Exercise Training Reduces Reward for High-Fat Food in Adults with Overweight/Obesity

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ABSTRACT

BEAULIEU, K., M. HOPKINS, C. GIBBONS, P. OUSTRIC, P. CAUDWELL, J. BLUNDELL, and G. FINLAYSON. Exercise Training Reduces Reward for High-Fat Food in Adults with Overweight/Obesity. Med. Sci. Sports Exerc., Vol. 52, No. 4, pp. 900–908, 2020. Purpose: There is increasing evidence that exercise training may facilitate weight management via improvements in homeostatic appetite control, but little is known about how exercise training affects food reward and susceptibility to overeating. Methods: This study examined changes in food reward and eating behavior traits after a supervised 12-wk exercise intervention (10.5 MJ·wk−1) in inactive individuals with overweight/obesity (exercisers; n = 46, 16 men/30 women; mean (SD) body mass index, 30.6 (3.8) kg·m−2; and mean (SD) age, 43.2 (7.5) yr) compared with nonexercising controls (n = 15; 6 men/9 women; mean (SD) body mass index, 31.4 (3.7) kg·m−2; and mean (SD) age, 41.4 (10.7) yr). Liking and wanting scores for high-fat relative to low-fat foods were assessed with the Leeds Food Preference Questionnaire before and after consumption of an isoenergetic high-fat or high-carbohydrate lunch. Eating behavior traits were assessed using the Three-Factor Eating Questionnaire and Binge Eating Scale. Results: A week–group interaction indicated that wanting scores decreased from baseline to postintervention in exercisers only (MPre_post = −4.1, P = 0.03, ηp² = 0.09, 95% confidence interval [CI], −7.8 to −0.4), but there was no exercise effect on liking. There was also a week–group interaction for binge eating, which decreased in exercisers only (MPre_post = −1.5, P = 0.01, ηp² = 0.11; 95% CI, −2.7 to −0.4). A small reduction in disinhibition was also apparent in exercisers (MPre_post = −0.7, P = 0.02, ηp² = 0.10; 95% CI, −1.3 to −0.1). Conclusions: This study showed that 12 wk of exercise training reduced wanting scores for high-fat foods and trait markers of overeating in individuals with overweight/obesity compared with nonexercising controls. Further research is needed to elucidate the mechanisms behind these exercise-induced changes in food reward. Key Words: FOOD REWARD, EATING BEHAVIOR TRAITS, APPETITE CONTROL, LIKING AND WANTING, PHYSICAL ACTIVITY

Physical activity is widely recommended as a strategy for weight management, and exercise interventions improve body composition in both men and women (1). In addition to potential effects on body weight via increased energy expenditure, it is becoming apparent that habitual physical activity and exercise training improve markers of appetite control, such as increased satiety response to food and gastric emptying (2,3). However, variability in the interindividual weight loss response to exercise interventions has been reported (4). This variability suggests that some individuals may compensate for an increase in physical activity (and energy expenditure) through changes in meal size, frequency, or food choice, attenuating or even reversing the effect of exercise on weight loss.

Liking and wanting components of food reward may be heightened for palatable food in individuals with overweight and obesity compared with individuals who are lean (5). Food reward is also potentially influenced by physical activity, but evidence has been inconsistent, and as highlighted by a recent systematic review on weight management interventions (6), findings to date offer limited evidence for the effect of exercise interventions on food reward. We have shown that an acute postexercise increase (both at baseline and postintervention) in food liking and wanting (particularly of high-fat foods), was present in those with a less-than-expected reduction in body weight during a 12-wk exercise intervention (7). No overall changes in food reward in individuals with obesity were found after 12 wk of moderate continuous or high-intensity interval training (8); however, we have previously
reported a trend for a decrease in implicit wanting measured in the hungry state in response to 12 wk of structured exercise training (9). How meal consumption or macronutrient composition influences these responses has yet to be explored.

In terms of eating behavior traits, studies have shown that with exercise-induced weight loss, greater changes in restraint were associated with greater weight loss (10). Exercise training has also been shown to decrease disinhibition in individuals with overweight and obesity (10). A recent systematic review suggested that physical activity may reduce binge eating through potential effects of physical activity on the reward system as they may share similar brain pathways (11). Other proposed mechanisms include changes in negative affect, homeostatic appetite control, and/or body composition (11).

Few studies have assessed the effect of exercise training on food reward and eating behaviors together. One study found reductions in the neuronal responses to visual food cues using functional magnetic resonance imaging but no changes in restraint or disinhibition after a 6-month exercise intervention (12). Whether changes in eating behaviors are associated with changes in food reward in response to exercise remains to be elucidated.

Overeating traits and food reward states interact with the fat content of food with the potential to enhance or undermine appetite control (13). Disinhibition and binge eating have both been linked to greater intake and preference for high-fat or high-fat/sweet foods (14). Indeed, we have previously shown that food reward was reduced after consumption of a fixed-energy low-fat meal but not after an energy-matched high-fat meal (15). Whether exercise training interacts with the fat content of the diet has gained interest in recent years and has relevance for weight management (2).

Therefore, the main objective of this study was to investigate the effect of a supervised 12-wk exercise intervention on food reward and eating behavior traits in inactive individuals with overweight and obesity compared with nonexercising controls. This was examined during exposure to high-fat (HFAT) and high-carbohydrate (HCHO) feeding conditions. A secondary aim was to examine relationships among changes in eating behavior traits, food reward, and body composition.

METHODS

Participants

Men and women with overweight and obesity age 18–55 yr were recruited via poster advertisements and e-mail lists at the University of Leeds, United Kingdom, and surrounding areas. Participants were screened on the following inclusion criteria: body mass index (BMI) between 26.0 and 38.0 kg·m−2, non-smoker, inactive (≤2 h·wk−1 of exercise over the previous 6 months), weight stable (±2 kg for previous 3 months), not currently dieting or participating in a weight loss regime, no history of eating disorders, not taking any medication known to affect metabolism or appetite, and acceptance of the study foods. Participants were asked to keep lifestyle habits and activities constant throughout the study. The study was approved by the Leeds West NHS Research Ethics Committee (09/H1307/7). Participants provided written informed consent before taking part. The study was registered under international standard trials approval (ISRCTN47291569).

Study Design

Forty-six participants (exercisers; 16 men/30 women, mean [SD] age, 43.2 [7.5] yr) completed a 12-wk exercise intervention in which they exercised 5 d·wk−1 under supervision of research staff in the Human Appetite Research Unit, University of Leeds, United Kingdom, between November 2011 and July 2013. Aspects of these data have been previously reported (9,15–17).

Fifteen control participants (controls; 6 men/9 women, mean [SD] age, 41.4 [10.7] yr) completed 12 wk of maintaining current low activity levels between July 2012 and July 2013. The nonexercising controls were not made aware of the exercise arm of the study; participants were requested not to change their dietary or exercise patterns for the duration of the study.

At baseline and postintervention, food reward and eating behavior variables were assessed (described hereinafter). Food reward was measured during HFAT and HCHO probe days before and after a fixed-energy meal. These days were separated by at least 1 d and in a randomized crossover order. Before each laboratory session, participants were instructed to maintain their usual diet, not to engage in physical activity for 24 h, refrain from consuming alcohol for 24 h, and fast overnight (10–12 h).

Measurements

Anthropometrics and body composition. At baseline and postintervention, participants completed a measurement day. Anthropometrics and body composition measures were taken while participants were wearing tight-fitting clothing and a swim cap. Standing height without shoes was measured to the nearest 0.1 cm using a stadiometer (Leicester height measure, SECA, United Kingdom). Body weight was measured using an electronic balance and recorded to the nearest 0.1 kg (BodPod; Life Measurement, Inc., Concord, CA). Fat mass, fat-free mass, and percentage body fat were estimated via air displacement plethysmography (BodPod) following the manufacturer’s instructions.

Eating behavior questionnaires. Psychometric questionnaires were completed after the body composition measures. The Three-Factor Eating Questionnaire is a validated 51-item instrument that measures three dimensions of eating behavior: cognitive control of restraint (i.e., concern over weight gain and the strategies adopted to prevent this), disinhibition of eating (i.e., tendency of an individual to overeat and to eat opportunistically in the obesogenic environment), and susceptibility to hunger (i.e., extent to which feelings of hunger are perceived and how these sensations result in food intake) (18). The Binge Eating Scale is a validated 16-item questionnaire that assesses the severity of binge eating (19). The questions are based on both behavioral characteristics (e.g., amount of food consumed) and the emotional, cognitive response (e.g., guilt or shame).
Test meals. At baseline and postintervention, participants completed two separate probe meal days in which they consumed foods that were either HFAT or HCHO. The ingredients of the foods provided during the meal days were covertly manipulated to be as follows: 10.4 (SD, 1.1) kJ·g⁻¹ for HFAT, 10.6 (SD, 0.8) kJ·g⁻¹ for HCHO. The energy in the foods consumed during the meal days were consumed foods that were either HFAT or HCHO. The ingredients of the foods provided during the meal days were covertly manipulated to be as follows: 10.4 (SD, 1.1) kJ·g⁻¹, 37.7% carbohydrate, 54.4% fat, and 7.9% protein for HFAT, or 6.6 (SD, 0.8) kJ·g⁻¹, 72.4% carbohydrate, 19.3% fat, and 8.3% protein for HCHO. Four hours after a standardized breakfast (ad libitum) on baseline probe day and quantities consumed replicated on postintervention probe day; see meal details in Table, Supplemental Digital Content 1, Food items and macronutrient composition of the ad libitum HFAT and HCHO breakfast at baseline, http://links.lww.com/MSS/B826, the participants consumed a fixed-energy lunch composed of food items providing 3347 kJ (matched for weight across HFAT and HCHO; see meal details in Table, Supplemental Digital Content 2, Food items and macronutrient composition of the fixed energy HFAT and HCHO lunches, http://links.lww.com/MSS/B827). Foods were designed to be similar in appearance and palatability between conditions.

Food reward. The Leeds Food Preference Questionnaire (LFPQ; [20]) was administered during the HFAT and HCHO meal days before and after fixed-lunch consumption (3347 kJ) to assess food reward behaviors (liking and wanting) in the hungry and fed states. The LFPQ computes scores of implicit wanting and explicit liking for high-fat (>50% energy) and low-fat (<20% energy) foods images matched for familiarity, sweetness, protein, and acceptability.

Before the procedure, screening of the images used in the task was completed by each participant to improve internal validity. If a participant did not know or recognize, or would never/rarely eat a particular food item used in the study, replacement images were chosen from a database of images of similar composition.

The LFPQ is composed of two tasks: one based on subjective ratings (explicit liking) and the other based on a forced-choice task (implicit wanting). During the measure of explicit liking, participants were presented with one food image at a time, in a randomized order, and rated the extent to which they like each food (How pleasant would it be to taste this food now?). Participants made their ratings using a 100-mm visual analog scale. Implicit wanting was assessed by asking participants to select as fast as possible between successive pairs of foods from specific categories the food “they most want to eat now.” Scores for implicit wanting were computed from mean response times adjusted for frequency (21). To calculate liking and wanting fat appeal bias as a measure of hedonic preference for high-fat relative to low-fat foods, low-fat scores were subtracted from high-fat scores; thus, a positive score indicates greater explicit liking/implicit wanting toward high-fat compared with low-fat foods. The LFPQ has been validated in a wide range of research (e.g., Ref. 22).

Twelve-Week Exercise Intervention

During the 12-wk supervised exercise intervention (5 d·wk⁻¹), each exercise session was individually prescribed to expend 2092 kJ at an intensity of 70% of age-predicted heart rate maximum, and to ensure compliance to the exercise prescription, the duration and intensity of each exercise session were recorded (Polar RS400, Polar, Finland). A selection of aerobic exercise equipment was available (i.e., treadmill, rower, cycle ergometer, and elliptical) from which the participants were free to choose and change within each session as long as they met the energy expenditure requirements. The duration needed to expend 2092 kJ at 70% heart rate maximum at baseline was calculated based on the relationship between heart rate, VO₂, and VCO₂ for each individual during an incremental maximal aerobic capacity (VO₂max) test. This test was also performed at week 6 of the intervention to account for changes in energy metabolism and after the intervention to assess overall changes in cardiorespiratory fitness with the intervention. Total exercise-induced energy expenditure during the intervention was 116.98 ± 15.56 MJ, which represented >98% of the prescribed exercise-induced energy expenditure. VO₂max was not measured in the controls.

Statistical Analyses

Data are presented as mean (SD), unless specified otherwise. Data were analyzed using the statistical package SPSS version 21. Data were checked for outliers before statistical analyses, and one of the controls had a change score (baseline to postintervention) in both liking and wanting that was 5 SD below the mean; therefore, this participant was excluded from the analysis. Independent-sample t-tests were used to evaluate differences in participant characteristics at baseline. Repeated-measures ANOVA with group (exercisers, controls), week (baseline, postintervention), condition (HFAT, HCHO), and state (hungry, fed), where appropriate, were used to assess changes in outcome variables. Where appropriate, Greenhouse-Geisser probability levels were used to adjust for nonsphericity, and post hoc analyses were performed using the Bonferroni adjustment for multiple comparisons. Where missing data were present, completers and intent-to-treat analyses (ITT) were conducted with the last observation carried forward method. To assess the associations among changes in food reward (overall mean of the two conditions and two states), eating behavior traits and body composition, Pearson’s correlations were conducted in the whole group and in exercisers and controls separately.

The magnitude of the mean weight change (exercisers–controls) was interpreted against a minimal clinically important difference of 2.5 kg (23), where a small clinically important effect was defined between 2.5 and 7.5 kg (3 ± 2.5 kg), a moderate effect between 7.5 and 15 kg (6 ± 2.5 kg), and a large effect >15 kg (24). The magnitude of the mean waist circumference change was interpreted against a minimal clinically important difference of 2 cm (4,25), where a small clinically important effect was defined between 2 and 6 cm, a moderate effect between 6 and 9 cm, and a large effect >9 cm (24).

Following the American Statistical Association’s policy statement on P values (26), all P values from specified statistical models were reported along with effect size and confidence.
RESULTS

Participant characteristics. Participant characteristics at baseline and postintervention are described in Table 1. Baseline characteristics of exercisers and controls were similar (P > 0.13 for all). There were interactions between week and group for BMI, total mass, body fat percentage, fat mass, and waist circumference (all, P < 0.001, η² ≥ 0.19). The week–group interaction for fat-free mass was weaker (P = 0.22, η² = 0.03).

In exercisers, the training intervention led to reductions in BMI (ΔMPre–Post = −0.6 kg·m⁻², P < 0.001, η² = 0.25; 95% CI, −0.9 to −0.3 kg·m⁻²), total mass (ΔMPre–Post = −1.8 kg, P < 0.001, η² = 0.27; 95% CI, −2.6 to −1.0 kg), body fat percentage (ΔMPre–Post = −1.9%, P < 0.001, η² = 0.42; 95% CI, −2.5 to −1.3%), fat mass (ΔMPre–Post = −2.2 kg, P < 0.001, η² = 0.37; 95% CI, −3.0 to −1.5 kg), and waist circumference (ΔMPre–Post = −3.7 cm, P < 0.001, η² = 0.57; 95% CI, −4.5 to −2.9 cm). There were also increases in fat-free mass (ΔMPre–Post = 0.4 kg, P = 0.01, η² = 0.10; 95% CI, 0.1 to 0.8 kg) and VO₂max (ΔMPre–Post = 5.7 mL·kg⁻¹·min⁻¹, P < 0.001, η² = 0.43; 95% CI, 3.7 to 7.6 mL·kg⁻¹·min⁻¹; not measured in controls).

In controls, there were increases in waist circumference (ΔMPre–Post = 0.4 kg·m⁻², P = 0.08, η² = 0.05; 95% CI, −0.1 to 0.9 kg·m⁻²), total mass (ΔMPre–Post = 1.3 kg, P = 0.06, η² = 0.06; 95% CI, −0.04 to 2.7 kg), body fat percentage (ΔMPre–Post = 0.8%, P = 0.15, η² = 0.04; 95% CI, −0.3% to 1.8%); fat mass (ΔMPre–Post = 1.3 kg, P = 0.06, η² = 0.06; 95% CI, −0.1 to 2.6 kg), and waist circumference (ΔMPre–Post = 2.1 cm, P = 0.005, η² = 0.12; 95% CI, 0.6 to 3.6 cm).

The 12-wk intervention produced a mean group (exercisers–controls) body weight difference of −3.1 kg (95% CI, −4.3 to −1.9 kg) and waist circumference difference of −5.8 cm (95% CI, −7.5 to −4.1 cm).

Food reward. Completers data were available in 38 exercisers and 14 controls (with the outlier removed).

For liking scores, exercisers had a lower liking than did controls overall (ΔMEx-C = −6.0 mm, P = 0.15, η² = 0.04 (95% CI, −14.2 to 2.2 mm); ITT: ΔMEx-C = −7.6 mm, P = 0.06, η² = 0.06 (95% CI, −15.5 to 0.2 mm)). A week–group interaction was not apparent (P = 0.75, η² = 0.002; ITT: P = 0.87, η² = 0.00), and there were no changes from baseline to postintervention in both groups (ΔMPre–Post = −1.1 mm, P = 0.24, η² = 0.03 (95% CI, −3.0 to 0.79 mm); ITT: ΔMPre–Post = −1.3 mm, P = 0.15, η² = 0.04 (95% CI, −3.1 to 0.5 mm)). Figure 1A shows only the main effect of the intervention on liking within each group, for clarity.

On comparison across test meal conditions (main effect of HFAT vs HCHO), liking was greater in HFAT relative to HCHO (ΔMHF-HC = 1.9 mm, P = 0.06, η² = 0.07 (95% CI, −0.1 to 3.9 mm); ITT: ΔMHF-HC = 1.9 mm, P = 0.05, η² = 0.06 (95% CI, −0.02 to 3.7 mm)). The interaction effect between condition and state (P = 0.02, η² = 0.11; ITT: P = 0.04, η² = 0.07) showed minimal differences in liking between HFAT and HCHO in the hungry state (ΔMHF-HC = 0.3 mm, P = 0.81, η² = 0.001 (95% CI, −2.2 to 2.8 mm); ITT: ΔMHF-HC = 0.4 mm, P = 0.73, η² = 0.002 (95% CI, −2.1 to 2.9 mm)), but liking was greater in HFAT relative to HCHO in the fed state (ΔMHF-HC = 3.5 mm, P = 0.003, η² = 0.17 (95% CI, 1.3 to 5.7 mm); ITT: ΔMHF-HC = 3.3 mm, P = 0.003, η² = 0.14 (95% CI, 1.2 to 5.4 mm)). There were no other apparent effects or interactions.

For wanting scores, exercisers had lower wanting than did controls overall (ΔMEx-C = −11.1, P = 0.17, η² = 0.04 (95% CI, −27.2 to 4.9); ITT: ΔMEx-C = −15.4, P = 0.06, η² = 0.06 (95% CI, −31.3 to 0.5)). The week–group interaction effect (P = 0.08, η² = 0.06; ITT: P = 0.06, η² = 0.06) showed that exercisers reduced wanting from baseline to postintervention (ΔMPre–Post = −4.1, P = 0.03, η² = 0.09 (95% CI, −7.8 to 0.3)).

TABLE 1. Characteristics of exercisers and controls at baseline and postintervention.

<table>
<thead>
<tr>
<th></th>
<th>Exercisers (n = 46)</th>
<th>Controls (n = 15)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Postintervention</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>BMI, kg·m⁻²</td>
<td>30.5</td>
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<tr>
<td>Total mass, kg</td>
<td>87.6</td>
<td>14.3</td>
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<tr>
<td>Body fat, %</td>
<td>40.0</td>
<td>7.6</td>
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<tr>
<td>Fat mass, kg</td>
<td>35.1</td>
<td>9.2</td>
</tr>
<tr>
<td>Fat-free mass, kg</td>
<td>52.5</td>
<td>10.4</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>101.6</td>
<td>10.6</td>
</tr>
</tbody>
</table>

*Within-group post hoc analyses: P < 0.001.
†Within-group post hoc analyses: P < 0.01.
RMR, resting metabolic rate. Repeated-measures ANOVA was conducted with post hoc Bonferroni adjustments.
Repeated-measures ANOVA was conducted with post hoc Bonferroni adjustments. *Exercisers baseline vs postintervention (completers: \( \eta^2_p = 0.09; \) ITT: \( \eta^2_p = 0.10 \)). †Exercisers vs controls postintervention (completers: \( \eta^2_p = 0.09; \) ITT: \( \eta^2_p = 0.06; \) ITT: \( \eta^2_p = 0.09 \)).

The week–state interaction effect \( (p = 0.04, \eta^2_p = 0.08; \) ITT: \( p = 0.06, \eta^2_p = 0.06 \)) suggested reductions in wanting from hungry to fed postintervention \( (M_{\text{AH-Fed}} = -5.3, P = 0.10, \eta^2_p = 0.05 (95\% \text{ CI}, -1.17 to 1.00); \) ITT: \( M_{\text{AH-Fed}} = -5.3, P = 0.08, \eta^2_p = 0.05 (95\% \text{ CI}, -1.13 to 0.7) \) and from baseline to postintervention in the fed state \( (M_{\text{APre-Post}} = -3.3, P = 0.10, \eta^2_p = 0.05 (95\% \text{ CI}, -7.2 to 0.6); \) ITT: \( M_{\text{APre-Post}} = -3.2, P = 0.10, \eta^2_p = 0.05 (95\% \text{ CI}, -7.1 to 0.6) \)).

The main effect of condition showed that wanting was greater in HFAT relative to HCHO \( (M_{\text{AH-HC}} = 3.0, P = 0.03, \eta^2_p = 0.09 (95\% \text{ CI}, 0.2 to 5.8); \) ITT: \( M_{\text{AH-HC}} = 2.9, P = 0.03, \eta^2_p = 0.08 (95\% \text{ CI}, 0.3 to 5.4) \)). The interaction effect between condition, state, and group \( (p = 0.08, \eta^2_p = 0.06; \) ITT: \( p = 0.09, \eta^2_p = 0.05 \)) suggested lower wanting in exercisers than in controls when hungry in HFAT \( (M_{\text{EX-C}} = -13.2, P = 0.11, \eta^2_p = 0.05 (95\% \text{ CI}, -29.6 to 3.3); \) ITT: \( M_{\text{EX-C}} = -17.5, P = 0.04, \eta^2_p = 0.07 (95\% \text{ CI}, -34.1 to 0.9)) \) and HCHO \( (M_{\text{EX-C}} = -13.7, P = 0.12, \eta^2_p = 0.05 (95\% \text{ CI}, -31.2 to 3.8); \) ITT: \( M_{\text{EX-C}} = -17.7, P = 0.05, \eta^2_p = 0.07 (95\% \text{ CI}, -35.2 to 0.1)) \), and when fed in HFAT \( (M_{\text{EX-C}} = -12.0, P = 0.17, \eta^2_p = 0.04 (95\% \text{ CI}, -29.5 to 5.5); \) ITT: \( M_{\text{EX-C}} = -16.5, P = 0.06, \eta^2_p = 0.06 (95\% \text{ CI}, -33.7 to 0.6)) \). Controls also had greater wanting after HFAT compared with HCHO in the fed state \( (M_{\text{AH-HC}} = 7.3, P = 0.01, \eta^2_p = 0.12 (95\% \text{ CI}, 1.6 to 13.1); \) ITT: \( M_{\text{AH-HC}} = 7.3, P = 0.009, \eta^2_p = 0.11 (95\% \text{ CI}, 1.9 to 12.8)) \). There were no other apparent effects or interactions.

**Eating behavior traits.** Completers’ data were available for 46 exercisers (45 for binge eating) and 12 controls, and ITT for 14 controls. As shown in Table 2, baseline scores for exercisers and controls were similar \( (P > 0.15 \text{ for all}) \).

For restraint, there were minimal differences from baseline to postintervention across groups \( (M_{\text{APre-Post}} = -0.5, P = 0.37, \) 

<p>| TABLE 2. Eating behavior traits in exercisers and controls at baseline and postintervention. |
|-----------------------------------------------|----------------|----------------|----------------|
|                                             | Exercisers (n = 46) | Controls (n = 12) |</p>
<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Postintervention</th>
<th>Baseline</th>
<th>Postintervention</th>
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<tr>
<td><strong>Restraint</strong></td>
<td>7.5</td>
<td>7.1</td>
<td>8.8</td>
<td>8.2</td>
</tr>
<tr>
<td><strong>Disinhibition</strong></td>
<td>8.2</td>
<td>7.5*</td>
<td>8.4</td>
<td>8.5</td>
</tr>
<tr>
<td><strong>SUSCEPTIBILITY TO HUNGER</strong></td>
<td>5.1</td>
<td>4.9</td>
<td>6.3</td>
<td>6.8</td>
</tr>
<tr>
<td><strong>Binge eating score</strong></td>
<td>11.9</td>
<td>10.4</td>
<td>12.5</td>
<td>13.3</td>
</tr>
</tbody>
</table>

Repeated-measures ANOVA was conducted with post hoc Bonferroni adjustments.

*Exercisers, n = 45.

**Within-group post hoc analyses:** \( P < 0.05 \).
than did controls overall (\(P = 0.38, \eta^2 = 0.01\) (95% CI, −1.4 to 0.5)); and between groups (\(M_{\text{Ex-C}} = -1.2, P = 0.32, \eta^2 = 0.02\) (95% CI, −3.6 to 1.2); ITT: \(M_{\text{Ex-C}} = -1.2, P = 0.31, \eta^2 = 0.02\) (95% CI, −3.5 to 1.1)), and no apparent week–group interaction (\(P = 0.89, \eta^2 = 0.00\); ITT: \(P = 0.94, \eta^2 = 0.00\)).

For disinhibition, there were minimal differences between groups (\(M_{\text{Ex-C}} = -0.6, P = 0.60, \eta^2 = 0.005\) (95% CI, −2.8 to 1.6); ITT: \(M_{\text{Ex-C}} = -0.9, P = 0.40, \eta^2 = 0.01\) (95% CI, −2.9 to 1.2). The interaction effect between week and group (\(P = 0.23, \eta^2 = 0.03\); ITT: \(P = 0.20, \eta^2 = 0.03\)) suggested a decrease in disinhibition from baseline to postintervention in exercisers (\(M_{\text{Pre-Post}} = -0.7, P = 0.02, \eta^2 = 0.10\) (95% CI, −1.3 to −0.1); ITT: \(M_{\text{Pre-Post}} = -0.7, P = 0.01, \eta^2 = 0.10\) (95% CI, −1.2 to −0.1)) but not in controls (\(M_{\text{Pre-Post}} = 0.4, P = 0.94, \eta^2 = 0.0\) (95% CI, −1.1 to 1.1); ITT: \(M_{\text{Pre-Post}} = 0.4, P = 0.94, \eta^2 = 0.0\) (95% CI, −1.0 to 1.0)).

For susceptibility to hunger, exercisers had lower scores than did controls overall (\(M_{\text{Ex-C}} = -1.5, P = 0.18, \eta^2 = 0.03\) (95% CI, −3.7 to 0.7); ITT: \(M_{\text{Ex-C}} = -1.9, P = 0.07, \eta^2 = 0.06\) (95% CI, −4.0 to 0.1)). The week–group interaction (\(P = 0.33, \eta^2 = 0.02\); ITT: \(P = 0.35, \eta^2 = 0.02\)) suggested that exercisers had lower scores than did controls postintervention (\(M_{\text{Ex-C}} = -1.9, P = 0.11, \eta^2 = 0.04\) (95% CI, −4.2 to 0.5); ITT: \(M_{\text{Ex-C}} = -2.2, P = 0.04, \eta^2 = 0.07\) (95% CI, −4.4 to −0.01)).

For binge eating score, differences between groups were minimal (\(M_{\text{Ex-C}} = -1.8, P = 0.46, \eta^2 = 0.01\) (95% CI, −6.4 to 2.9); ITT: \(M_{\text{Ex-C}} = -2.5, P = 0.25, \eta^2 = 0.02\) (95% CI, −6.9 to 1.8). The interaction between week and group (\(P = 0.06, \eta^2 = 0.06\); ITT: \(P = 0.06, \eta^2 = 0.06\)) revealed a decrease in exercisers (\(M_{\text{Pre-Post}} = -1.5, P = 0.01, \eta^2 = 0.11\) (95% CI, −2.7 to −0.4); ITT: \(M_{\text{Pre-Post}} = -1.5, P = 0.01, \eta^2 = 0.11\) (95% CI, −2.6 to −0.4)) but not in controls (\(M_{\text{Pre-Post}} = 0.9, P = 0.44, \eta^2 = 0.01\) (95% CI, −1.4 to 3.1); ITT: \(M_{\text{Pre-Post}} = 0.8, P = 0.46, \eta^2 = 0.009\) (95% CI, −1.3 to 2.8)).

**Relationship between changes in food reward, eating behavior traits, and body weight and composition.** In the whole sample and in controls, changes in wanting scores were weakly associated with changes in binge eating, and weakened further in exercisers alone (Tables, Supplemental Digital Content 3, Pearson correlation matrix of the associations among changes in food reward, eating behavior traits and body composition in the whole group, http://links.lww.com/MSS/B828; Supplemental Digital Content 4, Pearson correlation matrix of the associations among changes in food reward, eating behavior traits, and body composition in exercisers, http://links.lww.com/MSS/B829; Supplemental Digital Content 5, Pearson correlation matrix of the associations among changes in food reward, eating behavior traits, and body composition in controls, http://links.lww.com/MSS/B830). In the whole sample, changes in body weight, fat mass, and, more weakly, body fat percentage were associated with changes in eating behavior traits but not with changes in food reward. These associations were weaker in the exercisers alone and not apparent in the controls alone, except for disinhibition.

**DISCUSSION**

This study examined the impact of a 12-wk supervised exercise intervention on state measures of food reward and trait characteristics of susceptibility to overeating in inactive individuals with overweight and obesity compared with nonexercising controls under conditions of HFAT and HCHO feeding. The 12-wk intervention led to improvements in body composition and fitness in exercisers, whereas there was a small increase in adiposity in controls. The mean group (exercisers–controls) differences in body weight and waist circumference were small but clinically meaningful according to agreed guidelines on obesity management (23). In exercisers, there was a reduction in food reward (specifically wanting) that was accompanied by improvements in eating behavior traits (clearly for binge eating and weakly for disinhibition), whereas no changes were apparent in controls.

**The impact of exercise training on food reward.** In the current study, a 12-wk exercise intervention led to a small reduction in wanting scores for high-fat relative to low-fat foods in exercisers compared with controls, but no differences in liking were found. Differences in food reward between exercisers and controls suggested that liking and wanting were generally lower in exercisers than in controls, but this effect was small and the variability was high. The group differences were more apparent in the ITT analyses, where a larger sample size and power strengthened the analysis. The changes in wanting in the exercisers from positive toward negative values indicated greater wanting scores for low-fat relative to high-fat foods after the exercise intervention. Although this reduction in wanting scores for high-fat foods was accompanied by a small reduction in intake at an *ad libitum* dinner test meal in the high-fat condition (~544 kJ; data not reported in the current article) (28), overall HFAT daily intake remained unchanged after the exercise intervention. The reduction in wanting observed in the current study may not have been large enough to elicit meaningful changes in food intake, but provides insight for a potential mechanistic influence of exercise (with modest weight loss) on food reward, specifically wanting. It is also important to consider that the design of the probe meal days (1) contained two fixed meals, and thus, if all test meals had been *ad libitum*, perhaps larger effects on energy intake may have been observed (given that a small reduction was seen at the dinner meal), and (2) did not allow for choices between high-fat and low-fat foods to be made, as each probe day was specifically designed to contain either HFAT or HCHO foods. Therefore, future studies assessing reward for high-fat versus low-fat (or sweet vs savory/nonsweet) foods in response to exercise should also include a food choice component to the assessment of food intake with *ad libitum* test meals including foods varying in fat content/taste.

**Furthermore,** on an individual level, we have shown that an increase in food liking and wanting (particularly of high-fat foods) in response to acute exercise led to less-than-expected weight loss during a prior 12-wk exercise intervention (7). This suggests a role for food reward in the compensatory...
eating response to exercise. Indeed, this may be related to changes in between-network connectivity occurring in the brain, specifically between the posterior cingulate cortex and a visuospatial network, with chronic exercise, as these have been found to be associated with changes in susceptibility to hunger assessed by the TFEQ (29).

We have recently shown in a systematic review that reward for high-fat/energy food generally decreases after weight management interventions including a range of modes of weight loss (6). The review found limited available evidence on exercise interventions; therefore, this study adds to the sparse literature in this area. Future studies could examine characteristics of exercise interventions (e.g., frequency, intensity, type, duration, and timing) that could potentially have a larger effect on reward, eating behavior, and food intake/choices than the effects demonstrated in the current study.

Cross-sectional differences in the reward value of foods (liking and wanting) have been observed in active compared with inactive men who differed in BMI (30), whereas in individuals with similar BMI (healthy range), level of habitual physical activity did not seem to influence food reward (31,32). Other studies using functional magnetic resonance imaging have found a reduction in the neural response to food cues with greater levels of habitual physical activity (33) and after exercise training (12), with inconsistencies regarding the role of body fat loss or status in the responses observed. In individuals with overweight and obesity, a 6-month exercise training intervention was associated with attenuated neural response to food cues despite no effect on behavioral measures of appetite, raising the question of whether exercise could improve weight management through attenuated hedonic motivation to eat (12). Interestingly, changes in the default mode network activity (reflecting an individual’s internal mental state) during this 6-month intervention was positively associated with changes in fat mass as well as hunger (measured via TFEQ and in response to a test meal) (34).

In contrast to functional magnetic resonance imaging, the LFPQ methodology allows for a quantified behavioral assessment of food reward. Interestingly, in a study conducted in inactive individuals with overweight and obesity, 12 wk of exercise training (523–1046 kJ, 3 d·wk\(^{-1}\)) did not affect liking or wanting scores measured by the LFPQ (8), whereas the 12-wk intervention in the current study, at a higher dose of exercise (2092 kJ, 5 d·wk\(^{-1}\)), reduced the wanting scores for high-fat food relative to nonexercising controls. The potential effects of exercise training dose (and other parameters of exercise such as those mentioned previously) on food reward warrant further investigation. Moreover, future studies combining the LFPQ with measures of neural activation (12) and changes in food intake would provide convincing evidence of the potency and specificity of exercise on food reward.

The major innovative aspect of this current study is that exercise training affected wanting rather than liking for high-fat foods. However, this effect was small and the clinical relevance for weight management cannot be determined. Wanting may be interpreted as the anticipatory reward (i.e., motivation or desire to eat before the consumption), whereas liking is the pleasure to eat (35). It could be hypothesized that exercise affects wanting more than liking, as exercise has an indirect effect on dietary habits and rather affects cognition and executive function (36). This strengthening of cognitive processes such as inhibitory control would be expected to have an effect on wanting rather than liking for high-fat food (36). On the contrary, diet interventions may have a greater effect on liking, as they are directly manipulating food patterns. In a recent systematic review, three dietary interventions reduced liking; however, wanting was not measured in these studies (6). Our study demonstrates that, in assessing effects on food reward, it is necessary to measure both liking and wanting as differing responses may be seen. We show beneficial effects of exercise on the hedonic motivation to eat through a small reduction in wanting scores for high-fat relative to low-fat foods, but not liking. Changes in food reward did not seem to be associated with changes in body weight; however, associations between fasting leptin and food reward in response to exercise training have previously been shown with or without controlling for body fat (9). It remains unknown whether the influence of chronic exercise on wanting is due to improvements in cognitive processes, to a modulation of the brain reward system or to other mechanisms. A better understanding of the neurocognitive effect of exercise and its relationship with food reward and eating behaviors is needed. It is also important to acknowledge, as shown in Figure 1, that large individual variability in the food reward responses existed, and more studies should be conducted to identify the reasons for such differences.

**Exercise training and eating behaviors promoting overconsumption.** Regarding the assessment of eating behavior traits, a week–group interaction showed that binge eating decreased in exercisers in response to the exercise intervention, whereas no changes were observed in controls. Disinhibition also showed a small decrease in exercisers, with a weaker week–group interaction, but corroborates an earlier exercise training study from our group that also found a reduction in disinhibition (10). Interestingly, the changes in eating behaviors in that study were more pronounced in those who lost more weight compared with those who lost less weight in response to the exercise intervention (10).

Cross-sectional studies in lean individuals matched for BMI ranging in physical activity levels suggest little influence of physical activity on eating behavior traits (31,32). However, across a larger range of BMI, negative associations were observed between time spent in moderate-to-vigorous physical activity and disinhibition and binge eating, but these weakened after controlling for body fat (37), and also a study by Shook et al. (38) found greater disinhibition in their lowest quintile of moderate-to-vigorous physical activity but not when controlling for body weight. Further evidence examining the effects of exercise on other trait markers of susceptibility to overeating is inconsistent, with a 6-month exercise training study reporting no effect on food cravings (12), whereas another study suggested that physical activity could
modulate craving control (39). This latter study showed that individuals who increased total exercise time over a 1-yr free-living period had a reduction in the difficulty to resist food cravings (39).

This could mean that the effect of chronic exercise and habitual physical activity on trait measures of susceptibility to overeating may be more influenced by or dependent on body weight/composition. Indeed, in the current study, changes in eating behaviors were associated with changes in body weight (more strongly in the whole group than in the exercisers alone). In contrast, food liking and wanting are considered as more state dependent, with acute exercise able to modulate short-term food reward responses (7,40), and did not seem to be influenced by changes in body weight. The effects of chronic exercise and body weight/composition on trait and state markers of overeating remain to be fully understood.

Furthermore, it has been suggested that chronic exercise may reduce binge eating through a mechanistic effect on the reward system (11). In the current study, correlational analyses suggested potential associations between changes in wanting and changes in trait binge eating in the whole sample; however, the uncertainty in our data does not allow for any conclusions to be made at this time regarding the effect of exercise on this relationship. Clearly, more work is needed to elucidate the effect of chronic exercise on the food reward and neurocognitive systems as well as on psychological eating behavior traits.

**Liking and wanting in response to HFAT and HCHO feeding conditions.** Prior baseline analyses of the current study showed that not only are high-fat (and energy-dense) foods less satiating than HCHO foods (lower satiety quotient response) and lead to an overconsumption of energy, but also consumption of these foods modulates liking and wanting (15). In the present study and in line with our previous findings (15), we show that regardless of the exercise intervention, liking and wanting scores for high-fat relative to low-fat foods were dependent on the composition of the foods consumed. Moreover, the composition of the food consumed interacted with the hunger state of the participants, showing a greater liking and wanting scores after consumption of HFAT foods compared with HCHO foods (for wanting, this effect was more prominent in the controls). However, food composition did not interact with the reward responses to exercise training. This emphasizes the importance of the energy density of the diet in determining both homeostatic (satiety and energy intake) and food reward (liking and wanting) responses. It also suggests that exercise-induced improvements in appetite control are unlikely to (on their own) overcome the overconsumption of energy typically seen with high-fat foods, as the palatable nature of energy-dense foods can offset homeostatic satiation and satiety signals (13).

**Limitations.** Despite the present study being among the few in this area to include a nonexercising control group, the relatively small number of controls compared with exercisers adds some additional uncertainty (i.e., increased size of CI) to the study outcomes. In addition, this study was not a randomized controlled trial; exercisers and controls were recruited separately. Although the exercise intervention in exercisers was supervised and closely monitored for adherence, no free-living exercise or food intake data were collected in the controls to confirm that they had not changed their behavior during the 12 wk. Furthermore, the menstrual cycle of female participants was not considered and may have affected the appetite responses. However, because the study was 12 wk in duration, the female participants should have been in the same phase of their cycle at both baseline and postintervention measures days. The interrelationships between exercise and changes in body composition make it difficult to tease out specific contributors (whether direct or indirect) to the changes in appetite observed in the current study. A future study design could attempt to control body weight during exercise training with a systematic dietary protocol or compare well-defined subgroups of weight loss responders and nonresponders to exercise with a nonexercise control group.

**CONCLUSIONS**

In inactive individuals with overweight and obesity, a 12-wk exercise intervention reduced wanting scores for high-fat foods and trait binge eating relative to nonexercising controls. A reduction in trait disinhibition with exercise was apparent but to a lesser degree. The intervention improved body composition in the exercisers compared with the nonexercising controls. Taken together with previous work on the effect of physical activity on appetite, our cautious interpretation is that exercise training, in general, enhances appetite control through an effect on homeostatic and hedonic processes occurring around an eating episode and has an improved effect on more enduring eating behavior traits promoting overconsumption. Whether these traits effects are dependent on changes in body weight/composition remains to be fully understood. Furthermore, it cannot be claimed that such an improvement will be seen in all people undertaking exercise. The effects of exercise on the body are complex and involve simultaneous physiological adjustments. Effects should be treated cautiously, and our modest interpretation is that exercise has the potential to generate biological signals that cause adaptation to the dietary environment; this will be greater in some individuals than in others. Despite the degree of uncertainty in the outcomes, we feel that it is important to continue to attempt to understand a complicated situation and to openly debate the findings.

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