, -0.1)	-0.2 (-0.6, 0.1)	-0.1 (-0.5, 0.2)	0.51	0.13	0.22	0.09
Total body fat %								
	Baseline	43.2 (41.3, 45.0)	41.6 (39.7, 43.5)	41.8 (39.8, 43.8)				

VAT (kg)	Change	0.1 (-0.4, 0.6)	-0.1 (-0.6, 0.4)	-0.9 (-1.5, -0.4) <sup>‡</sup>	0.02*	0.09	0.52	0.44
	Baseline	1.40 (1.17, 1.63)	1.25 (1.01, 1.48)	1.30 (1.04, 1.55)				
VAT-to-total-fat ratio	Change	-0.02 (-0.07, 0.04)	-0.01 (-0.07, 0.04)	-0.04 (-0.10, 0.02)	0.71	0.02	0.13	0.15
	Baseline	0.0361 (0.0302, 0.0420)	0.0336 (0.0276, 0.0396)	0.0363 (0.0298, 0.0428)				
Trunk fat mass (kg)	Change	-0.0008 (-0.0016, 0.0001)	-0.0002 (-0.0011, 0.0007)	-0.0004 (-0.0014, 0.0006)	0.65	0.17	0.11	0.06
	Baseline	21.1 (19.6, 22.7)	20.0 (18.4, 21.6)	19.3 (17.5, 21.0)				
Trunk fat %	Change	0.0 (-0.5, 0.4)	-0.3 (-0.8, 0.2)	-0.8 (-1.3, -0.3)	0.07	0.14	0.43	0.29
	Baseline	46.9 (45.0, 48.7)	45.0 (43.2, 46.9)	44.8 (42.7, 46.8)				
Arms fat mass (kg)	Change	-0.1 (-0.7, 0.5)	-0.2 (-0.8, 0.4)	-1.1 (-1.8, -0.4)	0.06	0.04	0.42	0.38
	Baseline	4.01 (3.72, 4.30)	3.84 (3.54, 4.14)	3.60 (3.28, 3.93)				
Arms fat %	Change	0.06 (-0.02, 0.15)	0.06 (-0.03, 0.15)	-0.07 (-0.17, 0.02)	0.06	0.01	0.41	0.39
	Baseline	39.9 (37.5, 42.3)	38.5 (36.1, 40.9)	38.4 (35.7, 41.0)				
Legs fat mass (kg)	Change	0.7 (0.2, 1.1)	0.7 (0.2, 1.1)	-0.1 (-0.5, 0.4)	0.04*	0.01	0.42	0.43
	Baseline	12.74 (11.70, 13.77)	12.19 (11.14, 13.24)	12.42 (11.28, 13.57)				
	Change	-0.01 (-0.28, 0.26)	-0.05 (-0.33, 0.22)	-0.54 (-0.83, -0.24) <sup>‡</sup>	0.02*	0.04	0.50	0.45

Legs fat %	Baseline	42.0 (39.6, 44.3)	41.0 (38.6, 43.3)	41.6 (39.0, 44.1)				
	Change	0.3 (-0.2, 0.7)	-0.2 (-0.7, 0.2)	-1.0 (-1.5, -0.5) <sup>‡</sup>	<0.01*	0.26	0.72	0.46
Trunk-fat-to-limb-fat ratio	Baseline	1.32 (1.22, 1.42)	1.28 (1.18, 1.38)	1.27 (1.16, 1.38)				
	Change	-0.03 (-0.06, 0.00)	-0.02 (-0.05, 0.01)	-0.02 (-0.05, 0.02)	0.90	0.06	0.08	0.03

1 Abbreviations: BMI, body mass index; ES, effect size; KKW, kcal/kg/week; VAT, visceral adipose tissue.

2 Data are estimated marginal mean (95% CI). Change values are adjusted for age and sex.

3 \*Significant ANCOVA omnibus test for study group ( $P < 0.05$ ).

4 <sup>†</sup>Significantly different from control group ( $P < 0.05$ ).

5 <sup>‡</sup>Significantly different from control group and 8 KKW group ( $P < 0.05$ ).

6

1 **Table 3.** Multiple linear regression analysis for association between retrospective desire, eat and  
 2 compensatory health beliefs and change in VAT.

	R <sup>2</sup>	B	95% CI	$\beta$	P
VAT (kg)	0.115				
Retrospective VAS, Desire to eat		0.0023	(0.0002, 0.0045)	0.21	0.03*
Compensatory health		0.0047	(-0.0007, 0.0101)	0.16	0.09
Age		0.0011	(-0.0023, 0.0046)	0.07	0.51
Sex <sup>†</sup>		-0.0868	(-0.1939, 0.0202)	-0.20	0.11
VAT at baseline		-0.0001	(-0.0001, 0.0000)	-0.23	0.07

3 Abbreviations: VAS, visual analogue scale; VAT, visceral adipose tissue.

4 \*Statistically significant ( $P < 0.05$ ).

5 <sup>†</sup>Male = 1, female = 2.

#### 6 **Figure legends**

7 **Figure 1.** Change in waist circumference (A), VAT (B), VAT-to-total-fat ratio (C), and trunk-  
 8 fat-to-limb-fat ratio (D) in individuals who displayed zero or negative weight compensation (i.e.,  
 9 those who lost more or equal weight to that which was predicted) (N = 19) and individuals who  
 10 displayed positive weight compensation (i.e., those who lost less weight than predicted) (N = 90)  
 11 during exercise. Abbreviations: VAT, visceral adipose tissue. Black bars are individuals who  
 12 showed zero or negative compensation; white bars are individuals who showed positive  
 13 compensation. Data are estimated marginal mean (95% CI). \*Significant ANCOVA omnibus  
 14 comparison between those who displayed zero or negative compensation and those who  
 15 displayed positive compensation ( $P < 0.05$ ).

16

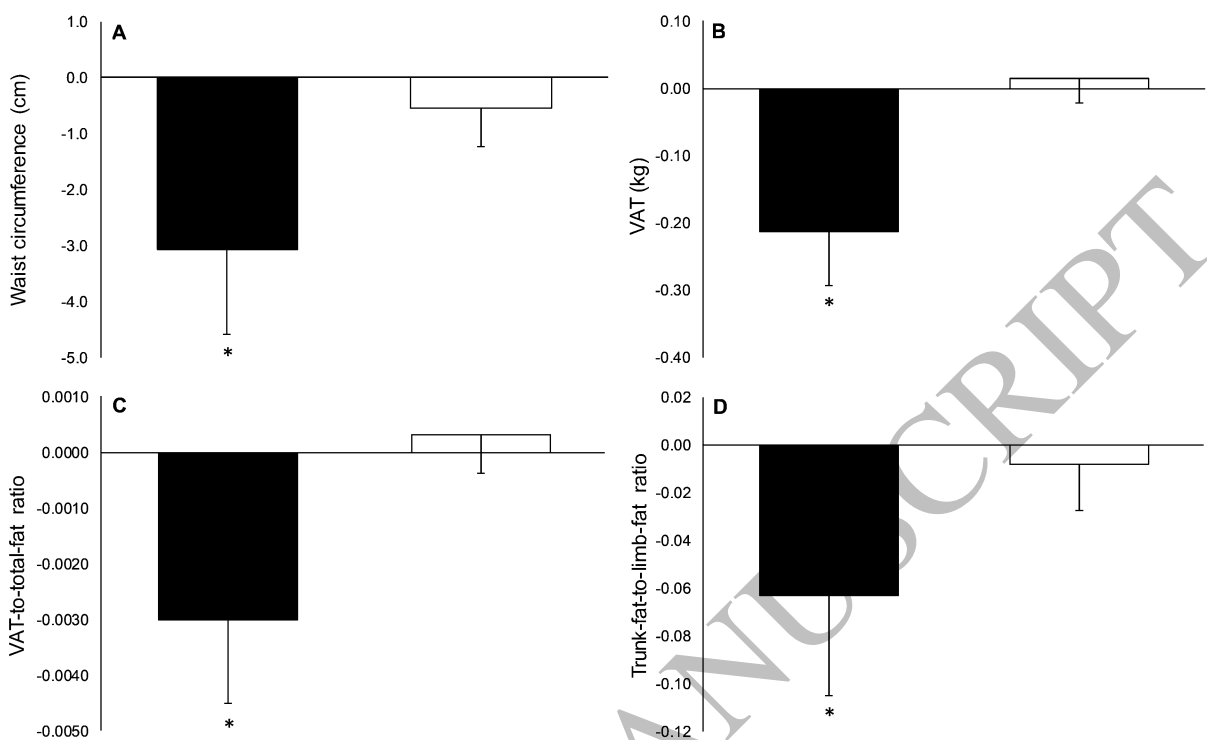


Figure 1  
257x159 mm (x DPI)

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