Aerobic exercise reduces neuronal responses in food reward brain regions

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The current obesity epidemic is largely due to a gradual but progressive gain in body weight over time (31), which is related to an increased risk for Type 2 diabetes, cardiovascular disease, and other complications (1, 11, 39). Regular physical activity is widely regarded as playing an important role in obesity prevention efforts, including the prevention of body weight gain and body weight loss maintenance (7, 9, 60). Both observational and clinical trial data suggest that physically active individuals gain less body weight over time compared with sedentary individuals (20, 21, 26, 47). Physical activity, in the form of 30–60 min of daily structured exercise, minimizes body weight gain because it directly raises energy expenditure and resting energy expenditure (7, 21).

Emerging research has also documented an impact of acute exercise on energy intake. Previous studies have shown that exercise suppresses ad libitum energy intake, but little is known about the effects of exercise on food reward brain regions. After an overnight fast, 30 presses ad libitum energy intake, but little is known about the effects of exercise on food reward brain regions. Exercise, compared with no-exercise, significantly (P < 0.005) reduced the neuronal response to food (high and low food) cues vs. control cues in the insula (−0.37 ± 0.13% vs. −0.10 ± 0.09%), and putamen (−0.39 ± 0.10 vs. −0.10 ± 0.09%), with rolandic operculum (−0.37 ± 0.17 vs. 0.17 ± 0.12%). Exercise alone significantly (P < 0.005) reduced the neuronal response to high food vs. control and low food vs. control cues in the inferior orbitofrontal cortex (−0.94 ± 0.33%), insula (−0.37 ± 0.13%), and putamen (−0.41 ± 0.10%).

No-exercise alone significantly (P < 0.005) reduced the neuronal response to high vs. control and low vs. control cues in the middle (−0.47 ± 0.15%) and inferior occipital gyrus (−1.00 ± 0.23%). Exercise reduced neuronal responses in brain regions consistent with reduced pleasure of food, reduced incentive motivation to eat, and reduced anticipation and consumption of food. Reduced neuronal response in these food reward brain regions after exercise is in line with the paradigm that acute exercise suppresses subsequent energy intake.

Brain activity; food intake; visual attention; food reward; inhibitory control

METHODS AND PROCEDURES

Overview. Using a counterbalanced, crossover, within study design, subjects completed 60 min of rest (no-exercise) or 60 min of high-intensity exercise on a cycle ergometer. BOLD levels in response to rewarding food images were immediately measured after both conditions.

Subjects. Thirty healthy, habitually active (>3 h of physical activity/wk) adults were recruited through advertisement and fliers from the local community of San Luis Obispo, CA (Table 1). All subjects were right hand dominant, nonsmoking, free of any metabolic or chronic disease, and physically capable of performing 1 h of exercise on a stationary cycle ergometer, assessed by Health and Fitness History and Physical Activity Readiness questionnaires (PAR-Q). Exclusion criteria included standard MRI contraindications (e.g., metal and/or electronic implants, claustrophobia, and pregnant or...
trying to become pregnant), neurologic or psychiatric conditions, unsafe dieting practices, body mass index (BMI) >30 kg/m², body fat >30%, peak oxygen consumption \((V\dot{O}_2)_{peak} < 35 \text{ ml·kg}^{-1} \text{·min}^{-1}\), and any orthopedic or health problem that may have prohibited physical activity. Four of thirteen women were on a triphasic birth control regimen that was maintained during the study. Women were tested in the early follicular phase (1 to 4 days after menstruation) of the menstrual cycle. The study was approved by the Human Subjects Committee at California Polytechnic State University, and verbal and written informed consent was obtained from all subjects.

**Procedures.** Subjects were asked to refrain from exercise, alcohol, and caffeine for 24 h prior to all trials. After an 8- to 12-h overnight fast, subjects arrived at Templeton Imaging Medical (Templeton, CA) and completed a subjective appetite questionnaire using a visual analog scale as previously described (22). Subjects then either rested for 60 min (no-exercise) or exercised for 60 min in a counterbalanced, crossover fashion. There was a minimum of 1 wk between conditions. In the exercise condition, subjects exercised at 83% of HR for 60 min on a cycle ergometer (estimated \(V\dot{O}_2 = 29.3 \pm 0.9 \text{ ml·kg}^{-1} \text{·min}^{-1}\); estimated energy expenditure = 2,680 ± 108 kJ; power output = 140 ± 69 W). Average HR (beats/min), and power output (W) were recorded and relative oxygen consumption (ml·kg^{-1}·min^{-1}) and total exercise energy expenditure was estimated (42) as \(V\dot{O}_2 (\text{ml·min}^{-1}) = (\text{kg·min}^{-1}·1.9 \text{ ml/kg}) + (3.5 \text{ ml/kg·kg body wt}) + (260 \text{ ml/min}). \) Water intake during exercise was standardized, as all subjects consumed 1 liter of water.

Immediately (168 ± 9 s) following the 60 min of rest or exercise, subjects completed another appetite questionnaire and then proceeded to the MRI machine. Subjects were then instructed to lie supine on the MRI scanner table to be fitted with headphones and head coil by the MRI technician. Visual stimuli were presented from a laptop computer (Dell Latitude E5410) onto a 32-in. monitor (Vizio, Irvine, CA) using E-Prime software (Psychology Software Tools, Pittsburgh, PA). Changes in BOLD signals to high- and low-energy food cues using fMRI were assessed (see Visual food cue paradigm). Subjects viewed images of a 32-in. monitor through a two-way mirror mounted to the head coil. After the scan, subjects were given a final appetite questionnaire and completed a 24-h dietary recall.

**Visual food cue paradigm.** The food cue paradigm was adapted from Killgore et al. (35) by using the high quality photographs obtained from the authors. During the fMRI scan subjects completed two stimulation paradigms over two scanning runs in a counterbalanced order: 1) control images and low-energy foods and 2) control images and high-energy foods. Control images consisted of non-food objects with similar visual complexity, texture, and color (e.g., trees, shrubs, flowers, etc.). Low-energy food pictures included images depicting fresh fruits and vegetables, whole-grain cereal, garden salads, etc. High-energy food pictures included images depicting cheeseburgers, hot dogs, french fries, ice cream sundaes, chocolate chip cookies, barbecued chicken, pasta with meat sauce, etc. Each paradigm lasted for 180 s and consisted of 30-s control blocks that alternated with three 30-s stimulation blocks. Each block consisted of 10 images (2 dummy + 8 control/stimulation) being presented for 3 s each. The two dummy images (i.e., images of utensils) were used to provide transition from control to stimulation images.

**fMRI data acquisition.** Functional neuroimaging data were acquired in two runs on a 1.5-T Siemens Symphony MRI scanner (Siemens, New York, NY) equipped with a standard head coil. For anatomical localization, matched T1-weighted high-resolution images were collected of the entire brain (256 × 256 matrix, field of view = 256 mm², 1-mm slice thickness) in the sagittal plane as a reference for 5 min. Functional imaging was collected using a whole-brain imaging sequence \((TR = 3,000 \text{ ms}, TE = 56 \text{ ms}, field of view = 200 \text{ mm}²), 64^2\) acquisition matrix, 30 axial slices, and 3.5-mm slice thickness). BOLD data were collected during 12 blocks in one 12-min session.

**fMRI data processing and statistical analysis.** Functional imaging data were processed and analyzed in SPM8 (Wellcome Trust Centre for Neuroimaging) within the context of the general linear model on a voxel-by-voxel basis (24). Images were corrected for motion using an intrarun realignment algorithm, convolved into the standard Mon-
treat Neurological Institute (MNI) space (using the EPI template found within SPM8) (23), and smoothed using an isotropic Gaussian kernel (full width half-maximum = 7 mm) and resliced to 2 × 2 × 2 mm.

As adapted from Stoeckel et al. (53), a two-stage procedure was used for the statistical analysis of a mixed-effects design. At the first level (fixed effects), the fMRI data from each subject was used to generate statistical contrasts maps to compare brain activation to 1) control vs. high-energy foods and 2) control vs. low-energy foods. These contrasts were then entered into a second level (random effects) analysis to compare the exercise and no-exercise conditions in response to the visual food cues, using a repeated-measure ANOVA. In a separate repeated-measure ANOVA analysis, we treated both the high-energy and low-energy food cues as the same (labeled “food” in RESULTS) and compared this with control cues. Given that no previous study has comprehensively examined brain responses to visual food cues after a single bout of exercise, a priori regions of interest (ROI) that have previously been reported in the regulation of food intake were used [i.e., dorsolateral prefrontal cortex (DLPFC), orbitofrontal cortex (OFC), insula, and hypothalamus] (12, 14, 35, 44). ROI clusters were created using the Wake Forest University PickAtlas (41) and the MarsBaR toolbox (6) in addition to the Anatomic Automatically Labeling toolbox (57) to determine the anatomical location of each locus. As activation within these search territories was predicted to differ between exercise vs. no-exercise, the ROI analysis was conducted using the MarsBaR toolbox (6). To correct for multiple comparisons in the ROI approach, a Bonferroni correction was applied to the ROIs. An $\alpha < 0.05$ was considered significant. In our analyses, we started with a whole brain analysis and were able to determine the ROIs from the whole brain activity.

Also, whole brain, voxel-wise group contrasts were performed to identify differences in brain activity outside a priori ROI. Brain activation was evaluated at a threshold of $P < 0.005$ (uncorrected), with an extent threshold of five contiguous voxels, which is standard for exploratory fMRI studies given the relatively novel approach (35). A repeated-measure ANOVA was used to determine differences between conditions, visual food conditions, and the interaction between conditions and visual food cues. BOLD percent signal changes of significantly active regions were measured using the MarsBaR toolbox (6).

Exploratory analyses were also conducted in each condition separately to compare neuronal responses with high-energy food vs. control cues and low-energy food vs. control cues. A one-factor ANOVA was used to determine significantly activated/deactivated brain regions ($P < 0.005$, uncorrected). This analysis was performed to get a sense of direction of the condition effects of each run (i.e., regions being activated or deactivated) in response to the visual food cues.

Minitab 16 Statistical Software (Minitab, State College, PA) was used for statistical analyses of the subjective appetite ratings. A general linear model was used to determine differences in hunger, satiety, fullness, and prospective food consumption between the exercise and no-exercise conditions. An $\alpha < 0.05$ was considered significant, and a Bonferroni simultaneous post hoc test was applied. Pearson product moment correlations were used to assess the relationship between changes in BOLD signal and changes in appetite rating.

**RESULTS**

**ROI analysis.** Exercise, relative to no-exercise, significantly reduced ($P < 0.05$; Table 2) the neuronal response to high food vs. control cues and food vs. control cues in the insula ($−0.35 \pm 0.13$ vs. $0.05 \pm 0.17\%$ and $−0.45 \pm 0.10$ vs. $−0.06 \pm 0.11\%$, respectively). Exercise, relative to no-exercise, increased the neuronal response to food vs. control cues in the hypothalamus but did not reach significance ($P = 0.064$; Table 2). No other a priori ROI was significantly active (Table 2).

**Whole brain analysis.** Exercise, relative to no-exercise, significantly reduced the neuronal response to high food vs. control cues in the left and right insula ($P < 0.005$, uncorrected; Table 3). Also, exercise, relative to no-exercise, significantly reduced the neuronal response to food vs. control cues in the left postcentral gyrus, right putamen, right insula, right Rolandic operculum, and right supramarginal gyrus ($P < 0.005$, uncorrected; Table 3, Fig. 1). Exercise, relative to no-exercise, significantly reduced the neuronal response to low food vs. control cues in the left postcentral gyrus ($P < 0.005$, uncorrected).

**Table 3. Foci of significantly active clusters ($P < 0.005$, uncorrected) for whole brain analysis in exercise and no-exercise conditions**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Region</th>
<th>MNI Coordinate</th>
<th>$T$ Statistic</th>
<th>Cluster Size</th>
<th>EX, %Signal Change</th>
<th>NEX, %Signal Change</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Exercise &lt; No-Exercise</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>High food vs. Con</td>
<td>L insula</td>
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<td>8</td>
<td>−8</td>
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</tr>
<tr>
<td></td>
<td>R insula</td>
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<td>5</td>
<td>−5</td>
<td>3.76</td>
<td>115</td>
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<tr>
<td>Low food vs. Con</td>
<td>L postcentral gyrus</td>
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<td>−4</td>
<td>25</td>
<td>3.56</td>
<td>71</td>
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<td>Food vs. Con</td>
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<td>−7</td>
<td>28</td>
<td>4.32</td>
<td>315</td>
</tr>
<tr>
<td></td>
<td>R putamen</td>
<td>24</td>
<td>2</td>
<td>−5</td>
<td>3.91</td>
<td>64</td>
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<tr>
<td></td>
<td>R insula</td>
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<td>2</td>
<td>−11</td>
<td>3.78</td>
<td>74</td>
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<tr>
<td></td>
<td>R Rolandic operculum</td>
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<td>−22</td>
<td>13</td>
<td>3.78</td>
<td>90</td>
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<tr>
<td></td>
<td>R supramarginal gyrus</td>
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<td>−34</td>
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<td><strong>Exercise &gt; No-Exercise</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>High food vs. Con</td>
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<td>−55</td>
<td>34</td>
<td>4.79</td>
<td>234</td>
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<td>Low food vs. Con</td>
<td>No significant regions</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Food vs. Con</td>
<td>No significant regions</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Values are mean ± SE. EX, exercise; NEX, no-exercise. MNI refer to stereotactic coordinates in Montreal Neurologic Institute space. *Cluster mass center not specified by atlas, used proximal region.
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rected; Table 3). Figure 2 presents a brain view of the exercise and no-exercise conditions depicting the reduced neuronal response after exercise.

Exercise, relative to no-exercise, significantly increased the neuronal response to high food vs. control cues in the left precuneus (P < 0.005, uncorrected; Table 3). No other brain region was significantly altered in exercise vs. no-exercise condition (Table 3).

Whole brain analysis for exercise and no-exercise alone. Exercise was associated with reduced neuronal response (de-activated) to high food vs. control cues in the left middle occipital gyrus, right inferior occipital gyrus, right inferior frontal gyrus (triangular part), left superior parietal gyrus, right putamen, left and right insula, and cerebellar vermis (P < 0.005, uncorrected; Table 4), whereas it significantly increased neuronal response (activation) in the right precuneus (P < 0.005, uncorrected, Table 4). Similarly, exercise was associated with reduced neuronal response to low food vs. control cues in the right inferior occipital gyrus, left middle occipital gyrus, left inferior orbitofrontal cortex, left superior frontal gyrus (medial surface), and left precentral gyrus (P < 0.005, uncorrected; Table 4).

In contrast, the no-exercise condition was associated with reduced neuronal response to high food vs. control and low food vs. control cues only in the right inferior occipital gyrus and left middle occipital gyrus (P < 0.005, uncorrected; Table 4), whereas it significantly increased the neuronal response in the right lingual gyrus (P < 0.005, uncorrected; Table 4).

Subjective appetite responses. Exercise, relative to no-exercise condition, significantly lowered ratings of hunger (P = 0.02; Fig. 3) and prospective food consumption (P < 0.01; Fig. 3) and significantly increased perceptions of satiety (P = 0.02; data not shown) and fullness (P = 0.02; data not shown). Most correlations between BOLD signal in brain regions and subjective appetite responses were weak and/or nonsignificant (P > 0.05).

DISCUSSION

In the current study, an acute bout of exercise altered the neuronal (or BOLD) response to food cues in brain regions important in food reward, visual attention, and inhibitory control. Specifically, in support of our hypotheses, we found that exercise resulted in a decreased responsiveness to food cues in food reward regions (i.e., insula, putamen, rolandic operculum) and near significant for hypothalamus. In follow-up exploratory analyses, we similarly showed that exercise reduced the neuronal response in two food reward regions (right inferior orbitofrontal cortex and bilateral insula), which

Fig. 1. Mean blood oxygen level-dependent (BOLD) percent signal change (±SE) of significantly active clusters to food vs. control cues in exercise (EX) and no-exercise (NEX) conditions. Exercise significantly different than no-exercise (P < 0.05) in all brain regions.

Fig. 2. Whole brain view of neuronal responses to high food cues vs. control cues (A) and food cues vs. control cues (B) in exercise and no-exercise conditions. Exercise, relative to no-exercise, significantly (P < 0.005, uncorrected) reduced the neuronal response in the bilateral insular cortices and right putamen and significantly increased (P < 0.005, uncorrected) the neuronal response in the precuneus. Data are shown in neurological orientation (i.e., right hemisphere on right, left hemisphere on left). Color bar reflects suprathreshold value of the SPM{t} statistic for analysis.
have a central role in reward value (45, 46), expected reward value (25), regulation of food behavior (12, 14, 50, 51), and subjective pleasantness of food (40). Taken together, these data suggest that acute exercise reduces the neuronal response in food reward brain regions in a direction expected to decrease energy intake.

In the current study, we observed a clear effect of acute aerobic exercise on reducing neuronal responses to visual food cues. Acute exercise, relative to no-exercise, resulted in significantly reduced neuronal responses in the left and right insula, right putamen, right rolandic operculum, and orbitofrontal cortex, all of which are consistent with reduced food reward (13, 48, 52). Furthermore, reduced activity in the orbitofrontal cortex is consistent with reduced decision making and reduced pleasantness and palatability of food (61). To our knowledge, only two other published studies have evaluated neuronal response to liking and wanting in food reward regions (i.e., insula, putamen, rolandic operculum, orbitofrontal cortex) after acute exercise.

Food reward can be divided into two different components: liking (pleasure and palatability of food) and wanting (incentive motivation to eat) (3). Previous studies have shown that neuronal responses to liking and wanting may overlap or may be distinctly different (5, 25, 38, 49). Born et al. (5) found that liking task-related brain signaling was apparent in the insula, whereas wanting task-related brain signaling was in the cingulate cortex, thalamus, and putamen. Others found that the orbitofrontal cortex is a liking brain region (49). In the current study, although we did not directly measure liking and wanting, we found that acute exercise reduced both liking (i.e., insula, orbitofrontal cortex) and wanting (i.e., putamen) specific brain regions. Moreover, we found that acute exercise reduced activity in the right rolandic operculum, which is consistent with reduced anticipation and consumption of food (52). Thus our data are consistent with acute exercise reducing the pleasure of food (liking), incentive motivation to eat (wanting), and anticipation and consumption of food.

### Table 4. Foci of significantly active clusters (P < 0.005, uncorrected) for whole brain analysis in exercise and no-exercise conditions alone

<table>
<thead>
<tr>
<th>Condition</th>
<th>Region</th>
<th>MNI Coordinate</th>
<th>T Statistic</th>
<th>Cluster Size</th>
<th>%Signal Change</th>
</tr>
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<tr>
<td><strong>Exercise</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High vs. Con</td>
<td>Activated</td>
<td>R precuneus</td>
<td>−12</td>
<td>−55</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td>Deactivated</td>
<td>L middle occipital gyrus</td>
<td>−36</td>
<td>−55</td>
<td>−17</td>
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<tr>
<td></td>
<td></td>
<td>R inferior occipital gyrus</td>
<td>42</td>
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<td>1</td>
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<tr>
<td></td>
<td></td>
<td>R inferior frontal gyrus (triangular part)</td>
<td>57</td>
<td>35</td>
<td>19</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L superior parietal gyrus</td>
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<td>27</td>
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<td></td>
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<td>L insula</td>
<td>−45</td>
<td>11</td>
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<tr>
<td></td>
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<td>Cerebellar vermis*</td>
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<td>4</td>
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<td>−73</td>
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<td>42</td>
<td>−82</td>
<td>5</td>
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<td></td>
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<td>L inferior OFC</td>
<td>−27</td>
<td>35</td>
<td>−14</td>
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<td></td>
<td></td>
<td>L superior frontal gyrus (medial surface)</td>
<td>−12</td>
<td>65</td>
<td>16</td>
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<td></td>
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<tr>
<td>High vs. Con</td>
<td>Activated</td>
<td>R lingual gyrus</td>
<td>15</td>
<td>−79</td>
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<td>Deactivated</td>
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<td>−76</td>
<td>−2</td>
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<td></td>
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<td></td>
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<td>R inferior occipital gyrus</td>
<td>39</td>
<td>−91</td>
<td>10</td>
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Values are mean ± SE. MNI refers to stereotactic coordinates in Montreal Neurologic Institute space. *Cluster mass center not specified by atlas, used proximal region.
Interesting, in exploratory analyses, we also found that exercise led to significantly reduced neuronal response in cortical brain regions associated with visual attention and processing (i.e., left middle occipital gyrus and right inferior occipital gyrus). These findings suggest that exercise may decrease the awareness and reward of external food cues, especially with the concomitant deactivation of prefrontal regions. This is in line with previous findings (12–14, 32, 43) showing that energy imbalance, with or without exercise, alters visual processing brain centers. However, we also observed that exercise increased the neuronal response in the precuneus, which is consistent with reward anticipation. Interestingly, Janse Van Rensburg et al. (32) found similar activation of the precuneus after 10 min of exercise. Emerging evidence indicates that the role of the precuneus is extremely complex and, in addition to being associated with a visual-processing center, may also play a role in spatial relations of body movement, motor imagery, and attention shift between objects (10). Thus, in the current study, the increased activation of the precuneus in response to exercise may be related to higher-order cognitive functions and not necessarily a heightened awareness to the visual food cues.

In general, a single bout of exercise suppresses subsequent ad libitum energy intake (36). Previous studies have shown that exercise alters appetite hormones in a direction expected to suppress energy intake (8, 28, 29, 36), whereas others have focused on body temperature, dehydration, and gastric motility to suppress energy intake (33, 59). To our knowledge, few studies examined the effects of brain activity on energy intake (15, 52), and none, including the present study, evaluated whether exercise-induced changes in brain activity alter ad libitum energy intake. The lack of prior data limits our ability to infer that reduced neuronal responses in food reward and visual attention brain regions would have suppressed actual energy intake in our exercise treatment.

Surprisingly, we did not find changes in the dorsolateral prefrontal cortex (a commonly recognized inhibitory region) in response to exercise, and in our exploratory analyses we found that exercise reduced activation in the superior frontal gyrus (medial surface), which is associated with an inhibitory control region. These data, coupled with our findings that activity in other frontal regions were decreased [right inferior OFC, right inferior frontal gyrus (triangular part)], are at least partially in line with the “transient hypofrontality” hypothesis (17, 18). The transient hypofrontality hypothesis proposes that aerobic exercise decreases neural activity to brain regions that are not necessary to the preservation of the exercise bout (18). These brain areas with decreased activity include the higher cognitive centers of the frontal lobe (19). During exercise the metabolic demands of the brain are increased (34), but blood flow to the cerebral cortex and other frontal brain regions, as a proportion of cardiac output, is reduced during high-intensity exercise (19, 55). Therefore, in response to exercise it becomes challenging to maintain neural activity to all brain structures and it seems that frontal lobe regions have decreased activity.

Consistent with the current brain findings, after exercise, participants reported lower self-perceptions of hunger and prospective food consumption. These results are consistent with most previous studies showing that exercise alters appetite ratings in a direction expected to suppress food intake (8, 36, 37). However, in the current study many correlations between brain activity and appetite ratings were weak and nonsignificant, which is in disagreement with a previous fMRI study (43). The lack of correlation in our data suggests a possible disconnect between appetite ratings and food reward brain activity. This is partially supported by a recent study (4) showing that appetite hormones and appetite ratings respond to different metabolic signals (i.e., changes in energy balance vs. meal size, respectively). Although we did not measure appetite hormones in the current study, it is plausible that brain activity and appetite ratings are also responding to different metabolic signals, although this is highly speculative.

There are several limitations of the current study. First, because all conditions occurred at our fMRI facilities not in our laboratory, we were unable to directly measure oxygen consumption and energy expenditure during exercise. We chose a similar dose of exercise (high intensity, 60 min) as previous studies showing robust changes in appetite hormones and decreased energy intake (8, 36). Also, estimating oxygen consumption and energy expenditure using the Latin et al. (42)
equation has a high correlation with actual exercise energy expenditure. Second, due to our convenient sample of healthy, habitually active subjects, the generalizability to sedentary, clinical overweight or obese groups is limited. Previous studies (16, 44) have shown brain responses to visual food cues to differ between individuals of different weight classifications, but further research is still needed to determine the impact of acute exercise on brain responses in at-risk, sedentary groups. Third, the strength of the fMRI machine we used, a 1.5-Tesla, may not have been robust enough to detect differences in homeostatic and hedonic control feeding centers deep within the brain, although the hypothalamus approached significance (P = 0.06). Previous studies have shown increased activity in the hypothalamus and other inner brain regions using a 1.5-Tesla (35), but more recent studies have shown greater activity in inner brain regions using a more powerful 3.0-Tesla (14, 53). Fourth, we did not assess body weight and body temperature in response to exercise, both of which may influence subsequent energy intake (59). In our previous studies (27–29) using a similar dose of exercise, we observed <0.45-kg change in body weight pre- to postexercise, and it remains unclear the impact of exercise-induced changes in weight and body temperature on brain activity. Fifth, subjects were overnight fasted in both conditions, and it is possible that feeding subjects prior to exercise may alter the brain activity patterns observed in the current study. To fully explore this, future studies are needed to evaluate fed vs. fasted on exercise-induced brain activity. Finally, in the no-exercise condition, we observed minimal changes in neuronal activity, which is in disagreement with previous studies using similar normal-weight subjects (35). However, our subjects were habitually active and had moderate to high cardiorespiratory fitness levels, which may have had an independent effect on neuronal activity.

In summary, we observed a clear difference in the way acute exercise alters neuronal responses to visual food cues. Exercise reduced neuronal responses in food reward and visual attention brain regions. This has implications for both our understanding of basic science (e.g., brain regions that alter energy intake) and practical perspective (e.g., exercise and body weight gain). This has implications for both our understanding of basic science (e.g., brain regions that alter energy intake) and practical perspective (e.g., exercise and body weight gain). This has implications for both our understanding of basic science (e.g., brain regions that alter energy intake) and practical perspective (e.g., exercise and body weight gain).

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS


REFERENCES


Exercise and Neuronal Response in Food Reward Brain Regions  


