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Randomized Trial of a Dissonance-Based Transdiagnostic Group Treatment for Eating Disorders: An Evaluation of Target Engagement

Eric Stice, Sonja Yokum, Paul Rohde, Heather Shaw, Jeff M. Gau, and Sarah Johnson
Oregon Research Institute, Eugene, Oregon

Aviva Johns
University of Texas at Austin

Objective: Test whether a dissonance-based transdiagnostic eating disorder treatment reduces valuation of the thin beauty ideal and high-calorie binge foods, the intervention targets, and eating pathology relative to waitlist controls. ***Method:*** Women with *Diagnostic and Statistical Manual of Mental Disorders–5* eating disorders ($N = 100$) were randomized to an 8-week group-implemented Body Project Treatment (BPT) redesigned to encourage rapid symptom reduction or a waitlist control condition, completing functional MRI paradigms assessing neural response to thin models and binge foods, questionnaires, and diagnostic interviews at pretest and posttest. ***Results:*** Compared to controls, BPT participants showed greater reductions in responsivity of regions involved in reward valuation (ventromedial prefrontal cortex, dorsolateral prefrontal cortex, caudate) to thin models but not binge foods, pursuit of the thin ideal ($d = .72$), palatability ratings of binge foods ($d = .78$), and greater increases in attractiveness ratings of average-weight models ($d = .44$), the intervention targets. BPT participants also showed significantly greater reductions in body dissatisfaction ($d = .83$), negative affect ($d = .76$), and eating disorder symptoms ($d = .59$), and marginally greater abstinence from binge eating and compensatory behaviors (39% vs. 21%) than controls. ***Conclusions:*** Results provide novel evidence that BPT affected the hypothesized intervention targets and reduced variables that are putatively secondary to pursuit of the thin ideal, including body dissatisfaction, negative affect, and eating disorder symptoms. Symptom reductions were smaller than in past trials, suggesting that it may be optimal to reduce valuation of the thin ideal before asking participants to reduce disordered eating behaviors that are used to pursue this ideal.

What is the public health significance of this article?
This study provides the first evidence that a novel dissonance-based transdiagnostic eating disorder treatment, the Body Project Treatment, reduced reward region response to thin models, pursuit of the thin ideal, and palatability ratings of binge foods and increased attractiveness ratings of average-weight women, the intervention targets. This treatment also reduced body dissatisfaction, negative affect, and eating disorder symptoms. Data suggest that this inexpensive and efficient treatment might prove useful in treating the full range of eating disorders.

Keywords: eating disorder, treatment, target engagement, dissonance

Eating disorders affect 13–15% of women by young adulthood and are marked by chronicity, relapse, distress, functional impairment, and premature morbidity (Dakanalis et al., 2017; Stice, Marti, & Rohde, 2013). Unfortunately, over 80% of afflicted individuals do not receive treatment (Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), in part because it is difficult to locate clinicians who deliver evidence-based treatments and most evidence-based treatments involve 20 individual sessions (Wilson & Zandberg, 2012), which is intensive and expensive. The fact that evidence-based treatments differ for the various eating disorders (cognitive–behavioral therapy [CBT] for bulimia nervosa [BN] and binge eating disorder [BED] vs. family therapy for anorexia nervosa [AN]) also hinders implementation. Further, many treated individuals do not receive evidence-based care (Lilienfeld et al., 2013) and only 47% of those who receive evidence-based treatments achieve abstinence from binge eating and compensatory behaviors for at least a month (Hay, 2013). Individuals often continue to report pursuit of the thin ideal and body dissatisfaction posttreatment, which increases relapse risk (Bardone-Cone et al., 2010), suggesting that a treatment that reduces these factors should reduce relapse.

Eric Stice, Sonja Yokum, Paul Rohde, Heather Shaw, Jeff M. Gau, and Sarah Johnson, Oregon Research Institute, Eugene, Oregon; Aviva Johns, Department of Psychology, University of Texas at Austin.

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Correspondence concerning this article should be addressed to Eric Stice, Oregon Research Institute, 1776 Millrace Drive, Eugene, OR 97403. E-mail: estice@ori.org

The recognition that a transdiagnostic treatment for eating disorders would be useful prompted the development of enhanced CBT (CBT-E), designed for use with the full spectrum of eating disorders. CBT-E did not produce significantly greater abstinence from binge eating and compensatory behaviors by 4-month follow-up than integrative cognitive–affective therapy (ICAT) in one trial (23% vs. 33%, respectively; Wonderlich et al., 2014). ICAT focuses on adapting emotional and interpersonal behavior through structured meal planning, identifying interpersonal and self-directed behavior (e.g., excessive self-control) used to avoid negative emotions, and developing coping strategies to handle negative emotions. Likewise, CBT-E did not produce significantly greater abstinence than interpersonal therapy (IPT) by 15-month follow-up (34% vs. 35%, respectively; Fairburn et al., 2015). The fact that CBT-E, like ICAT and IPT, is delivered in 20 individual sessions makes these expensive treatments that would be difficult to implement broadly.

We therefore developed a brief treatment for the spectrum of eating disorders that could be easily and inexpensively implemented. In this eight-session group intervention, referred to as Body Project Treatment (BPT), women with eating disorders complete verbal, written, and behavioral activities in which they discuss negative effects of pursuing the thin beauty ideal and engaging in disordered eating behaviors. These activities putatively create dissonance that reduces valuation of the thin ideal and eating disorder behaviors, as people are motivated to align their attitudes with their publically displayed behavior (Festinger, 1957). BPT is an extension of the Body Project (Stice, Marti, Spoor, Presnell, & Shaw, 2008), a dissonance-based eating disorder prevention program in which women with body image concerns discuss costs of pursuing the thin ideal. The Body Project has produced greater reductions in eating disorder risk factors and symptoms compared to both assessment-only controls and alternative interventions, including trials conducted by independent labs (e.g., Becker et al., 2010; Ciao, Latner, Brown, Ebneter, & Becker, 2015; Halliwell & Diedrichs, 2014; Stice et al., 2008; Stice, Rohde, Shaw, & Gau, 2017). It produced a 60% reduction in eating disorder onset relative to assessment-only controls over a 3-year follow-up (Stice et al., 2008) and a 74% reduction in eating disorder onset relative to an Internet-based eating disorder prevention program over a 7-month follow-up (Stice et al., 2017). It produced larger symptom reductions for women with versus without a threshold or subthreshold eating disorder at baseline (Cohen's d = .71 and .18, respectively; Müller & Stice, 2013), supporting its use as the foundation for a new eating disorder treatment. Further, women with body image concerns who completed the Body Project showed a pre-to-post reduction in functional MRI (fMRI)-assessed reward region (caudate) response to thin models compared to educational brochure controls, suggesting that participants perceive the thin ideal as a less desirable goal after completing the intervention (Stice, Yokum, & Waters, 2015) and providing evidence of target engagement using objective brain imaging.

In an initial trial, 72 women with any threshold or subthreshold *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*)*–5* eating disorder were randomized to BPT or usual care (any treatment participants usually received; Stice, Rohde, Butryn, Menke, & Marti, 2015). BPT participants showed significantly greater reductions in outcomes through 2-month follow-up compared to usual care, with effects for thin-ideal internalization (d = .79), dissonance regarding perpetuating the thin ideal (d = .65), body dissatisfaction (d = 1.14), negative affect (d = .55), and eating disorder symptoms (d = .95), but no effects for functional impairment (d = .04). Effects for thin-ideal internalization, dissonance regarding perpetuating the thin ideal, body dissatisfaction, and eating disorder symptoms were significantly larger for BPT participants who attended more sessions and completed more homework, providing evidence of a dose-response relation.

In a second trial, 84 young women with any threshold or subthreshold *DSM–5* eating disorder were randomized to BPT or a supportive mindfulness group treatment typical of that offered at US colleges (Stice, Rohde, Shaw, & Gau, 2019). By 6-month follow-up, 77% of BPT participants no longer met diagnosis for an eating disorder versus 60% of supportive mindfulness participants (relative risk ratio = 2.22; $p < .05$), though there were no differences in eating disorder symptom change. BPT versus supportive mindfulness participants showed significantly lower dissonance about affirming the thin ideal at posttest and 6-month follow-up (d = .38 and .32), body dissatisfaction at posttest and 6-month follow-up (d = .62 and .62), negative affect at posttest and 6-month follow-up (d = .49 & .48), and functional impairment at 6-month follow-up (d = .36); there were no effects for thin-ideal internalization. BPT participants showed higher abstinence from binge eating and compensatory behaviors in the past month than supportive mindfulness participants (55% vs. 39%), though this difference was not significant. Effects for thin-ideal internalization, dissonance regarding perpetuating the thin ideal, body dissatisfaction, negative affect, and eating disorder symptoms were significantly larger for BPT participants who attended more sessions, providing additional evidence of a dose-response relation. Thus, both treatments appeared effective, but BPT produced significantly larger effects and greater remission of eating disorder diagnoses than the supportive mindfulness intervention, a credible alternative treatment, which is very rare for trials that have compared eating disorder treatments. In support of the efficacy of mindfulness-based treatment, obese individuals with threshold or subthreshold BED assigned to a mindfulness eating disorder treatment showed significantly greater reductions in binge eating, depression, and BED diagnoses than waitlist controls, but not relative to CBT (Kristeller, Wolever, & Sheets, 2013). The 55% abstinence from binge eating and compensatory behaviors was larger than the 23%–34% abstinence rate produced by CBT-E, 33% abstinence rate produced by ICAT, and 35% abstinence rate produced by IPT (Fairburn et al., 2015; Wonderlich et al., 2014).

We next conducted a trial that evaluated target engagement of BPT. Aim 1 was to test whether BPT participants would show greater reductions in reward region responsivity to thin models and high-calorie binge foods, reduced attractiveness ratings for thin models and palatability and monetary valuation ratings for high-calorie binge foods, and increased attractiveness ratings for average-weight women and palatability and monetary valuation ratings of low-calorie foods (primary outcomes) than control participants. We used a waitlist control condition to permit an examination of the test–retest reliability of target engagement measures. We hypothesized reductions in reward region response to thin models and high-calorie binge foods because the treatment involves dissonance-based activities that theoretically reduce valuation of the thin ideal and eating disordered behaviors, such as

binge eating, that cross-cut various eating disorders. Although it is reasonable to expect differences in neural response to thin models and high-calorie binge foods for participants with restricting AN versus the other eating disorders that involve binge eating and compensatory weight control behaviors, there is evidence that both individuals with AN (Cowdrey, Park, Harmer, & McCabe, 2011; Vocks, Herpertz, Rosenberger, Senf, & Gizewski, 2011) and those with other eating disorders show elevated reward region response to high-calorie foods (Geliebter et al., 2006; Schienle, Schäfer, Hermann, & Vaitl, 2009; Simon et al., 2016; Uher et al., 2004). We were unable to locate studies that compared neural response to thin models for individuals with AN or other eating disorders versus healthy controls. Secondary outcomes were body dissatisfaction, negative affect, eating disorder symptoms, and abstinence from binge eating and compensatory behaviors over the past 30 days, which are putatively driven by valuation of the thin ideal and high-calorie binge foods.

Aim 2 was to test whether participants who attended more sessions showed greater symptom reductions than those who attended fewer sessions to establish a dose-response relation that might provide an indication of the optimal dose of BPT. Because participants in the first trials showed greater reductions in outcomes when they attended more sessions, we refined the intervention to improve engagement and acceptability based on qualitative input from participants. In addition, based on experimental evidence that a CBT intervention that encouraged rapid symptom reduction produced larger reductions in symptoms than a CBT intervention that did not encourage rapid symptom reduction (MacDonald, McFarlane, Dionne, David, & Olmsted, 2017), we revised the intervention script to encourage rapid symptom reduction in the second session, telling participants that this is because it interrupts processes that maintain these behaviors. In contrast, the version of the BPT evaluated previously focused primarily on reducing pursuit of the thin ideal in the first three sessions and did not focus on symptom reductions until the fourth session.

Method

Participants and Procedure

We recruited 100 women (M_{age} = 21.46 ± 3.63) from universities and surrounding communities in Oregon and Texas. Web postings, flyers, and mailings invited women with body image and eating concerns to participate in a treatment study. We also encouraged local clinics that treat eating disorders and eating disorder recovery centers to refer people to our trial. Informed consent was obtained for this institutional review board-approved trial. Participants completed a web-screener; a brief phone screen or in-person interview verified inclusion and exclusion criteria. Women with AN with a body mass index (BMI) below 17 were excluded because they were not deemed appropriate for outpatient treatment without medical monitoring, similar to the exclusion criteria used for transdiagnostic outpatient treatment trials (e.g., Fairburn et al., 2015). Suicidal ideation and substance abuse were also exclusion criteria. Suicidal ideation was assessed using the item "Have you had thoughts that you would be better off dead or hurting yourself in some way?" on the Patient Health Questionnaire–9 (Kroenke, Spitzer, & Williams, 2001) and substance misuse was assessed with the Drug Abuse Screening Test (Skinner, 1982) and Alcohol Use Identification Test (Saunders, Aasland, Babor, de la Fuente, & Grant, 1993), though no one was excluded for either.

We focused on subthreshold AN (BMI between 90% and 85% expected for age and sex, definite fear of weight gain for more than 25% of the days in the past 3 months, weight/shape was a key aspect of self-evaluation), rather than atypical AN, because we wanted to parallel the focus on subthreshold BN and BED. The sample was 61% Caucasian, 13% Hispanic, 3% Black, 16% Asian, 4% multiracial, 1% other, and 2% who did not report their race. Highest parental education was 3% some high school, 4% high school graduate, 16% some college, 30% college graduate, and 47% advanced degree.

Participants were randomized to BPT (n = 51) or waitlist control (n = 49) conditions using a random number table. The project coordinator was solely responsible for allocation and did not know which condition participants would be assigned to until each had completed their pretest assessment (there were 12 exceptions due to needing to assign participants to the waitlist condition because we could not implement groups due to the holiday season). Assessors were not informed of the allocation of participants to condition (but 33 of the 92 follow-up assessments [17% of all assessments] had to be completed by the project coordinator because no other assessor was available). At baseline, participants completed surveys, electrocardiogram measurements (to assess risk for cardiac problems), a diagnostic interview, and the fMRI scan. The average time between the pretest scan to starting treatment was 2.3 weeks (SD = 1.3). Soon after finishing BPT groups and a parallel time for waitlist controls, participants repeated all assessments. The average time between completing treatment to posttest scan was 1.6 weeks (SD = 1.8). Figure 1 depicts enrollment and participation flow in this study.

Given that we anticipated that some participants would not be scan eligible because of braces, tattoos, or metal implants, our goal was to recruit at least 80 participants who could provide scan data because it should have provided a power of .80 to detect medium effect sizes (d = .50) or larger. We overrecruited because we anticipated attrition over follow-up. In total, 86 women completed baseline fMRI scans. Of these, 75 completed posttest scans.

Body Project Treatment (BPT)

BPT consisted of eight weekly 1-hr group sessions with four to nine participants led by two therapists. Sessions began with participants verbally stating their willingness to actively participate. During sessions participants completed written and verbal exercises, including defining the thin ideal, discussing costs of pursuing this ideal and disordered eating symptoms (dietary restriction, weight and shape overvaluation, binge eating, purging, compensatory behaviors), role-plays in which participants dissuade facilitators from pursuing the thin ideal and engaging in disordered eating behaviors, motivational exercises (e.g., discussing the importance of addressing their eating disorder), and shared their home exercises. Between sessions, participants completed exercises such as writing letters (e.g., to their eating disorder), motivational exercises (e.g., writing about the importance of improving their body image), a mirror body appreciation exercise, generating lists of "body activism" behaviors women can do to resist the thin ideal, reducing "linchpin" eating disorder symptoms (including

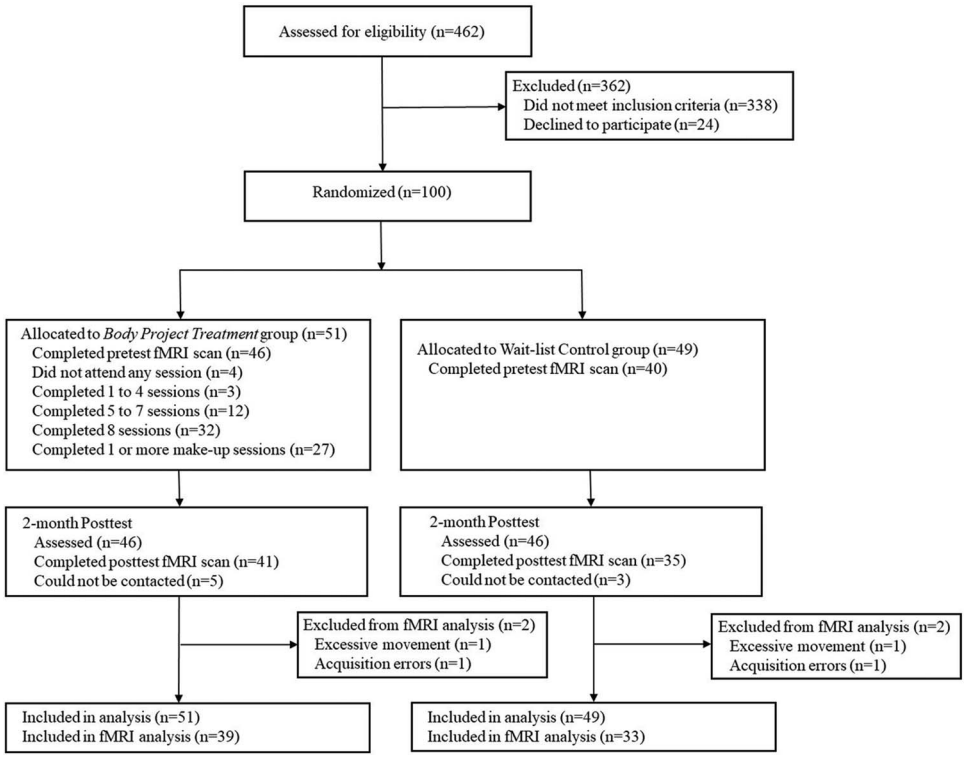

Figure 1. Participant flow through the study.

consuming three healthy meals daily), and tracking their binge episodes and compensatory behaviors. The major changes from the earlier version of BPT are that participants were encouraged to begin reducing eating disorder symptoms earlier (in Session 2 vs. 4) and discussed the costs of each eating disorder symptom. The intervention script is available for free at www.bodyprojectsupport.org.

Facilitators had a doctoral or master's degree in clinical psychology. Training involved reading the manual and attending an 8-hr workshop to learn the intervention rationale, roleplay delivery, and discuss process issues. Sessions were video-recorded and reviewed by Paul Rohde and Heather Shaw, who provided emailed supervision before the next session. Facilitators tracked attendance (4-point scale; *absent*, *partial attendance*, *full attendance*, *make-up session*), participation (3-point scale; *none* or *negative*, *minimal*, *good/active*), and homework completion (4-point scale; *none*, *some*, *all*, *didn't bring materials*).

Measures

fMRI paradigms. Participants were asked to consume their regular meals but refrain from eating or drinking caffeinated beverages for 3 hr before scans. Prior to each scan, they reported the last time they ate and hunger (assessed on 20-cm cross-modal visual analog scales anchored by −10 [*not at all*], 0 [*neutral*], and 10 [*never been hungrier*]). Mean fasting (±*SD*) hours were 5.8 ± 5.3 (pretest) and 5.8 ± 5.0 (posttest). M_{hunger} (± *SD*) was −2.71 ±5.3 and −1.08 ±4.1 at pre- and posttest, respectively. Participants completed a food image exposure paradigm assessing neural response to images of high-calorie binge foods and low-calorie foods. During this event-related paradigm, participants were exposed to 20 high-calorie binge food images (e.g., chocolate cookies) and 20 low-calorie food images (e.g., broccoli) and asked to think about how much they wanted to eat each food. Participants completed a model image exposure paradigm assessing neural response to images of thin and average-weight models. Participants were asked to think about the attractiveness of each model. In both paradigms, images were presented for 5 s in a randomized order and a 4- to 8-s jitter occurred between each image during which a blank screen with a crosshair was shown. Order of presentation of the images was randomized across participants. During each paradigm, stimuli were presented in one scanning run (total duration 7.5 min). Order of the two paradigms was counterbalanced across participants. To prevent order habituation from pretest to posttest, order of paradigms was also randomized across the two assessments.

Picture ratings. Immediately after the scans, participants rated palatability (scale ranging from 1 [*not at all appetizing*] to 9 [*extremely appetizing*]) and monetary value (scale ranging from 1 [*$1 or less*] to 10 [*$10 or more*]) of the foods and attractiveness (scale ranging from 1 [*not at all attractive*] to 9 [*extremely attractive*]) of the models. Food images were adapted from prior work examining foods most associated with overeating (Stice, Yokum, Veling, Kemps, & Lawrence, 2017) and were piloted with university students to ensure that each was easily identified (i.e., high-calorie binge foods and fruits/vegetables). Palatability and monetary ratings of high-calorie and low-calorie food images and

attractiveness ratings of thin and average-weight models have shown sensitivity to detecting intervention effects (Stice et al., 2017; Stice, Yokum, et al., 2015). At baseline, participants rated the thin models as significantly more attractive ($M = 6.38$, $SD = 1.2$) than the average-weight models ($M = 4.69$, $SD = 1.4$), $t(83) = 8.50$, $p < .001$.

Thin-ideal internalization. The 8-item Thin-Ideal Internalization Scale assessed pursuit of the thin ideal (Stice et al., 2017) using response options ranging from 1 = *strongly disagree* to 5 = *strongly agree*. Items were generated from a focus group that was asked to list descriptors of the thin beauty ideal espoused for women, rather than through factor analyses. Items were averaged for this and the other scales. This scale has shown 2-week test–retest reliability ($r = .80$), predictive validity for onset of BN, BED, and purging disorder, convergent validity with positive expectances for thinness, and sensitivity to detecting the effects of an intervention designed to reduce pursuit of the thin ideal (Stice et al., 2008, 2017); $\alpha = .71$ at pretest.

Body dissatisfaction. The 17-item Satisfaction and Dissatisfaction With Body Parts Scale (Berscheid, Walster, & Bohrnstedt, 1973) assessed body dissatisfaction using response options ranging from 1 = *strong positive feelings* to 5 = *strong negative feelings*. It has exhibited internal consistency ($\alpha = .94$), 3-week test–retest reliability ($r = .90$), predictive validity for bulimic symptom onset, and sensitivity to detecting the effects of an intervention designed to improve body satisfaction (Stice et al., 2008, 2017); $\alpha = .89$ at pretest.

Negative affect. Negative affect was assessed with 20 items from the sadness, guilt, and fear/anxiety subscales of the Positive Affect and Negative Affect Scale–Revised (Watson & Clark, 1992). Participants reported the extent to which they had felt each emotion on scales ranging from 1 = *very slightly or not at all* to 5 = *extremely*. This scale has shown internal consistency ($\alpha = .95$), 3-week test–retest reliability ($r = .78$), convergent validity with alternative measures of negative affect, and predictive validity for bulimic symptom onset (Stice et al., 2008; Watson & Clark, 1992); $\alpha = .93$ at pretest.

Eating disorder symptoms. The semistructured Eating Disorder Diagnostic Interview (Stice et al., 2013) assessed eating disorder symptoms and *DSM–5* eating disorder diagnoses (though we focused on subthreshold AN rather than atypical AN). Items were generated to capture the symptoms of each of the *DSM–5* eating disorders, rather than through factor analyses. It assessed symptoms on a month-to-month basis in the past 3 months before the pretest assessment and since the last assessment at posttest. Eating Disorder Diagnostic Interview eating disorder diagnoses have shown 1-week test–retest reliability ($\kappa = .79$), interrater agreement ($\kappa = .75$), sensitivity to detecting intervention effects, and correlate with functional impairment, emotional distress, and mental health treatment utilization (Stice et al., 2008, 2017). We also calculated a continuous symptom composite, which reflected symptoms in the past month (frequency of binge eating, vomiting, laxative/diuretic use, fasting, and excessive exercise; yes/no questions regarding distress about binge eating and key features of binge eating [e.g., rapid eating, feeling disgusted, depressed or guilty about binge eating]; and Likert questions about overvaluation of weight/shape, fear of weight gain/becoming fat, use of behaviors to avoid weight gain, and feeling fat; less than 85% of expected weight). This composite has shown internal consistency ($\alpha = .92$), interrater agreement (ICC $r = .93$), 1-week test–retest reliability (ICC $r = .95$), convergence with alternative measures of eating disorder symptoms, and sensitivity to detecting the effects of eating disorder prevention and treatment interventions (Stice, Rohde, et al., 2015; Stice, Telch, & Rizvi, 2000); $\alpha = .80$ at pretest. We examined the percentage of participants who reported no binge eating or compensatory behaviors in the past month at posttest because other trials have reported this outcome (e.g., Fairburn et al., 2015).

Statistical Method

Preliminary analyses. We examined the distribution of outcomes and applied normalizing transformations as necessary to reduce the potential for disproportionate influence of outliers and decrease residual heterogeneity. Comparisons between conditions were made for pretest values of outcomes, demographics (race, ethnicity, age, and parental education) and ancillary treatment to assess whether randomization created equivalent groups. Comparisons between participants who completed all assessments and those who did not were made for study condition, pretest values of outcomes, and demographics to assess differential attrition. We evaluated the 10-week test–retest reliability of self-reported intervention target measures in waitlist controls.

Model building. Intent-to-treat analyses of condition effects for nonfMRI outcomes were evaluated with models fit with SAS 9.2 PROC MIXED. Individual variability in outcomes was modeled separately at posttest, as a function of condition (0 = waitlist; 1 = BPT), adjusting for pretest version of the outcome. Partially nested models accounted for group variability, where participants were clustered in BPT groups; group level variance was nonsignificant for all models and the clustering effects were removed. Effect sizes were computed as Cohen's *d* by dividing the difference between the estimated means of conditions at posttest by the baseline pooled *SD*; a $d = .30$, .50, and .80 correspond to a small, medium, and large effect, respectively. Missing data ranged from 0% to 14% at pretest and 8% to 28% at posttest. Missing data were imputed using PROC MI and the imputation model included demographics (age, race, and parent education) and ancillary treatment. Based on the recommendation to impute many more data sets than previously recommended to render multiple imputation equivalent to full-information maximum likelihood (Graham, Olchowski, & Gilreath, 2007), we imputed data in 50 data sets. The data sets were analyzed separately and model parameters and standard errors were combined using SAS PROC MIANALYZE.

MRI acquisition. Data were acquired using Siemens Skyra 3 Tesla MRI scanners. A 32-channel head coil acquired data from the entire brain. We collected 240 scans for each paradigm. Functional scans used a $T2^*$ weighted gradient single-shot echo planar imaging sequence (echo time = 25 ms, repetition time = 2,000 ms, flip angle = 90°) with an in plane resolution of 2.0×2.0 mm^2 (64×64 matrix; 256×256 mm^2 field of view). To cover the whole brain, 72 slices, each 2 mm, were acquired along the anterior commissure–posterior commissure transverse, oblique plane as determined by the midsagittal section. Structural scans were collected using an inversion recovery T1-weighted sequence in the same orientation as the functional sequences to provide detailed anatomical images aligned to the functional data. High-resolution structural MRI sequences (field of view = 256×256

mm^2, 256 × 256 matrix, thickness = 1.0 mm, and in-plane resolution of 1 × 1 mm) were acquired.

FMRI data preprocessing. DICOM images were converted to NIfTI format via MRIConvert (http://lcni.uoregon.edu/~jolinda/MRIConvert/), and nonbrain tissue was removed using FSL's Brain Extraction Tool (Smith, 2002). Data were preprocessed and analyzed using statistical parametric mapping (SPM) Version 12 (SPM12; Wellcome Department of Imaging Neuroscience, University College London) in MATLAB. During preprocessing, functional data were preprocessed as follows: (a) adjusted for variation in magnetic field distortion using field maps, (b) realigned to the mean functional from that run and coregistered with the anatomical, and (c) normalized to Montreal Neurological Institute (MNI) space using the DARTEL template and deformation fields output. Functional data were smoothed to 6-mm Gaussian full width at half maximum. A 128-s high-pass filter removed low-frequency noise and signal drift. Head motion greater than 2 mm or degrees in any direction was our a priori exclusion criteria. Artifact Detection Toolbox (Gabrieli Laboratory, McGovern Institute for Brain Research, Cambridge, MA) was used to detect global mean response spikes and motion outliers. Motion parameters were used as regressors and outlier image volumes (<2 mm) were deweighted during individual-level model estimation. Anatomical images were coregistered to the mean functional image and segmented into six tissue types using unified segmentation approach (Ashburner & Friston, 2005). DARTEL was used to create a group anatomical template, transformations from which were applied to warp functional data to the ICBM-152 template supplied with SPM12 (Ashburner, 2007). fMRI data of two participants were collected with an acquisition error, one participant missed too many images during both scans (she fell asleep, as detected by an eye-tracker we use to monitor participants), and one participant failed to meet the movement inclusion criteria at post scan (i.e., within-run movement exceeded 2 mm in translational movement and 2° in rotational movement), resulting in complete pretest and posttest scan data for 72 participants (BPT n = 38; control n = 34).

fMRI data analysis. A general linear model was defined for each participant with two regressors of interest each for the food image paradigm (high-calorie foods and low-calorie foods) and the model paradigm (thin models and average-weight models) modeled as events. Individual maps were constructed to compare the activations within each participant during exposure to high- versus low-calorie foods and during exposure to thin versus average-weight models and were constructed for pretest and posttest separately. We conducted 2 (Group: BPT, control) × 2 (Time: pretest, posttest) repeated-measures analysis of variance models on blood-oxygen-level-dependent (BOLD) responses to examine group differences in change in neural response to high-calorie foods versus low-calorie foods and to thin models versus average-weight models between conditions using these individual maps. Fasting hours before each scan and BMI were included as covariates.

Whole-brain analyses were conducted. To correct for multiple comparisons across brain voxels, we calculated the cluster extent thresholds for each analysis at $p < .001$ with the SPM cluster size threshold tool (https://github.com/CyclotronResearchCentre/SPM_ClusterSizeThreshold). Results indicated that activity surviving a threshold of $p < .001$, with a cluster (k) ≥30 contiguous voxels, being statistically significant at family wise error (FWE)-corrected $p = .05$ across the whole brain.

We performed a priori regions-of-interest (ROI) analyses within the caudate to test for changes in neural activity within this region in response to thin- versus average-weight models, seeking to replicate previous findings (Stice et al., 2015). We also tested for changes in caudate response to high- versus low-calorie foods, as food cue exposure activates the caudate (Tang, Fellows, Small, & Dagher, 2012). Further, ROI analyses were performed to test for significant changes in the ventromedial prefrontal cortex (vmPFC) in response to the same contrasts, as the vmPFC dynamically tracks the subjective value of stimuli, including images of attractive people (Smith et al., 2010; Winston, O'Doherty, Kilner, Perrett, & Dolan, 2007) and foods (Hare, Camerer, & Rangel, 2009). For ROI analyses, 10-mm spheres were drawn centering on the peak voxel from published articles (Hare et al., 2009; Smith et al., 2010; Stice et al., 2015; Winston et al., 2007). Peak activity with values of $p < .05$ corrected using voxel-level familywise error rate (p_{FWE}) over the 10-mm sphere were considered significant. Bonferroni corrections were then used to correct for the number of regions of interest ($p = .05/2$ regions $= 0.025$). Effect sizes (r) were derived from the Z values ($Z/\sqrt{N}$). fMRI data were inspected to insure that influential outliers (parameter estimates exceeding 3 SDs from the mean parameter estimate) did not drive significant effects.

We investigated the 10-week test–retest reliability of brain activation in response to the paradigms in waitlist controls by calculating intraclass-correlation coefficients (ICC). We analyzed neural response to each of the two events (pre, post) per paradigm (e.g., thin-models and average-weight models) with one-sample t tests, using the beta-images obtained in the single subject analysis as input data. We calculated the ICC on a ROI basis, as this approach is less prone to the effects of noise than calculating ICCs for each voxel, choosing the caudate and vmPFC ROIs. We also calculated reliability statistics for one reference region per event. We used the single voxel with the highest Z score and largest cluster size (i.e., most significant) for each event at baseline as reference regions, as these were most strongly activated by the paradigms. Subject-level parameter estimates (averaged across all voxels in the ROIs) were extracted from SPM for each task and scanning session, and analyzed with the SPSS Version 24. We then calculated ICCs for each ROI between the 2 scan days. We assessed reliability with a two-way mixed effects ICC (Shrout & Fleiss, 1979). Due to the possibility that participants might habituate to the stimuli over time, we employed a "consistency" measure of ICC, rather than testing the absolute agreement between scan days.

Results

Preliminary Analyses

Distributions of outcomes approximated normality, except eating disorder symptoms, which were normalized with natural log transformations. BPT and control groups did not differ on demographics, including the percent recruited from universities (78%), ancillary treatment (25% control and 16% BPT) or outcome measures at pretest (see Table 1); Table 2 provides means and standard deviations for outcomes at pretest and posttest for each group.

Table 1
Study Demographic and Pretest Characteristics by Study Condition

Demographic	Waitlist control	Body Project Treatment	Test statistic	*p*
Age, *M* (*SD*)	22.09 (3.59)	21.83 (3.61)	$t(98) = .36$	.722
Hispanic, %	16.7	9.8	$\chi^2(1,99) = 1.02$	.312
Race, %			$\chi^2(3,98) = 1.01$	.798
Asian	18.8	20.0		
Black or African American	4.2	4.0		
Caucasian	77.1	74.0		
Other	.0	2.0		
Maximum parental education, %			$\chi^2(4,100) = 5.44$	.245
Some high school	.0	5.9		
High school graduate	2.0	5.9		
Some college	20.4	15.7		
College graduate	32.7	21.6		
Advanced degree	44.9	51.0		
Body mass index, *M* (*SD*)	25.38 (6.81)	25.68 (6.79)	$t(98) = -.22$	.824
Binge eating frequency, monthly, *M* (*SD*)	5.57 (5.77)	7.02 (8.00)	$t(98) = 1.70$	.093
Compensatory behaviors, monthly, *M* (*SD*)	6.42 (16.28)	2.31 (5.80)	$t(98) = -1.04$	.300
No binge eating at pretest, %	22.4	17.6	$\chi^2(1,100) = .36$	.548
No compensatory at pretest, %	22.3	25.5	$\chi^2(1,100) = .12$	.722
Diagnosis, %				
Full syndrome AN	2.1	5.9	$\chi^2(1,100) = .92$	.337
Full syndrome BN	28.6	41.2	$\chi^2(1,100) = 1.75$	.186
Full syndrome BED	12.2	13.7	$\chi^2(1,100) = .05$	.337
Full syndrome PD	12.2	9.8	$\chi^2(1,100) = .15$	.697
Partial syndrome AN	6.3	2.0	$\chi^2(1,100) = .05$	.826
Partial syndrome BN	30.6	23.5	$\chi^2(1,100) = .64$	.425
Partial syndrome BED	8.2	3.9	$\chi^2(1,100) = .80$	.372

Note. AN = anorexia nervosa; BN = bulimia nervosa; BED = binge eating disorder; PD = purging disorder.

Participants in the BPT condition and control condition who provided fMRI data also did not differ on these variables. Those recruited from universities did not differ from those not recruited at universities on any demographic characteristics or baseline variables. At baseline 22% of control and 29% of BPT participants were taking medications for eating/weight problems or mental health problems, but rates did not significantly differ, $\chi^2(1,100) = 0.63$, $p = .427$.

Retention was 92% at posttest. BPT participants either attended or made-up an average of 6.6 of the eight sessions ($SD = 2.4$); 63% attended or made-up all eight sessions and 12% attended or made-up fewer than two sessions. Among those who missed a session, 88% received an individual make-up session. Participants completed 67% of the home exercises. Rates of missing data were 9%–27% at posttest and the missing-completely-at-random assumption remained tenable; Little's missing-completely-at-random test, $\chi^2(183) = 160.34$, $p = .885$. Attrition was not related to condition, demographics, or pretest values of outcome measures.

The 10-Week Test–Retest Reliability of Intervention Target Measures

We found robust activity in visual processing regions in response to all four events: inferior occipital gyrus in response to thin models (MNI coordinates: 45, −82, −7, Z = infinity, $k = 8249$) and average-weight models (MNI coordinates: 30, −88, −13, Z = inf, $k = 12{,}202$), lingual gyrus (MNI coordinates: −9, −97, −4, Z = inf, $k = 16{,}301$) in response to high-calorie foods, and cuneus (MNI coordinates: 15, −100, 8, Z = inf, $k = 12{,}721$) in response to low-calorie foods. Amplitude of BOLD response in these regions showed moderate to good test–retest reliability (thin models ICC = 0.74, $p < .001$; average-weight models ICC = 0.79, $p < .001$; high-calorie foods ICC = 0.66, $p < .001$; low-calorie foods ICC = 0.57, $p < .001$). We repeated these analyses for the caudate and vmPFC ROIs. In the model paradigm, the amplitude of BOLD responses to thin models showed moderate test–retest reliability in the caudate ROI (ICC = 0.53, $p < .01$) and vmPFC (ICC = 0.49, $p < .01$; see Table 3). The amplitude of BOLD responses to average models showed moderate test–retest reliability in the caudate ROI (ICC = 0.42, $p < .01$), but lower reliability in the vmPFC ROI (ICC = 0.37, $p < .05$). In the food image paradigm, the amplitude of BOLD responses to high- and low-calorie foods in the caudate and vmPFC showed low reliability (ICC range = .16–.37), except in the vmPFC ROI in response to high-calorie foods (ICC = 0.45, $p < .01$; see Table 3). Self-report measures of the intervention targets showed higher test–retest reliability (attractiveness of thin models: $r = .82$; attractiveness of average-weight models: $r = .83$; willingness to pay high-calorie foods: $r = .80$; willingness to pay low-calorie foods: $r = .80$; palatability high-calorie foods: $r = .69$; palatability low-calorie foods: $r = .59$).

Intervention Effects on Neural Response to High-Calorie Binge Foods

Whole brain analyses comparing BPT and control participants on change in BOLD activity in response to high-calorie foods > low-calorie foods showed a significant Group × Time interaction in the right parahippocampal gyrus ($r = .48$). The interaction revealed that the BPT group showed greater decreases in BOLD

Table 2
Means and Standard Deviations for Outcomes by Condition at Pretest and Posttest

Variable	Pretest		Posttest	
	M	*SD*	*M*	*SD*
Eating disorder symptoms				
Body Project Treatment	1.56	.26	1.29	.40
Waitlist control	1.61	.22	1.46	.26
Palatability high-calorie binge foods				
Body Project Treatment	6.02	1.36	4.51	1.93
Waitlist control	5.91	1.31	5.47	1.66
Palatability low-calorie foods				
Body Project Treatment	5.04	1.25	4.91	1.39
Waitlist control	4.95	1.25	4.83	1.15
Monetary value high-calorie binge foods				
Body Project Treatment	3.66	1.29	3.17	1.27
Waitlist control	3.66	1.32	3.43	1.20
Monetary value low-calorie foods				
Body Project Treatment	3.82	1.03	4.12	1.29
Waitlist control	3.88	.95	3.97	1.06
Attractiveness thin models				
Body Project Treatment	6.45	1.14	6.08	1.55
Waitlist control	6.37	1.31	6.21	1.63
Attractiveness average-weight models				
Body Project Treatment	4.86	1.33	5.33	1.62
Waitlist control	4.62	1.52	4.50	1.57
Thin-ideal internalization				
Body Project Treatment	3.66	.48	3.30	.39
Waitlist control	3.77	.40	3.69	.51
Body dissatisfaction				
Body Project Treatment	4.01	.82	3.33	.80
Waitlist control	4.28	.58	4.05	.86
Negative affect				
Body Project Treatment	3.46	.76	2.47	.89
Waitlist control	3.56	.77	3.11	.88

Note. Log transformed values of eating disorder symptoms reported.

activity in this region compared to controls (Figure 3B). Because the parahippocampal gyrus responses at pretest differed between BPT and control participants (Figure 3B), it is possible that this interaction is partially driven by differences in BOLD activity at pretest. We therefore extracted the main effect parameter estimates at the individual level from the peak coordinate at pretest. Although the groups did not differ in parahippocampal gyrus activity in response to high-calorie foods (M_{BPT}= 0.01, SD = 0.50; $M_{control}$ = −0.03, SD = 0.37), they did differ in parahippocampal gyrus response to low-calorie foods (M_{BPT} = −0.07, SD = 0.30; $M_{control}$ = 0.11, SD = 0.34), $t(69) = -2.44$, $p < .05$, suggesting that this interaction was partly driven by neural differences at pretest. This may have occurred because when SPM tests for time-by-condition interactions, it searches for instances in which the change in the intervention condition is maximally different than change in the control condition, which appears to result in the identification of peaks in which the BOLD signal in the intervention condition decreases and BOLD signal in the control condition increases. We conducted ROI analyses using peaks in the vmPFC and caudate; there were no significant Group × Time interactions in these ROIs.

We tested whether pre–post changes in BOLD response in the right parahippocampal gyrus correlated with pre–post changes in palatability and monetary valuation ratings of the high-calorie foods and low-calorie foods across all participants. We extracted parameter estimates of the significant Group × Time interaction in the parahippocampal gyrus and tested whether change in parameter estimates correlated with change in palatability and monetary valuation ratings of the high-calorie foods and low-calorie foods. Bonferroni corrections were used to correct for the number of tests. There were no significant correlations between changes in BOLD activity in the parahippocampal gyrus and changes in palatability and monetary valuation ratings of the high-calorie foods and low-calorie foods (smallest p = .20).

Intervention Effects on Neural Response to Thin Models

Whole brain analyses comparing the BPT and control participants on change in BOLD response to thin models > average-weight models showed significant group x time interactions in the right posterior cingulate cortex (PCC; r = .55), left posterior cerebellar lobe (r = .55), right superior temporal gyrus (STG; r = .52), left middle temporal gyrus (MTG; r = .51), left dorsolateral prefrontal cortex (dlPFC: r = .50), and left precuneus (r = .44; Figure 2A–F). The BPT group showed significantly greater decreases in BOLD activity in these six regions than controls. The groups did not differ in BOLD activation in any of these regions at pretest, except the dlPFC in response to thin models (M_{BPT} = 0.12, SD = 0.33; $M_{control}$ = −0.08, SD = 0.38), $t(69) = 2.32$, $p < .05$, suggesting that this interaction is partially driven by differences in BOLD activity in response to thin models at pretest.

We conducted ROI analyses using peaks in the caudate and vmPFC identified in past studies. We found a significant group x time interaction in the vmPFC (r = .51; MNI: 6, 56, −13, Z = 4.28, k = 14, p_{FWE} = 0.007; Figure 3a). The interaction in the vmPFC revealed that the BPT group showed significantly greater decreases in BOLD activity versus controls. There was no significant group x time interaction in the caudate ROI.

We tested whether pre–post changes in BOLD response correlated with pre–post changes in attractiveness ratings of the thin and average-weight models across all participants. There were no significant correlations between change in parameter estimates in the peak coordinates and changes in attractiveness ratings across all participants.

Exploratory fMRI Analyses

Given the possibility that effects might be different for participants with restricting AN versus the other eating disorders that involve binge eating and compensatory weight control behaviors, we conducted exploratory analyses regarding the nature of the effects when we excluded the three participants with restricting AN. In these analyses, the parahippocampal gyrus peak from the high-calorie food image paradigm became nonsignificant and the STG, dlPFC, and precuneus peaks from the thin model image paradigm because nonsignificant, though we did observe a significantly greater reduction in caudate response to thin-models in BPT versus control participants (r = .53, p_{FWE} = 0.002). The caudate is another region that encodes the reward value of stimuli, and as such, this effect converges with the vmPFC effect that remained significant in these exploratory analyses.

Table 3
Significant Condition × Stimulus × Time Interactions in Brain Activation During Exposure to Food and Model Images: Flexible Factorial 2 × 2: Intervention (n = 38) Versus Control (n = 34)

Contrasts and regions	*k*	*Z*	MNI coordinates	*r*
High-calorie foods > low-calorie foods				
Baseline > follow-up: Intervention > control				
Parahippocampal gyrus	32	4.08	18, −16, −25	.45
Thin models > average-weight models				
Baseline > follow-up: Intervention > control				
Posterior cingulate cortex	33	4.69	0, −31, 38	.55
Posterior cerebellar lobe	63	4.63	−27, −79, −31	.55
Posterior cerebellar lobe		4.19	−21, −73, −34	.49
Posterior cerebellar lobe		3.48	−36, −67, −28	.41
Superior temporal gyrus	57	4.41	48, −31, 2	.49
Superior temporal gyrus		4.36	48, −52, 11	.48
Superior temporal gyrus		3.75	48, −40, 17	.41
Middle temporal gyrus	38	4.36	−51, −31, −7	.48
Middle temporal gyrus		4.25	−60, −31, −7	.47
Dorsolateral prefrontal cortex	33	4.25	−24, 47, 38	.47
Dorsolateral prefrontal cortex		3.77	−21, 56, 29	.42
Precuneus	35	3.74	−9, −64, 44	.41
Precuneus		3.60	0, −61, 47	.40

Note. MNI = Montreal Neurological Institute. Peaks within the regions were considered significant at $p < .001$, $k \geq 30$, $p < .05$, corrected for multiple comparisons across the entire brain. Fasting, body mass index, and scan site were controlled for in the analyses.

Intervention Effects on Self-Reported Outcomes

Intent-to-treat analyses of group effects at posttest for self-reported continuous outcomes are shown in Table 4. BPT versus control participants showed significantly greater reductions in thin-ideal internalization ($d = .38$), palatability ratings of high-calorie foods ($d = .78$), body dissatisfaction ($d = .83$), and negative affect ($d = .76$), as well as significantly greater increases in attractiveness ratings for average-weight models ($d = .44$). Critically, BTP versus control participants showed significantly greater reduction in the eating disorder symptom composite ($d = .59$). Baseline BMI did not significantly moderate the effects on eating disorder symptom reduction. A significantly higher number of BPT participants showed reliable change (reductions) in eating disorder symptoms (65%) than control participants (41%), $\chi^2(1, 92) = 4.67$, $p = .031$, following Jacobson and Truax (1991). Further, 39% of BPT participants and 21% of control participants reported no binge eating or compensatory behaviors in the past 30-days, but this effect was only marginal, $\chi^2(1, 92) = 3.92$, $p = .070$.

Dose-Response Relations

Establishing change in eating disorder symptoms as a function of sessions attended was inconclusive, in part, due to a lack of variability in this dosage measure. A large majority of BPT participants attended or made up seven (10%) or eight (63%) sessions and the correlation between sessions attended and eating disorder symptom composite reduction was small and nonsignificant, $r = .16$, $p = .301$. Due to this restriction in range, we also tested whether eating disorder symptom reduction correlated with degree of verbal participation in sessions, $r = .13$, $p = .384$, and homework completion, $r = .27$, $p = .074$.

Sensitivity Analyses

We tested whether the intervention effects for the outcomes reported in Table 4 differed significantly when the 12 participants who were not randomized to condition because we could not run a group treatment over the holidays were excluded (12% of all participants). The new parameter estimates for the tests of intervention effects were not outside the 95% confidence intervals of the original parameter estimates for the tests of intervention effects from the full sample. We likewise tested whether the intervention effects for these outcomes differed significantly when the 33 participants who were interviewed by an assessor at posttest who could have known the condition to which the participants had been assigned were excluded. The new parameter estimates for the tests of intervention effects were not outside the 95% confidence intervals of the original parameter estimates of the tests for intervention effects from the full sample. Results suggest that the inability to randomize all participants to condition and to completely conceal allocation assignment from assessors did not compromise the tests of the intervention effects.

Discussion

Results revealed that, compared to waitlist controls, BPT participants showed significantly greater reductions in the PCC, posterior cerebellar lobe, temporal regions (STG, MTG), dlPFC, precuneus, and vmPFC response to thin versus average-weight

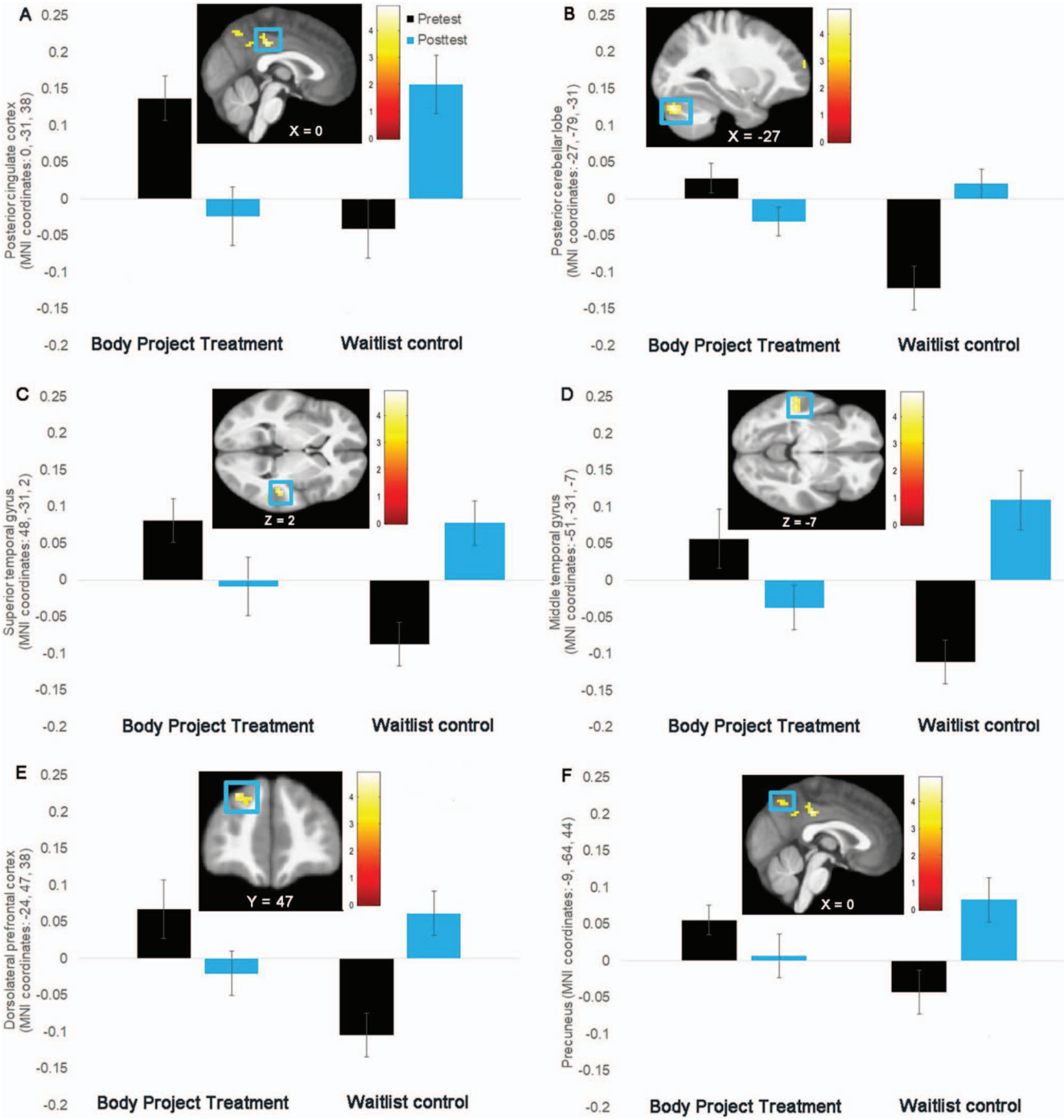

Figure 2. Greater pre- to post-BOLD response decreases in (A) posterior cingulate cortex (Montreal Neurological Institute [MNI] coordinates: 0, −31, 38, $Z = 4.69$, $k = 33$), (B) posterior cerebellar lobe (MNI coordinates: −27, −79, −31, $Z = 4.63$, $k = 63$), (C) superior temporal gyrus (MNI coordinates: 48, −31, 2, $Z = 4.41$, $k = 57$), (D) middle temporal gyrus (MNI coordinates: −51, −31, −7, $Z = 4.36$, $k = 38$), (E) dorsolateral prefrontal cortex (MNI coordinates: −24, 47, 38, $Z = 4.25$, $k = 33$), and (F) precuneus (MNI coordinates: −9, −64, 44, $Z = 3.74$, $k = 35$) in response to the contrast thin model > average-weight model images in the Body Project Treatment versus waitlist control condition. BOLD = blood-oxygen-level-dependent. See the online article for the color version of this figure.

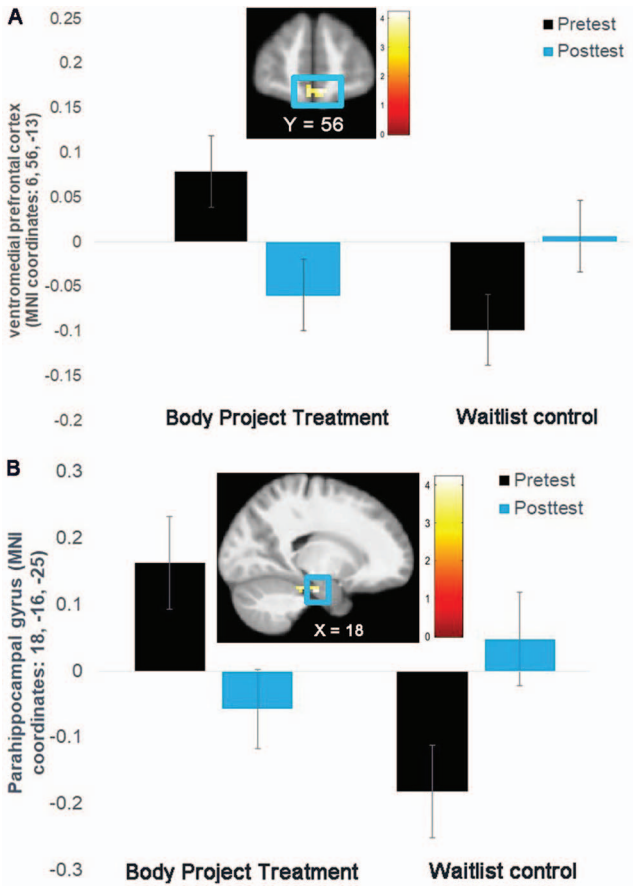

Figure 3. Greater pre- to post-BOLD response decreases in (A) the ventromedial prefrontal cortex (Montreal Neurological Institute [MNI] coordinates: 6, 56, −13, $Z = 4.28$, $k = 14$, peak activity with values of $p < .05$ corrected using voxel-level familywise error rate = 0.007) in response to the contrast thin model > average-weight model images and (B) in the parahippocampal gyrus (MNI coordinates: 18, −16, −25, $Z = 4.08$, $k = 32$) in response to the contrast high-calorie binge food images > low-calorie food images in the Body Project Treatment condition versus waitlist control condition. BOLD = blood-oxygen-level-dependent. See the online article for the color version of this figure.

models. The PCC, posterior cerebellar lobe, and MTG are involved in visual processing, memory, and attentional motivation (Leech & Sharp, 2014; Stoodley, Valera, & Schmahmann, 2012). The PCC and precuneus are also activated during processing of emotional and social stimuli (Laird et al., 2011). The STG has been implicated in face perception (Haxby, Hoffman, & Gobbini, 2000). The dlPFC, precuneus, and vmPFC are part of the network of brain regions involved in reward processing. The dlPFC is involved in the representation and integration of potential rewards (Miller & Cohen, 2001) and transmits reward information to the mesolimbic and mesocortical dopamine systems, such as the nucleus accumbens and ventral tegmental area (Ballard et al., 2011). The dlPFC is also involved in working memory and top-down cognitive control (MacDonald, Cohen, Stenger, & Carter, 2000) and has been found to be responsive to body shape processing (Uher et al., 2005). The precuneus is functionally connected with reward-related regions, such as the striatum, through communication of the salience of visual stimuli (Engelmann et al., 2012). Activity in the vmPFC reflects the internalized reward value of stimuli and has been found to respond to facial attractiveness (Smith et al., 2010; Winston et al., 2007). Exploratory analyses indicated that although the vmPFC peak remained significant when three participants with restricting AN were excluded, the dlPFC and precuneus effects became nonsignificant. However, those exploratory analyses revealed a significant reduction in caudate response to thin models, which has been implicated in encoding reward valuation, like the vmPFC, and converges with evidence that participants who completed the Body Project eating disorder prevention program also showed a significant reduction in caudate response to thin models (Stice et al., 2015). Overall, our findings suggest that BPT participants show greater decreases in regions involved in visual processing, memory, attention, and reward processing in response to thin versus average-weight models than waitlist controls. This interpretation is consistent with the increase in attractiveness ratings of the average-weight models in the BPT group. However, pre–post changes in BOLD response did not correlate with pre–post changes in attractiveness ratings of the thin and average-weight models.

Results also indicated that BPT participants showed greater reductions in thin-ideal internalization and greater increases of attractiveness ratings of average-weight models, but no decreases in attractiveness ratings of thin models. It is tempting to argue that the self-report measures are more sensitive than the fMRI measures of this intervention target, but the effects for self-report outcomes may be larger because expectancies and demand characteristics contribute to changes in self-reported outcomes. Reductions in eating disorder symptoms correlated with reductions in thin-ideal internalization ($r = .27$) and with increases in attractiveness ratings of average-weight models ($r = -.24$), though symptom change did not correlate with change in attractiveness ratings of thin models ($r = -.06$). This mixed pattern of findings with regard to the validity of the fMRI measures of target engagement suggests that it might be useful to consider using implicit measures of valuation of the thin beauty ideal, such as the Implicit Association Test (Greenwald, McGhee, & Schwartz, 1998) to measure target engagement of BPT.

With regard to the high-calorie food intervention target, BPT participants showed a pre–post reduction in parahippocampal gyrus responsivity to high-calorie versus low-calorie foods. The parahippocampal gyrus is thought to play a role in emotion memory coding (LaBar & Cabeza, 2006) and hedonic processing of feeding and incentive motivation (Tataranni et al., 1999). The parahippocampal gyrus has found to respond to palatable food cues (Pelchat, Johnson, Chan, Valdez, & Ragland, 2004) and anticipated palatable food intake (Bohon, Stice, & Spoor, 2009). Parahippocampal activity is also positively associated with trait-based food craving (Chen, Dong, Jackson, Zhuang, & Chen, 2017) and inversely linked to measures of satiety (Leidy, Lepping, Savage, & Harris, 2011). This result may thus suggest that BPT participants showed a reduction in emotional memories and incentive motivation for palatable high-calorie food relative to low-calorie food versus waitlist controls. However, change in parahippocampal activation did not correlate with palatability and monetary valuation ratings of the pictured foods. Further, this interaction was partially driven by less parahippocampal activity in response to low-calorie food in the BPT group relative to the

Table 4
Test of Posttest Group Differences Adjusting for Pretest Scores

Outcome	Estimate	*SE*	*t*	*p*	*d*
Eating disorder symptoms	−.142	.069	−2.06	.040	.59
Palatability high-calorie binge foods	−1.043	.348	−2.99	.003	.78
Palatability low-calorie foods	.022	.222	.10	.922	.02
Monetary value high-calorie binge foods	−.260	.201	−1.30	.195	.20
Monetary value low-calorie foods	.203	.178	1.14	.254	.20
Attractiveness thin models	−.188	.263	−.71	.475	.15
Attractiveness average-weight models	.630	.264	2.39	.017	.44
Thin-ideal internalization	−.318	.080	−3.99	<.001	.72
Body dissatisfaction	−.593	.166	−3.57	<.001	.83
Negative affect	−.578	.160	−3.62	<.001	.76

waitlist control group at pretest. This one effect provides limited evidence that the BPT reduced valuation of high-calorie foods.

BPT participants showed greater reductions in palatability ratings of high-calorie binge foods, but no significant change in palatability ratings of low-calorie foods or monetary valuation of high-calorie or low-calorie foods. Thus, there was also limited evidence that BPT changes valuation of binge foods according to the self-report measures of this intervention target. This pattern of findings may imply that it would be useful to refine the BPT so that it is more effective in reducing valuation of binge foods. However, it is also possible that overvaluation of high-calorie binge foods does not play a role in the maintenance of disordered eating.

We assessed the test–retest reliability of the neural activation in response to models and foods in controls to test temporal stability of our fMRI paradigms. Overall, the highest ICC values were obtained for the most strongly activated brain regions (i.e., reference regions) within each paradigm. The amplitude of BOLD responses in the caudate and vmPFC ROIs showed low to moderate reliability. In particular, the test–retest reliability of caudate and vmPFC response to low-calorie foods was low. In general, test–retest reliability of BOLD response is influenced by numerous factors such as scanner noise, motion, and the time of day at which scanning occurred (Gorgolewski, Storkey, Bastin, Whittle, & Pernet, 2013). It is possible that other ROIs than the caudate and vmPFC might show more consistent within-subject reliability. Indeed, our reference regions suggest that activity in visual processing regions show greater temporal reliability. Overall, our results suggest that our fMRI paradigms showed moderate 10-week test–retest reliability. The 10-week test-retest reliabilities of the self-report measures of intervention targets were higher (M_r = .76), suggesting that more confidence can be placed in these target measures.

Results also indicated that BPT participants showed significantly greater reductions in eating disorder symptoms, body dissatisfaction, and negative affect, as well as marginally greater abstinence from binge eating and compensatory behaviors. It was noteworthy that the effect size for symptom reduction in this trial (d = .59) was smaller than the effect observed in an earlier trial with a similar minimal intervention control condition (d = .95; Stice et al., 2015). This effect may have been smaller because we refined the intervention to encourage participants to reduce their eating disordered behaviors before we had completed the activities designed to reduce pursuit of the thin ideal. In the two earlier trials the first three sessions focused on reducing pursuit of the thin ideal. We had initially reasoned that it might be best to reduce pursuit of the thin ideal before asking participants to begin relinquishing unhealthy compensatory weight control behaviors used to achieve the thin ideal, but the evidence that expressly encouraging rapid symptom reduction produces greater abstinence rates (MacDonald et al., 2017) led us to refine the intervention script to encourage early symptom reduction. Theoretically, reducing pursuit of the thin ideal promotes body satisfaction and potentially a willingness to reduce eating disordered behaviors motivated by this goal, such as fasting, vomiting, and laxative abuse. This analysis, which is consistent with the dual pathway model of eating pathology (Stice & Van Ryzin, 2019), suggests that pursuit of the thin ideal is a central driver for eating pathology. This finding converges with evidence that overvaluation of weight and shape is the most central symptom of BN (Levinson et al., 2017). Thus, it may be best for future trials to use the earlier version of the intervention script that focuses on reducing pursuit of the thin ideal before encouraging participants to decrease their eating disordered behaviors.

The first two trials of the BPT indicated that participants who attended more than half of the sessions showed larger reductions in outcomes than those who attended fewer sessions (Stice et al., 2015, 2019), providing evidence of a dose-response relation. However, session attendance did not correlate with the degree of symptom reductions in the present trial. One possible explanation is that because we refined the intervention to improve engagement, retention was so high that it constrained variability in session attendance. In the first trial only 16% of participants attended or made up all eight sessions, whereas 63% of participants in the present trial attended or made up all eight sessions. Symptom reduction showed a marginal relation with homework completion, but not with verbal participation in sessions. It is also possible that the lack of a dose-response relation was due to the intervention evaluated in the present trial not being as effective as the version evaluated in earlier trials. Perhaps most critically, the evidence that BPT produced abstinence from binge eating and compensatory behaviors for 39% of participants in the present trial and 55% in a past trial (Stice et al., 2019), which is higher than the 23%–34% abstinence rate produced by CBT-E, the 33% abstinence rate produced by ICAT, or the 35% abstinence rate produced by IPT (Fairburn et al., 2015; Wonderlich et al., 2014), suggests that the eight-session duration of BPT is efficacious. It is possible that

there were differences in the nature of the samples recruited in these trials that contributed to variation in the observed abstinence rates. Although all of these trials excluded individuals with suicidal ideation and substance misuse disorders, Fairburn and colleagues (2015) recruited exclusively from clinics, whereas Wonderlich and colleagues (2014) and the present trial recruited both from clinical settings and the community. A direct comparison trial will be necessary to definitively answer the question of relative efficacy of these transdiagnostic eating disorder treatments.

It is important to consider the limitations of this study. First, we had valid fMRI data from only 72 participants, which limited sensitivity to detect small effects. Second, we conducted multiple inferential tests in this trial. However, the fact that 60% of the effects reported in Table 3 were statistically significant, which is considerably higher than the 5% that would have been expected based on chance alone, implies it is unlikely that we are interpreting chance findings. Third, we planned on excluding individuals with suicidal ideation and substance misuse, which could have limited generalizability of the findings. However, no individuals were excluded for these reasons, suggesting that the findings should be generalizable. Further, other eating disorder treatment trials have had similar exclusion criteria (e.g., Fairburn et al., 2015; Kristeller et al., 2013; Wonderlich et al., 2014). Fourth, we did not have a large enough sample to allow analyses that tested whether BPT produces effects for each eating disorder type. Fifth, we did not use scales that have been shown to assess only a single latent construct. Sixth, we were not able to randomize all participants to condition and to conceal allocation assignment from assessors in all cases, which reduces the confidence that can be placed in the findings. Fortunately, the deviation from full random assignment did not result in systematic differences between participants assigned to the two conditions at baseline and sensitivity analyses suggested that the deviation from full random assignment and the incomplete allocation concealment did not bias the tests of intervention effects.

In conclusion, this report provides evidence that the dissonance-based eating disorder treatment produced significant reductions in responsivity of regions implicated in reward processing to thin models, but not to high-calorie binge foods, compared to waitlist controls, providing evidence of target engagement based on an objective biological outcome. Results also provided evidence that BPT participants reported greater reductions in pursuit of the thin ideal and increased attractiveness ratings of average-weight models, providing further evidence of target engagement. In addition, BPT participants reported greater reductions in palatability ratings of high-calorie binge foods, providing some evidence of target engagement for devaluation of binge foods. BPT participants also showed greater reductions in eating disorder symptoms, body dissatisfaction, and negative affect, and marginally greater reductions in abstinence from binge eating and compensatory behaviors. Yet, the effect size for symptom reduction was smaller in the present trial than the past trial that used the original intervention script, suggesting that BPT is more effective in decreasing eating disorder symptoms if it first reduces pursuit of the thin ideal before encouraging reductions in eating disorder symptoms, many of which are used to pursue the thin ideal. We thus recommend that future trials use the older version of the BPT. Results from this trial, and the previous trials of BPT (Stice, Rohde, et al., 2015, 2019) imply that the new dissonance-based eating disorder treatment produces clinically meaningful reductions in outcomes and is more cost-effective than 20-session individually administered treatments for eating disorders. It will be important to conduct fully powered efficacy and effectiveness trials of this novel front-line transdiagnostic eating disorder treatment that could be more broadly implemented than more intensive individual therapies for eating disorders.

References

Ashburner, J. (2007). A fast diffeomorphic image registration algorithm. *NeuroImage, 38,* 95–113. http://dx.doi.org/10.1016/j.neuroimage.2007.07.007

Ashburner, J., & Friston, K. J. (2005). Unified segmentation. *NeuroImage, 26,* 839–851. http://dx.doi.org/10.1016/j.neuroimage.2005.02.018

Ballard, I. C., Murty, V. P., Carter, R. M., MacInnes, J. J., Huettel, S. A., & Adcock, R. A. (2011). Dorsolateral prefrontal cortex drives mesolimbic dopaminergic regions to initiate motivated behavior. *The Journal of Neuroscience, 31,* 10340–10346. http://dx.doi.org/10.1523/JNEUROSCI.0895-11.2011

Bardone-Cone, A. M., Harney, M. B., Maldonado, C. R., Lawson, M. A., Robinson, D. P., Smith, R., & Tosh, A. (2010). Defining recovery from an eating disorder: Conceptualization, validation, and examination of psychosocial functioning and psychiatric comorbidity. *Behaviour Research and Therapy, 48,* 194–202. http://dx.doi.org/10.1016/j.brat.2009.11.001

Becker, C. B., Wilson, C., Williams, A., Kelly, M., McDaniel, L., & Elmquist, J. (2010). Peer-facilitated cognitive dissonance versus healthy weight eating disorders prevention: A randomized comparison. *Body Image, 7,* 280–288. http://dx.doi.org/10.1016/j.bodyim.2010.06.004

Berscheid, E., Walster, E., & Bohrnstedt, G. (1973). The happy American body: A survey report. *Psychology Today, 7,* 119–131.

Bohon, C., Stice, E., & Spoor, S. (2009). Female emotional eaters show abnormalities in consummatory and anticipatory food reward: A functional magnetic resonance imaging study. *International Journal of Eating Disorders, 42,* 210–221. http://dx.doi.org/10.1002/eat.20615

Chen, S., Dong, D., Jackson, T., Zhuang, Q., & Chen, H. (2017). Trait-based food-cravings are encoded by regional homogeneity in the parahippocampal gyrus. *Appetite, 114,* 155–160. http://dx.doi.org/10.1016/j.appet.2017.03.033

Ciao, A. C., Latner, J. D., Brown, K. E., Ebneter, D. S., & Becker, C. B. (2015). Effectiveness of a peer-delivered dissonance-based program in reducing eating disorder risk factors in high school girls. *International Journal of Eating Disorders, 48,* 779–784. http://dx.doi.org/10.1002/eat.22418

Cowdrey, F. A., Park, R. J., Harmer, C. J., & McCabe, C. (2011). Increased neural processing of rewarding and aversive food stimuli in recovered anorexia nervosa. *Biological Psychiatry, 70,* 736–743. http://dx.doi.org/10.1016/j.biopsych.2011.05.028

Dakanalis, A., Clerici, M., Bartoli, F., Caslini, M., Crocamo, C., Riva, G., & Carrà, G. (2017). Risk and maintenance factors for young women's *DSM–5* eating disorders. *Archives of Women's Mental Health, 20,* 721–731. http://dx.doi.org/10.1007/s00737-017-0761-6

Engelmann, J. M., Versace, F., Robinson, J. D., Minnix, J. A., Lam, C. Y., Cui, Y., . . . Cinciripini, P. M. (2012). Neural substrates of smoking cue reactivity: A meta-analysis of fMRI studies. *NeuroImage, 60,* 252–262. http://dx.doi.org/10.1016/j.neuroimage.2011.12.024

Fairburn, C. G., Bailey-Straebler, S., Basden, S., Doll, H. A., Jones, R., Murphy, R., . . . Cooper, Z. (2015). A transdiagnostic comparison of enhanced cognitive behaviour therapy (CBT-E) and interpersonal psychotherapy in the treatment of eating disorders. *Behaviour Research and Therapy, 70,* 64–71. http://dx.doi.org/10.1016/j.brat.2015.04.010

Festinger, L. (1957). *A theory of cognitive dissonance*. Stanford, CA: Stanford University Press.

Geliebter, A., Ladell, T., Logan, M., Schneider, T., Sharafi, M., & Hirsch, J. (2006). Responsivity to food stimuli in obese and lean binge eaters using functional MRI. *Appetite, 46,* 31–35. http://dx.doi.org/10.1016/j.appet.2005.09.002

Gorgolewski, K. J., Storkey, A. J., Bastin, M. E., Whittle, I., & Pernet, C. (2013). Single subject fMRI test-retest reliability metrics and confounding factors. *NeuroImage, 69,* 231–243. http://dx.doi.org/10.1016/j.neuroimage.2012.10.085

Graham, J. W., Olchowski, A. E., & Gilreath, T. D. (2007). How many imputations are really needed? Some practical clarifications of multiple imputation theory. *Prevention Science, 8,* 206–213. http://dx.doi.org/10.1007/s11121-007-0070-9

Greenwald, A. G., McGhee, D. E., & Schwartz, J. L. (1998). Measuring individual differences in implicit cognition: The implicit association test. *Journal of Personality and Social Psychology, 74,* 1464–1480. http://dx.doi.org/10.1037/0022-3514.74.6.1464

Halliwell, E., & Diedrichs, P. C. (2014). Testing a dissonance body image intervention among young girls. *Health Psychology, 33,* 201–204. http://dx.doi.org/10.1037/a0032585

Hare, T. A., Camerer, C. F., & Rangel, A. (2009). Self-control in decision-making involves modulation of the vmPFC valuation system. *Science, 324,* 646–648. http://dx.doi.org/10.1126/science.1168450

Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in Cognitive Sciences, 4,* 223–233. http://dx.doi.org/10.1016/S1364-6613(00)01482-0

Hay, P. (2013). A systematic review of evidence for psychological treatments in eating disorders: 2005–2012. *International Journal of Eating Disorders, 46,* 462–469. http://dx.doi.org/10.1002/eat.22103

Jacobson, N. S., & Truax, P. (1991). Clinical significance: A statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting and Clinical Psychology, 59,* 12–19. http://dx.doi.org/10.1037/0022-006X.59.1.12

Kristeller, J., Wolever, R., & Sheets, V. (2013). Mindfulness-based eating awareness training (MB-EAT) for binge eating: A randomized clinical trial. *Mindfulness, 5,* 282–297. http://dx.doi.org/10.1007/s12671-012-0179-1

Kroenke, K., Spitzer, R. L., & Williams, J. B. (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine, 16,* 606–613. http://dx.doi.org/10.1046/j.1525-1497.2001.016009606.x

LaBar, K. S., & Cabeza, R. (2006). Cognitive neuroscience of emotional memory. *Nature Reviews Neuroscience, 7,* 54–64. http://dx.doi.org/10.1038/nrn1825

Laird, A. R., Fox, P. M., Eickhoff, S. B., Turner, J. A., Ray, K. L., McKay, D. R., . . . Fox, P. T. (2011). Behavioral interpretations of intrinsic connectivity networks. *Journal of Cognitive Neuroscience, 23,* 4022–4037. http://dx.doi.org/10.1162/jocn_a_00077

Leech, R., & Sharp, D. J. (2014). The role of the posterior cingulate cortex in cognition and disease. *Brain: A Journal of Neurology, 137,* 12–32. http://dx.doi.org/10.1093/brain/awt162

Leidy, H. J., Lepping, R. J., Savage, C. R., & Harris, C. T. (2011). Neural responses to visual food stimuli after a normal vs. higher protein breakfast in breakfast-skipping teens: A pilot fMRI study. *Obesity, 19,* 2019–2025. http://dx.doi.org/10.1038/oby.2011.108

Levinson, C. A., Zerwas, S., Calebs, B., Forbush, K., Kordy, H., Watson, H., . . . Bulik, C. M. (2017). The core symptoms of bulimia nervosa, anxiety, and depression: A network analysis. *Journal of Abnormal Psychology, 126,* 340–354. http://dx.doi.org/10.1037/abn0000254

Lilienfeld, S. O., Ritschel, L. A., Lynn, S. J., Brown, A. P., Cautin, R. L., & Latzman, R. D. (2013). The research-practice gap: Bridging the schism between eating disorder researchers and practitioners. *International Journal of Eating Disorders, 46,* 386–394. http://dx.doi.org/10.1002/eat.22090

MacDonald, A. W., III, Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science, 288,* 1835–1838. http://dx.doi.org/10.1126/science.288.5472.1835

MacDonald, D. E., McFarlane, T. L., Dionne, M. M., David, L., & Olmsted, M. P. (2017). Rapid response to intensive treatment for bulimia nervosa and purging disorder: A randomized controlled trial of a CBT intervention to facilitate early behavior change. *Journal of Consulting and Clinical Psychology, 85,* 896–908. http://dx.doi.org/10.1037/ccp0000221

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience, 24,* 167–202. http://dx.doi.org/10.1146/annurev.neuro.24.1.167

Müller, S., & Stice, E. (2013). Moderators of the intervention effects for a dissonance-based eating disorder prevention program: Results from an amalgam of three randomized trials. *Behaviour Research and Therapy, 51,* 128–133. http://dx.doi.org/10.1016/j.brat.2012.12.001

Pelchat, M. L., Johnson, A., Chan, R., Valdez, J., & Ragland, J. D. (2004). Images of desire: Food-craving activation during fMRI. *NeuroImage, 23,* 1486–1493. http://dx.doi.org/10.1016/j.neuroimage.2004.08.023

Saunders, J. B., Aasland, O. G., Babor, T. F., de la Fuente, J. R., & Grant, M. (1993). Development of the alcohol use disorders identification test (AUDIT): WHO collaborative project on early detection of persons with harmful alcohol consumption II. *Addiction, 88,* 791–804. http://dx.doi.org/10.1111/j.1360-0443.1993.tb02093.x

Schienle, A., Schäfer, A., Hermann, A., & Vaitl, D. (2009). Binge-eating disorder: Reward sensitivity and brain activation to images of food. *Biological Psychiatry, 65,* 654–661. http://dx.doi.org/10.1016/j.biopsych.2008.09.028

Shrout, P. E., & Fleiss, J. L. (1979). Intraclass correlations: Uses in assessing rater reliability. *Psychological Bulletin, 86,* 420–428. http://dx.doi.org/10.1037/0033-2909.86.2.420

Simon, J. J., Skunde, M., Walther, S., Bendszus, M., Herzog, W., & Friederich, H. C. (2016). Neural signature of food reward processing in bulimic-type eating disorders. *Social Cognitive and Affective Neuroscience, 11,* 1393–1401. http://dx.doi.org/10.1093/scan/nsw049

Skinner, H. A. (1982). The drug abuse screening test. *Addictive Behaviors, 7,* 363–371. http://dx.doi.org/10.1016/0306-4603(82)90005-3

Smith, D. V., Hayden, B. Y., Truong, T. K., Song, A. W., Platt, M. L., & Huettel, S. A. (2010). Distinct value signals in anterior and posterior ventromedial prefrontal cortex. *The Journal of Neuroscience, 30,* 2490–2495. http://dx.doi.org/10.1523/JNEUROSCI.3319-09.2010

Smith, S. M. (2002). Fast robust automated brain extraction. *Human Brain Mapping, 17,* 143–155. http://dx.doi.org/10.1002/hbm.10062

Stice, E., Marti, C. N., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed *DSM–5* eating disorder diagnoses in an 8-year prospective community study of young women. *Journal of Abnormal Psychology, 122,* 445–457. http://dx.doi.org/10.1037/a0030679

Stice, E., Marti, C. N., Spoor, S., Presnell, K., & Shaw, H. (2008). Dissonance and healthy weight eating disorder prevention programs: Long-term effects from a randomized efficacy trial. *Journal of Consulting and Clinical Psychology, 76,* 329–340. http://dx.doi.org/10.1037/0022-006X.76.2.329

Stice, E., Rohde, P., Butryn, M., Menke, K. S., & Marti, C. N. (2015). Randomized controlled pilot trial of a novel dissonance-based group treatment for eating disorders. *Behaviour Research and Therapy, 65,* 67–75. http://dx.doi.org/10.1016/j.brat.2014.12.012

Stice, E., Rohde, P., Shaw, H., & Gau, J. M. (2017). Clinician-led, peer-led, and internet-delivered dissonance-based eating disorder prevention programs: Acute effectiveness of these delivery modalities. *Journal of Consulting and Clinical Psychology, 85,* 883–895. http://dx.doi.org/10.1037/ccp0000211

Stice, E., Rohde, P., Shaw, H., & Gau, J. M. (2019). Randomized trial of a dissonance-based group treatment for eating disorders versus a supportive mindfulness group treatment. *Journal of Consulting and Clinical Psychology, 87,* 79–90. http://dx.doi.org/10.1037/ccp0000365

Stice, E., Telch, C. F., & Rizvi, S. L. (2000). Development and validation of the Eating Disorder Diagnostic Scale: A brief self-report measure of anorexia, bulimia, and binge-eating disorder. *Psychological Assessment, 12,* 123–131. http://dx.doi.org/10.1037/1040-3590.12.2.123

Stice, E., & Van Ryzin, M. (2019). A prospective test of the temporal sequencing of risk factor emergence in the dual pathway model of eating disorders. *Journal of Abnormal Psychology, 128,* 119–128.

Stice, E., Yokum, S., Veling, H., Kemps, E., & Lawrence, N. S. (2017). Pilot test of a novel food response and attention training treatment for obesity: Brain imaging data suggest actions shape valuation. *Behaviour Research and Therapy, 94,* 60–70. http://dx.doi.org/10.1016/j.brat.2017.04.007

Stice, E., Yokum, S., & Waters, A. (2015). Dissonance-based eating disorder prevention program reduces reward region response to thin models: How actions shape valuation. *PLoS ONE, 10*(12), e0144530. http://dx.doi.org/10.1371/journal.pone.0144530

Stoodley, C. J., Valera, E. M., & Schmahmann, J. D. (2012). Functional topography of the cerebellum for motor and cognitive tasks: An fMRI study. *NeuroImage, 59,* 1560–1570. http://dx.doi.org/10.1016/j.neuroimage.2011.08.065

Swanson, S. A., Crow, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlates of eating disorders in adolescents. Results from the national comorbidity survey replication adolescent supplement. *Archives of General Psychiatry, 68,* 714–723. http://dx.doi.org/10.1001/archgenpsychiatry.2011.22

Tang, D. W., Fellows, L. K., Small, D. M., & Dagher, A. (2012). Food and drug cues activate similar brain regions: A meta-analysis of functional MRI studies. *Physiology & Behavior, 106,* 317–324. http://dx.doi.org/10.1016/j.physbeh.2012.03.009

Tataranni, P. A., Gautier, J. F., Chen, K., Uecker, A., Bandy, D., Salbe, A. D., . . . Ravussin, E. (1999). Neuroanatomical correlates of hunger and satiation in humans using positron emission tomography. *Proceedings of the National Academy of Sciences, 96,* 4569–4574. http://dx.doi.org/10.1073/pnas.96.8.4569

Uher, R., Murphy, T., Brammer, M. J., Dalgleish, T., Phillips, M. L., Ng, V. W., . . . Treasure, J. (2004). Medial prefrontal cortex activity associated with symptom provocation in eating disorders. *The American Journal of Psychiatry, 161,* 1238–1246. http://dx.doi.org/10.1176/appi.ajp.161.7.1238

Uher, R., Murphy, T., Friederich, H.-C., Dalgleish, T., Brammer, M. J., Giampietro, V., . . . Treasure, J. (2005). Functional neuroanatomy of body shape perception in healthy and eating-disordered women. *Biological Psychiatry, 58,* 990–997. http://dx.doi.org/10.1016/j.biopsych.2005.06.001

Vocks, S., Herpertz, S., Rosenberger, C., Senf, W., & Gizewski, E. R. (2011). Effects of gustatory stimulation on brain activity during hunger and satiety in females with restricting-type anorexia nervosa: An fMRI study. *Journal of Psychiatric Research, 45,* 395–403. http://dx.doi.org/10.1016/j.jpsychires.2010.07.012

Watson, D., & Clark, L. (1992). Affects separable and inseparable: On the hierarchical arrangement of the negative affects. *Journal of Personality and Social Psychology, 62,* 489–505. http://dx.doi.org/10.1037/0022-3514.62.3.489

Wilson, G. T., & Zandberg, L. J. (2012). Cognitive-behavioral guided self-help for eating disorders: Effectiveness and scalability. *Clinical Psychology Review, 32,* 343–357. http://dx.doi.org/10.1016/j.cpr.2012.03.001

Winston, J. S., O'Doherty, J., Kilner, J. M., Perrett, D. I., & Dolan, R. J. (2007). Brain systems for assessing facial attractiveness. *Neuropsychologia, 45,* 195–206. http://dx.doi.org/10.1016/j.neuropsychologia.2006.05.009

Wonderlich, S. A., Peterson, C. B., Crosby, R. D., Smith, T. L., Klein, M. H., Mitchell, J. E., & Crow, S. J. (2014). A randomized controlled comparison of integrative cognitive-affective therapy (ICAT) and enhanced cognitive-behavioral therapy (CBT-E) for bulimia nervosa. *Psychological Medicine, 44,* 543–553. http://dx.doi.org/10.1017/S0033291713001098

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