



# Effects of internet-delivered eating disorder prevention on reward-based eating drive: A randomized controlled trial

Taona P. Haderlein<sup>a,\*</sup>, A. Janet Tomiyama<sup>b</sup>

<sup>a</sup> VA Greater Los Angeles Healthcare System, Center for the Study of Healthcare Innovation, Implementation, and Policy, United States of America

<sup>b</sup> University of California, Los Angeles, United States of America

## ARTICLE INFO

### Keywords:

Internet  
Cognitive behavioral treatment  
Dissonance-based intervention  
Reward-based eating  
Eating disorder prevention  
Eating disorders

## ABSTRACT

**Objective:** This study is a secondary analysis testing the effects of an internet eating disorder prevention program on reward-based eating drive in a high-risk sample of college-aged women.

**Method:** We analyzed data from 278 women who were randomized to internet dissonance-based intervention (DBI-I), internet cognitive-behavioral treatment (CBT-I), or no intervention (NI). Both active conditions consisted of self-guided activities completed over the course of four weeks. Linear mixed effects modeling was used to test the effect of internet intervention on reward-based eating drive.

**Results:** DBI-I was associated with greater reductions in reward-based eating over time than NI. No other Condition  $\times$  Time effects were found.

**Discussion:** The results provide preliminary support for DBI-I as a strategy for reducing reward-based eating drive in a high-risk population relative to no intervention.

## 1. Introduction

Reward-based eating drive describes the tendency to engage in food consumption for emotional gratification and craving abatement, rather than in response to satiety cues (Epel et al., 2014). It is characterized by a lack of control over eating, lack of satiation, and preoccupation with food (Epel et al., 2014). Reward-based eating tends to occur in response to highly palatable foods (i.e., high in fat, sugar, and salt) and is theorized to relate to the neural reward system (Epel et al., 2012), rather than physiological hunger. Neural substrates research has linked reward-based eating with dopaminergic pathways that modulate reward sensitivity (Epel et al., 2012)—the same neuronal systems implicated in the context of palatable food consumption and binge eating (Giuliani et al., 2014).

Although reward-based eating drive is associated with binge eating (Pinaquy et al., 2003), the constructs of reward-based eating drive and binge eating differ in several respects. Reward-based eating drive describes a non-pathological response to palatable food (Epel et al., 2014). Reward-based eating can occur across a range of food serving sizes and is not time-limited (Epel et al., 2014). Binge eating, on the other hand, is defined by eating a larger than expected amount of food in a discrete period of time (American Psychiatric Association, 2013). In addition,

binge eating tends to occur in discrete episodes (American Psychiatric Association, 2013), whereas reward-based eating drive describes a pervasive pattern of eating behavior (Epel et al., 2014). People who binge eat may or may not have high reward-based eating drive; nonetheless, previous research shows that binge eating behavior correlates with reward-based eating drive (Epel et al., 2014).

To our knowledge, only one intervention has targeted reward-based eating drive. The Supporting Health by Integrating Nutrition and Exercise (SHINE) trial applied mindfulness techniques to promote weight loss for adults with obesity (Mason et al., 2016). The SHINE intervention aimed to increase eating awareness, stress management, emotion regulation, and exercise. The intervention significantly reduced reward-based eating drive, and that in turn predicted weight loss at 12-month follow-up. This study population comprised participants with overweight or obesity (Daubenmier et al., 2011; Mason et al., 2016), but importantly, these behaviors are also prevalent among people at risk for eating disorders (Stice et al., 2002). However, no known work has attempted to reduce reward-based eating drive in the context of eating disorder (ED) treatment or prevention.

ED prevention programs aim to reduce ED risk factors among people with subthreshold ED symptoms (Stice et al., 2013). In general, health

\* Corresponding author at: Center for the Study of Healthcare Innovation, Implementation, and Policy (CSHIP), VA Greater Los Angeles Healthcare System, 11301 Wilshire Blvd, Los Angeles, CA 90073, United States of America.

E-mail address: [taona.haderlein@va.gov](mailto:taona.haderlein@va.gov) (T.P. Haderlein).

<https://doi.org/10.1016/j.eatbeh.2021.101572>

Received 3 June 2019; Received in revised form 17 September 2021; Accepted 27 September 2021

Available online 2 October 2021

1471-0153/© 2021 Published by Elsevier Ltd.

prevention programs aim to either a) prevent the development of symptoms among healthy individuals (universal prevention); b) reduce existing symptoms among individuals at risk (selective prevention); or c) ameliorate symptoms among individuals presenting with symptoms of the targeted disease or behavior (indicated prevention; [Gordon, 1983](#)). The interventions applied in the current study were selective, as they targeted individuals at risk for EDs. Meta-analytic studies report that selective ED prevention programs yield superior post-intervention effects when compared to universal prevention for the reduction of negative affect, eating pathology, and dieting ([Fingeret et al., 2006](#); [Stice & Shaw, 2004](#)).

ED prevention programs adopting dissonance-based intervention (DBI) and cognitive-behavioral treatment (CBT) techniques are the most empirically supported ([Beintner et al., 2012](#); [Stice et al., 2013](#)). DBI encourages participants to challenge the media-propagated thin-ideal ([Stice et al., 2000](#)). It is theorized that participants experience psychological discomfort from the discrepancy between their pre-existing pro-thin-ideal attitudes and the public, anti-thin-ideal behavior exhibited during the intervention. To reconcile this dissonance, participants shift away from the thin-ideal, reducing body image distress and maladaptive eating behaviors ([Stice et al., 2007](#)). CBT focuses on the association between body-related cognitions, negative mood, and maladaptive eating behaviors ([Williamson et al., 2004](#)). CBT interventions aim to restructure distorted thoughts pertaining to body weight and shape, and disrupt associations among distorted thoughts, negative emotions, and maladaptive eating behaviors ([Wilson et al., 2002](#)).

Regarding specific treatment protocols, The Body Project, a dissonance-based ED prevention program, and the Healthy Weight prevention program, which promotes long-term adaptive exercise and eating habits, are the only programs to reduce ED symptoms and onset in trials across multiple independent teams ([Stice et al., 2019](#)). Evidence also exists supporting CBT for ED prevention ([Watson et al., 2016](#)). For example, Student Bodies, a computer-based CBT intervention, has been shown to reduce ED risk factors, such as body dissatisfaction, in multiple studies ([Beintner et al., 2012](#)).

Only one known study has compared DBI and CBT for ED prevention in a randomized controlled trial ([Chithambo & Huey, 2017](#)). The study tested the efficacy of internet CBT (CBT-I), internet DBI (DBI-I), and no intervention control (NI) for the reduction of ED risk factors, including body dissatisfaction, thin-ideal internalization, global eating pathology, dieting, and depression symptoms. Results suggested that CBT-I was more effective than NI at reducing dieting, eating pathology, and depression symptoms. No effects were found for body dissatisfaction or thin-ideal internalization.

Reward-based eating drive is associated with high emotional distress and is correlated with scores on the Binge Eating Scale ([Epel et al., 2014](#); [Gormally et al., 1982](#)). Yet, no known studies examine the efficacy of ED prevention for the reduction of reward-based eating drive. Utilizing internet technology for addressing reward-based eating is a novel approach that facilitates convenience and low-cost dissemination. The current study is a secondary analysis of data obtained in [Chithambo and Huey's \(2017\)](#) evaluation of DBI-I and CBT-I for internet eating disorder prevention. The intervention was delivered to a sample of college-age women at high-risk for EDs, as indicated by elevated Weight Concerns Scale scores ([Killen et al., 1996](#)). This study expands on the previous trial to evaluate a secondary outcome, reward-based eating drive, and includes data from a second intervention site.

Emerging neuroscience has supported the role of reward in eating behaviors ([Adam & Epel, 2007](#)). However, previous ED prevention studies focus on aversive aspects of disordered eating symptoms, such as food restriction or eating until uncomfortably full ([Le et al., 2017](#)). It is important to examine whether behavioral interventions can address reward-based eating drive, which involves appetitive aspects of eating behavior. CBT has the potential to influence reward-based eating drive by targeting thoughts and behaviors pertaining to the reward value of food. Conversely, DBI-I has the potential to affect reward-based eating

by addressing societally-influenced body preoccupation, a correlate of maladaptive eating behavior ([Rosen, 2013](#)).

[Kazdin \(2007\)](#) recommended that experimental investigations adopt a design that allows the researcher to identify specific mechanisms that account for predicted outcomes. In the context of treatment evaluation research, such a design would compare the efficacy of therapies that differ only with regard to the elements specific to their theoretical perspectives. Other features, such as the structure and duration of treatment, the appearance of therapy materials distributed to patients, and the nature of participant-provider interactions would be held constant across conditions whenever possible. In the current study, DBI-I and CBT-I were designed to be parallel in duration, appearance, and structure to increase the likelihood that differences in outcome could be attributed to theory-driven mechanisms specific to each treatment.

Because reward-based eating drive involves distorted cognitions around food and satiety ([Epel et al., 2014](#)), we hypothesized that CBT-I would result in a greater reduction in reward-based eating drive than DBI-I, which evokes a sociocultural perspective not explicitly related to eating behavior. We hypothesized that both CBT-I and DBI-I would be more effective at reducing reward-based eating drive than no intervention (NI).

## 2. Methods

Participants were students recruited from two large universities in southern California. The mean age for the sample was 20.50 ( $SD = 2.46$ ). The mean BMI for the sample was 22.23 ( $SD = 3.49$ ). Seven percent of participants were underweight, 77% normal weight, 12% overweight, and 3% obese. Forty-two percent of participants were Asian, 30% White, 15% Latinx, 8% Multi-ethnic, 5% Black, and 1% Other. Only females were included, because intervention content addressed body standards specific to women.

As research indicates that ED prevention is most effective for high-risk samples, participants were eligible if they scored  $\geq 34$  on the Weight Concerns Scale ([Killen et al., 1996](#)). This scale has shown predictive validity for ED onset ([Killen et al., 1996](#)). The cutoff represents the mean score from a community sample of women, and has been applied as a screening measure in previous ED prevention programs (e.g., [Manwaring et al., 2008](#)). Because the interventions were designed for subthreshold pathology, women who scored  $\geq 20$  on the Eating Attitudes Test ([Garner et al., 1983](#)) were excluded. Excluded individuals were provided a list of local mental health resources. Participants endorsing a "2" or "3" on the Beck Depression Inventory's suicidal thoughts item were disqualified (range = 0–3; [Beck et al., 1996](#)). In addition to providing referral information for local treatment providers, the first author called participants who expressed suicidal ideation ( $n = 1$ ) to assess risk and assure safety.

### 2.1. Design

Participants ( $n = 367$ ) from two universities were randomized to a study condition (University A  $n = 276$ ; University B  $n = 91$ ). Eighty-nine participants from University A were randomized prior to the addition of the RED scale, which was rolled out at both sites as a secondary measure upon the addition of University B. Therefore, in the current study, we analyzed RED scale data for the 278 participants (187 from University A, 91 from University B) who were randomized to a study condition after the rollout of the RED scale.

All participants completed measures assessing the primary study outcomes, which included body dissatisfaction, thin-ideal internalization, global eating pathology, dieting, and depression symptoms. Intervention effects for these outcome measures are described above and were reported in [Chithambo and Huey \(2017\)](#).

Measures were collected at pre- and post-intervention via the online survey platform Qualtrics. An automated randomization feature assigned participants to DBI-I, CBT-I, or NI immediately after the pre-treatment

assessment. The post-treatment assessment took place four weeks later. See Fig. 1 for a CONSORT diagram of participant flow through the study.

2.2. Procedures

Study procedures were approved by both universities' respective IRBs.

2.2.1. Screening and recruitment

Participants were recruited from the psychology subject pools of two large universities. Interested parties were emailed a link to a screening questionnaire assessing ED symptoms and suicidal ideation. Participants received a study information sheet for consent, and the pre-treatment assessment questionnaire, via email. Participants were randomized to a treatment condition, and participants randomized to CBT-I or DBI-I were directed to Session 1 immediately after completing the assessment questionnaire. Participants completed both pre-treatment and post-treatment measures independently; accordingly, participants and researchers were blinded to condition allocation. However, the first author was aware of participant allocation after randomization, as she collected homework assignments via email.

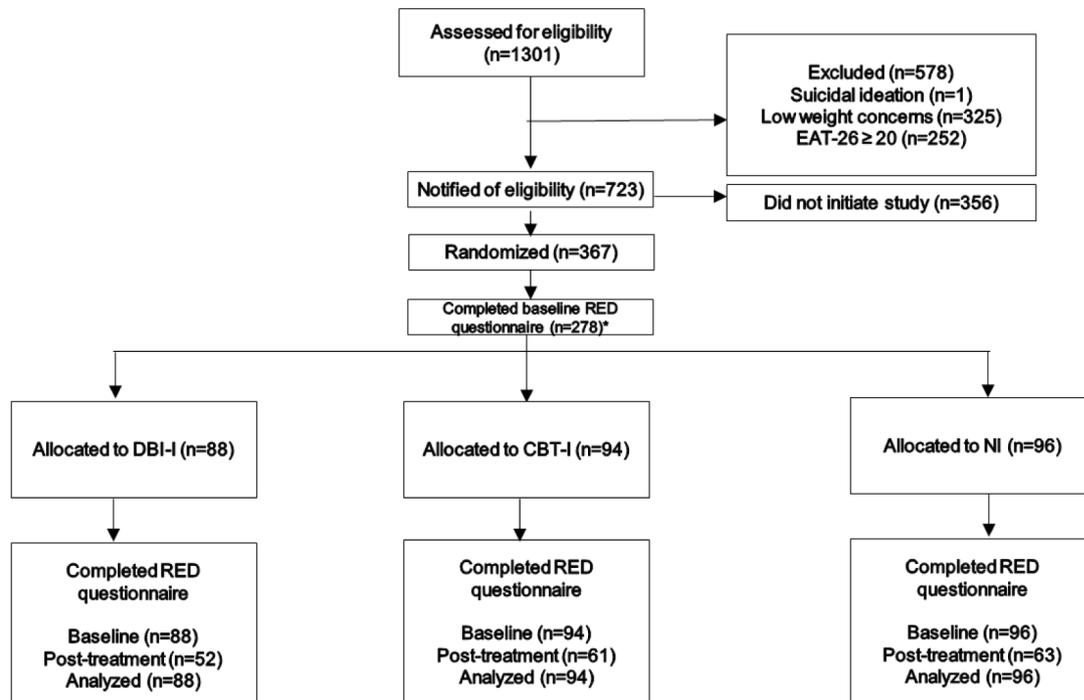
Subsequently, three additional sessions were emailed on a weekly basis, for a total of four intervention sessions. The emails were automatically sent once per week to the participants' registered email address via the Qualtrics software platform. Each email contained a hyperlink that connected participants to the present week's session. Participants completed one homework assignment each week, which they received in an automated email after completing the week's treatment session.

Compensation differed between the two universities due to diverging IRB requirements. Participants at University A were rewarded five research credits at the final assessment regardless of study condition. University B required that participants receive compensation proportionate to their time spent; therefore, NI participants received one credit, and DBI-I and CBT-I participants received five credits. At both sites, participants had the option to participate in alternative, non-research activities for course credit.

2.3. Intervention conditions

2.3.1. DBI-I

The DBI-I program comprised four sessions that took place over the place of 28 days at a frequency of one session per week. A between session assignment was assigned each week and was due one week after the preceding session. Content was informed by the facilitator manual for the *Body Project*, an established DBI-I protocol for ED prevention (Stice et al., 2007). Prior to the launch of the study, two of the developers of the Body Project reviewed content from the DBI-I program and provided feedback (Stice & Rohde, personal communication, November-December 2012). In response to their comments, a "hint" feature was added to sessions eliciting verbal feedback to prevent participants from becoming stuck on a prompt. During the first session, participants responded to prompts that provided education about the media-propagated thin-ideal, and generated arguments against pro thin-ideal statements. For homework, participants wrote a letter to an adolescent girl describing the costs of the thin-ideal. For session 2, participants wrote verbal challenges to statements that conveyed a drive



Note. DBI-I = Internet dissonance-based intervention; CBT-I=Internet cognitive behavioral treatment; NI=No intervention. \*89 individuals entered the study before the RED scale was added as an outcome measure

2.2 Procedures

Fig. 1. Participant flow chart

Note. DBI-I = Internet dissonance-based intervention; CBT-I=Internet cognitive behavioral treatment; NI=No intervention. \*89 individuals entered the study before the RED scale was added as an outcome measure.

towards thinness (e.g., “I am too chubby to eat dessert after dinner today”). For homework, participants provided examples of thin-ideal statements from their own lives, and provided counter-arguments. In session 3, participants wrote examples of comebacks to verbal statements that endorse the thin-ideal. For homework, participants were assigned to provide a list of 10 ways to publicly challenge the thin-ideal. During session 4, participants read information on how to identify thin-ideal talk in their own lives. They also generated verbal challenges to statements indicative of thin-ideal talk. For homework, participants were assigned to write a second letter to an adolescent girl, taking into account the information they had learned throughout the program.

### 2.3.2. CBT-I

CBT-I also consisted of four weekly internet-delivered sessions that occurred over a span of 28 days. CBT-I content was derived from sections of *The Body Image Workbook*, a self-help manual for body dissatisfaction (Cash, 1997). The program was selected because it is amenable to a self-directed format and contained exercises that could be readily adapted in parallel to DBI-I. Although this was the first web-based adaptation of the program, the program has demonstrated efficacy in reducing body dysphoria and increasing appearance satisfaction (Nye & Cash, 2006; Strachan & Cash, 2002). The sessions were designed to be parallel in structure and appearance to DBI-I, but were based on an alternative theoretical model positing that thoughts, emotions, and behaviors contribute to negative body evaluation and maladaptive eating behaviors. As such, participants in this condition challenged common appearance assumptions, and restructured thoughts associated with poor body image. In parallel to DBI-I, a hint feature allowed participants to view example responses.

During the first session, participants wrote answers to questions that assessed their knowledge of the symptoms and prevalence of poor body image. The session also included content on the association between critical body image thoughts and low mood. For homework, they were assigned to write a one-page summary of what they learned during the session. In session 2, participants generated alternative interpretations of cognitive distortions that sustain poor body image. For homework, they were assigned to generate examples of “appearance assumptions” relevant to their own lives and identify alternative interpretations. During session 3, participants generated rational responses to negative body cognitions (E.g., “I am less attractive than everyone else”). As homework, participants were assigned to come up with 10 potential benefits of changing their body image thoughts. For session 4, participants wrote responses to hypothetical insensitive body remarks made by others. The participants were asked to write a one-page letter summarizing what they had learned during the course of the intervention as a final homework assignment.

In both active intervention conditions, participants received an automated email reminder to engage in the session every week. They also received emails to remind them to email their homework to the first author. The first author was available to assist with technical and administrative issues (e.g., collecting homework, logging into sessions, participant compensation). Other than this, no guidance was provided by the authors or other study personnel.

### 2.3.3. NI

Participants in NI completed pre-treatment and post-treatment questionnaires. NI participants were offered the opportunity to participate in an active condition after the completion of the post-treatment assessment.

## 2.4. Measures

### 2.4.1. Demographics

A demographics questionnaire assessed age, self-reported height and weight, and self-identified racial/ethnic background.

### 2.4.2. Reward-based eating drive

The Reward-Based Eating Drive (RED) Scale was used to measure reward-based eating drive (Epel et al., 2014). The RED scale assesses lack of control over eating, lack of satiation, and preoccupation with food. The self-report questionnaire consists of nine items including “I feel out of control in the presence of delicious food” and “Food is always on my mind” using a five-point scale (0 = “very false” and 4 = “very true”). In a validation study of the RED scale, Epel et al. (2014) found correlations in the medium range for binge eating and ED symptoms, supporting reward-based eating as a unique construct. The RED scale has demonstrated convergent and discriminant validity and invariance across gender, age, and race/ethnicity (Epel et al., 2014). We used coefficient omega to evaluate internal consistency, as Cronbach's alpha assumes the existence of one latent construct and is therefore not well suited to measure reliability in multi-dimensional scales such as the RED (McNeish, 2018; Starkweather, 2012). Omega was 0.70, a value in the “acceptable” range.

We followed guidelines provided by Jacobson and Truax (1991) to calculate the Reliable Change Index (RC) from pre-treatment to post-treatment. The index provides a metric for examining whether observed changes in outcomes are clinically significant. RC was derived using the conservative approach to calculating the standard error of measurement of the difference recommended by Maassen (2004).

## 2.5. Analyses

Linear mixed-effects regression with full-information maximum likelihood estimation was conducted using R (R Core Team & R. C., 2013) with time (pre- or post-treatment) at level 1 and participant at level 2. Post-treatment missingness was included as a covariate for all analyses – a flexible technique to control for unknown predictors of missingness (Hedeker & Gibbons, 1997). The variable was non-significant and consequently was dropped from study analyses. The coefficient for the treatment main effect reflects differences between conditions at baseline (Twisk et al., 2018). Therefore, the Time x Condition term was evaluated to test intervention effects, as this term allows for the evaluation of between-group differences in change from pre- to post-intervention. We included race/ethnicity as a fixed covariate because Chithambo and Huey (2017) found that race moderated the effect of DBI-I on eating pathology. Race/ethnicity included the following categories: Asian, Latino, Black, White, Multi-racial, Other. We designated “Asian” as the reference group in the regression analysis based on majority representation in the sample. Age and BMI were also included as fixed effects covariates, as both variables have been associated with ED prevention program outcomes in previous literature (Müller & Stice, 2013; Stice et al., 2007). In addition, we included study site (University A = 0, University B = 1) as a covariate to control for potential differences between sites.

## 3. Results

### 3.1. Baseline characteristics

One-way ANOVA analyses found no group differences in baseline demographic characteristics, BMI, or reward-based eating ( $p$  values = .13–.38). Independent samples  $t$ -test analyses found that University A had higher baseline RED scores ( $M = 2.01$ ,  $SD = 0.06$ ) than University B ( $M = 1.81$ ,  $SD = 0.08$ ;  $t(276) = 2.13$ ,  $p = .03$ ). University A students reported higher age ( $M = 20.73$ ,  $SD = 0.20$ ) than University B students ( $M = 20.03$ ,  $SD = 0.08$ ;  $t(276) = 2.24$ ,  $p = .03$ ). University A students also reported lower body weight ( $M$  BMI = 21.83,  $SD = 0.26$ ) than University B students ( $M$  BMI = 23.04,  $SD = 0.35$ ;  $t(276) = 2.75$ ,  $p = .01$ ). The sites did not differ in race or ethnicity distribution. As described above, study site was included as a covariate in linear mixed effects analyses (Table 1).

**Table 1**  
Demographic characteristics and outcome variable.

	Baseline				Post-treatment			
	Total (n = 278)	DBI-I (n = 88)	CBT-I (n = 94)	NI (n = 96)	Total (n = 176)	DBI-I (n = 52)	CBT-I (n = 61)	NI (n = 63)
Age (years), M(SD)	20.50 (2.46)	20.69 (3.69)	20.51 (1.66)	20.33 (1.53)	–	–	–	–
Body mass index (BMI), M(SD)	22.23 (3.49)	22.65 (3.59)	22.20 (3.09)	21.87 (3.73)	22.04 (3.77)	22.89 (4.23)	22.27 (3.40)	21.10 (3.55)
BMI category								
Underweight, n (%)	20 (7.19)	8 (9.09)	4 (4.26)	8 (8.33)	12 (6.82)	4 (7.69)	4 (5.63)	8 (10.96)
Normal weight, n (%)	214 (76.98)	61 (69.32)	75 (79.79)	78 (81.25)	138 (78.41)	37 (71.15)	54 (76.06)	56 (76.71)
Overweight, n (%)	34 (12.23)	15 (17.05)	11 (11.70)	8 (8.33)	21 (11.93)	9 (42.86)	10 (14.08)	8 (0.10.96)
Obese Class 1, n (%)	6 (2.16)	4 (4.55)	2 (2.13)	0 (0)	3 (1.70)	1 (1.92)	2 (2.82)	0 (0)
Obese Class 2, n (%)	2 (0.72)	0 (0)	1 (1.06)	1 (1.04)	1 (0.57)	0 (0)	1 (1.41)	0 (0)
Obese Class 3, n (%)	1 (0.36)	0 (0)	0 (0)	1 (1.04)	1 (0.57)	1 (1.92)	0 (0)	1 (1.36)
Missing, n (%)	1 (0.36)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Ethnicity								
Asian, n (%)	112 (40.29)	36 (40.91)	36 (38.29)	40 (41.67)	–	–	–	–
White, n (%)	84 (30.22)	22 (25.00)	30 (31.91)	32 (33.33)	–	–	–	–
Latino, n (%)	42 (15.11)	14 (15.91)	17 (18.09)	11 (11.45)	–	–	–	–
Black, n (%)	14 (5.04)	7 (7.95)	5 (5.32)	2 (2.08)	–	–	–	–
Multi-ethnic, n (%)	22 (7.91)	6 (6.81)	6 (6.38)	10 (10.42)	–	–	–	–
Other, n (%)	3 (1.08)	3 (3.41)	0 (0.00)	0 (0.00)	–	–	–	–
RED Score, M(SD)	1.95 (0.78)	2.07 (0.72)	1.82 (0.79)	1.97 (0.79)	1.88 (0.79)	1.82 (0.75)	1.74 (0.81)	2.06 (0.76)

3.2. Attrition

Sixty-three percent of participants (n = 182) completed the post-intervention assessment. Dropout rates for DBI-I, CBT-I, and NI were 40% (n = 36), 35% (n = 33), and 34% (n = 33), respectively and did not vary between conditions ( $\chi^2(2) = 0.99, p = .61$ ). Overall, 37% of participants did not complete the post-treatment assessment and thus are missing data for that time point. Baseline RED scores were not associated with likelihood of dropout (OR = 1.16, 95% CI: 0.85, 1.59, p = .35).

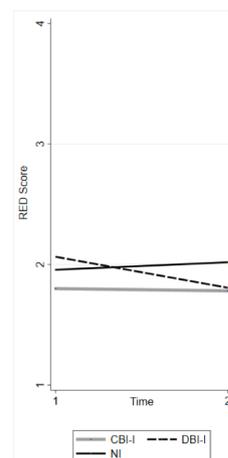
3.3. Main outcomes

The main study analyses are presented in Table 2 and Fig. 2. Significant main effects for the CBT-I vs. DBI-I and Site regression

**Table 2**  
Linear mixed effects regression results.

Random effects	Variance	SE	95% CI		
Participant (Intercept)	0.14	0.04	0.07, 0.25		
Fixed effects					
Predictor	$\beta$	SE	95% CI	z	p
Intercept	1.57	0.42	0.74, 2.39	3.72	<0.01*
Time	-0.02	0.11	-0.24, 0.19	-0.20	0.84
Age	0.02	0.02	-0.009, 0.05	1.34	0.18
Race (ref: Asian)					
Latino	-0.26	0.12	-0.50, -0.02	-2.15	0.03
Black	-0.10	0.19	-0.49, 0.28	-0.53	0.59
White	0.08	0.09	-0.11, 0.26	0.82	0.41
Multi-Racial	0.00	0.15	-0.29, 0.29	0.02	0.99
Other	-0.23	0.34	-0.88, 0.43	-0.68	0.49
BMI	-0.00	0.01	-0.03, 0.01	-0.90	0.37
Site	-0.26	0.08	-0.42, -0.09	-3.09	<0.01*
CBT-I vs. DBI-I	0.50	0.23	0.03, 0.97	2.11	0.04*
DBI-I vs. NI	0.42	0.23	-0.04, 0.89	1.80	0.07
CBT-I vs. NI	0.08	0.23	-0.53, 0.38	0.33	0.74
Time X DBI-I/CBT-I	-0.23	0.16	-0.54, 0.09	-1.42	0.15
Time X DBI-I/NI	-0.32	0.16	-0.62, -0.01	-2.00	0.05*
Time X CBT-I/NI	-0.08	0.15	-0.39, 0.22	-0.53	0.59

Note. \*p < .05. CBT-I=Internet cognitive-behavioral treatment. DBI-I=Internet dissonance-based intervention. NI = no intervention. Site was coded 0 = University A, 1 = University B. The DBI-I vs. CBT-I contrast variable was dummy coded 0 = CBT-I, 1 = DBI-I. The CBT-I vs. NI condition contrast variable was coded 0 = NI, 1 = CBT-I. The DBI-I vs. NI contrast variable was dummy coded 0 = NI, 1 = DBI-I.



Note. \*p<.05. CBT-I=Internet cognitive-behavioral treatment. DBI-I=Internet dissonance-based intervention.

**Fig. 2.** Effects of CBT-I and DBI-I on reward-based eating

Note. \*p < .05. CBT-I=Internet cognitive-behavioral treatment. DBI-I=Internet dissonance-based intervention.

coefficients indicated pre-treatment differences in RED scores between sites, and between the CBT-I and DBI-I groups. A significant race/ethnicity main effect indicated lower RED scores across both time periods among Latinos compared to Asians; no other race/ethnicity differences were found. Regarding our study hypotheses, Time X Condition coefficients indicated that DBI-I participants showed significantly greater reductions in reward-based eating drive over time than participants in the NI condition (z = -2.05, p = .045).  $\beta$  for the DBI-I/NI Time X interaction coefficient was 0.31, an effect size in the “medium” range (Cohen, 1988). No Time x Condition interaction effect was detected for comparisons between CBT-I and NI, or DBI-I and CBT-I.

3.3.1. Reliable change

Mean reliable change index scores for DBI-I, CBT-I, and NI were -0.64, 0.01, and -0.10, respectively (F(2) = 1.79, p = .17). Twenty-one percent of DBI-I completers (n = 11), 11% of CBT-I completers (n = 7), and 19% NI of participants (n = 12) showed clinically significant declines in RED scale scores from pre- to post-treatment per Reliable Change Index scores. Rates of clinically reliable RED scale score decline did not differ significantly between groups ( $\chi^2(2) = 2.14, p = .34$ ). Six percent of DBI-I

completers, 16% of CBT-I completers, and 14% of NI participants showed clinically significant increases in RED scale scores from pre- to post-treatment per Reliable Change Index scores. Rates of clinically reliable increase in RED scale scores did not differ significantly between groups ( $\chi^2(2) = 3.18, p = .20$ ). Thus, although regression analyses showed that pre to post-treatment decline in RED scale scores was larger for DBI-I relative to NI, the findings may not be clinically significant.

#### 4. Discussion

This study expanded on a previous randomized controlled trial by testing the effects of two strategies for internet ED prevention, CBT-I and DBI-I, on reward-based eating drive. We hypothesized that CBT-I would result in a greater reduction in reward-based eating drive than DBI-I. Contrary to our hypothesis, DBI-I was more effective at reducing reward-based eating drive than NI, whereas no effects were found for CBT-I. This finding is unexpected, as the CBT-I intervention was more effective at reducing global eating pathology and dieting than NI in primary data analyses, whereas DBI-I showed no effect (Chithambo & Huey, 2017). These findings may have occurred because CBT aims to restructure thought and behavior patterns; however, reward-based eating drive is characterized by descriptive traits (e.g., “food is always on my mind”) that may be less amenable to cognitive restructuring. Similar to previous trials utilizing dissonance-based interventions for eating behavior, the DBI-I vs. NI effect size was moderate in size (Stice et al., 2012). Our findings provide preliminary evidence in support of internet interventions for the reduction of reward-based eating drive.

DBI-I takes a sociocultural perspective, encouraging participants to argue against the thin-ideal (Stice et al., 2007). Strategies for changing eating behaviors are not explicitly discussed. However, dissonance-based intervention promotes an integrated self-concept by addressing the discrepancy between acknowledging the harm of the thin-ideal and harboring self-critical body attitudes. As body preoccupation and food preoccupation are closely associated (Rosen, 2013), challenging the thin-ideal may have reduced preoccupation with body shape and in turn, preoccupation with food. Although CBT-I discusses the associations between negative thoughts and eating behaviors, the intervention does not discuss the societal underpinnings of such thoughts, a potential explanation for the superior performance of DBI-I. Furthermore, the mechanism of change in CBT involves the identification of negative thoughts and emotions. Therefore, CBT may not be well suited to addressing non-pathological sensitivity to palatable food, as reward-based eating drive is theorized to be activated by environmental food cues, rather than negative thoughts or emotions. More work is needed to identify potential mechanisms that account for the effect of DBI-I on reward-based eating drive.

Although previous interventions have targeted the reduction of reward-based eating among participants with overweight or obesity using mindfulness techniques, this is the first study to examine interventions for reward-based eating in a sample with elevated ED risk, and predominantly of average weight. Previous research has found that mindfulness-based techniques are efficacious for reducing reward-based eating and related constructs (e.g., craving-related eating; Mason et al., 2016, Mason et al., 2018). Furthermore, reward-based eating has been shown to mediate the effects of a mindfulness-based weight loss intervention (Mason et al., 2016). However, little is known about associations between reward-based eating drive and clinical ED symptoms that contribute to weight gain, such as binge eating. This is a potential area for future study.

##### 4.1. Strengths and limitations

Regarding study strengths, this investigation showed that reward-based eating drive can be reduced with a remotely-delivered, self-guided program. It is the first to test the effect of ED prevention on reward-based eating drive. As some studies have reported that discussing eating

habits can be iatrogenic for subsyndromal individuals (Mann et al., 1997), it is encouraging that reward-based eating drive was reduced despite minimal content on altering eating behavior. The sample was racially/ethnically diverse, supporting generalizability.

Regarding limitations, longer term effects of the interventions on reward-based eating drive were not examined. The lack of follow-up data allows us to draw only preliminary conclusions at best regarding the effects of internet treatment on reward-based eating drive. The treatment content did not include specific strategies for modifying eating habits, making it difficult to ascertain mechanisms that explain the outcomes. Though the application of CBT-I and DBI-I allowed for the direct comparison of active treatments, the NI control group lacked active intervention components and did not account for demand characteristics. In addition, we did not objectively assess biological measures that are less susceptible to expectancies and demand characteristics, such as BMI. Instead, we utilized self-reported BMI as a control variable, potentially biasing our results. Though the first author reviewed the participants' responses to confirm they were on task, the intervention content did not include validation questions to ascertain that participants read the material. Thus, we are unable to verify whether participants were fully engaged with the intervention content.

Because our study was 100% female, the results cannot be generalized to males. As the study was intended for a high-risk, non-clinical sample, we did not utilize clinical assessment measures to determine study eligibility. Instead, we used the community mean of the Weight Concerns Scale as a cutoff to identify eligible participants. However, adopting a more rigorous standard (such as quartiles) may have increased the likelihood of identifying clinically relevant differences between groups. Due to differing IRB boards, participants at University A were compensated similarly regardless of condition; in contrast, participants at University B were compensated in proportion to time spent on the study. This could have biased University B participants' engagement in study activities. We were unable to examine individual-level session progress because of a technical error that anonymized a portion of the respondents' data. Therefore, the extent to which between-condition discrepancies in study adherence and/or engagement affected study outcomes is unclear.

The RED scale evinces high reliability and concurrent and predictive validity in other studies (Epel et al., 2014; Mason et al., 2015; Mason et al., 2016). However, the reliability of the reward-based eating measure in our study was relatively low ( $\omega = 0.70$ ). The discrepancy in reliability estimates may relate to differences between our sample and previous RED validation samples. Our sample consisted of women with elevated risk for developing an ED as indicated by high weight concern. Previous RED validation studies, on the other hand, did not use samples with elevated weight concern (e.g., Epel et al., 2014). In addition, the RED scale was originally validated in community samples (Epel et al., 2014), whereas the current sample comprises university students. These differences in sample composition (younger, more educated, and with elevated ED risk) may be the source of the discrepancy. As such, the RED scale may not adequately measure reward-based eating drive in our study sample. In addition, though a significant effect was found for the DBI-I vs. NI interaction, reliable change index analyses showed no difference between conditions in symptom declines over time. Consequently, the results suggest that our findings, although statistically significant, were not clinically reliable. Moreover, although our study was powered at 0.87 to detect a medium effect size, power to detect a small effect size was 0.17. Thus, the analysis was not adequately powered to detect small effect sizes, reducing our chances of detecting a true effect for the CBT-I vs. NI and DBI-I vs. CBT-I comparisons.

As the interventions were designed to be parallel in content and structure, their protocols did not precisely replicate previously validated ED prevention protocols. For example, DBI-I was shorter in length than the Body Project, the most empirically supported dissonance-based intervention (Stice et al., 2019), and did not include a group interaction component, potentially attenuating treatment effects. CBT-I was adapted from the self-help manual *The Body Image Workbook* (Cash, 1997), and

was designed to replicate the structure of DBI-I. Accordingly, CBT-I as applied in this study was a novel approach compared to previously validated CBT prevention programs, such as Student Bodies (Beintner et al., 2012). It is possible that effect sizes may have been larger had the study applied interventions adherent to the original protocols. This limitation is particularly relevant to CBT—I. Although the intervention was designed to be parallel in structure with DBI-I, no known previous studies have demonstrated efficacy of a four-session CBT ED prevention program such as the one applied in the current study. As such, though this study provides a theory-based comparison of dissonance-based intervention and CBT, the findings do not speak to the efficacy of specific protocols such as The Body Project and Student Bodies.

The Cochrane Collaboration recommends selecting target outcomes prior to data collection risk of bias (Higgins & Green, 2011). The RED outcome measure was introduced to the study after addition of the second research site; the increase in participants allowed the study to reach adequate power to detect significant results. As AJT was based at the second study site and sought to contribute substantively to the project, given her previous research the authors agreed that assessing reward-based eating drive would be an innovative addition to the literature. However, the later inclusion of the RED scale presents a study limitation by increasing risk for outcome reporting bias.

#### 4.2. Future research

Given that this is the first randomized-controlled trial to test internet ED prevention for the reduction of reward-based eating, replication is needed. Future research should utilize larger, more diverse samples to optimize the generalizability of study findings. More research is needed to test potential mechanisms of the effect of DBI-I on reward-based eating, such as reductions in food or body preoccupation. The influence of participant adherence on treatment effects for reward-based eating drive interventions also needs further study. Given the role of reward-based eating drive in the development of both ED pathology and obesity, this study provides a critical first step in the amelioration of this important eating symptom.

#### Declaration of competing interest

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Dr. Haderlein and Dr. Tomiyama designed the study. Dr. Tomiyama contributed to the literature review and selection of statistical procedures. Dr. Haderlein conducted statistical analyses and produced the first draft of the manuscript. All authors contributed to and have approved the final manuscript.

All other authors declare that they have no conflicts of interest.

The views expressed in this article are those of the authors and do not necessarily reflect the position or policy of the Department of Veterans Affairs or the United States government.

#### Acknowledgements

This material is based upon work supported with resources and the use of facilities at the West Los Angeles VA Medical Center, 11301 Wilshire Blvd, Los Angeles, CA 90073.

The views expressed in this article are those of the authors and do not necessarily reflect the position or policy of the Department of Veterans Affairs or the United States government.

#### References

- Adam, T. C., & Epel, E. S. (2007). Stress, eating and the reward system. *Physiology & Behavior*, 91(4), 449–458.
- American Psychiatric Association. (2013). *Diagnostic and statistical of mental disorders* (5th ed.). <https://doi.org/10.1176/appi.books.9780890425596>

- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck depression inventory-II (BDI-II)*. San Antonio, TX: Psychological Corporation.
- Beintner, I., Jacobi, C., & Taylor, C. B. (2012). Effects of an Internet-based prevention programme for eating disorders in the USA and Germany — A meta-analytic review. *European Eating Disorders Review*, 20(1), 1–8. <https://doi.org/10.1002/erv.1130>
- Cash, T. F. (1997). *The body image workbook: An 8-step program for learning to like your looks*. New Harbinger Publications Inc.
- Chithambo, T. P., & Huey, S. J., Jr. (2017). Internet-delivered eating disorder prevention: A randomized controlled trial of dissonance-based and cognitive-behavioral interventions. *International Journal of Eating Disorders*, 50(10), 1142–1151.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates.
- Daubenmier, J., Kristeller, J., Hecht, F. M., Maninger, N., Kuwata, M., Jhaveri, K., Lustig, R. H., Kemeny, M., Karan, L., & Epel, E. (2011). Mindfulness intervention for stress eating to reduce cortisol and abdominal fat among overweight and obese women: An exploratory randomized controlled study. *Journal of Obesity*, 2011.
- Epel, E. S., Dallman, M. F., & Tomiyama, A. J. (2012). Stress and reward: Neural networks, eating, and obesity. In *Food and addiction: A comprehensive handbook* (pp. 266–272). New York: Oxford University Press.
- Epel, E. S., Tomiyama, A. J., Mason, A. E., Laraia, B. A., Hartman, W., Ready, K., Kessler, D., ... (2014). The reward-based eating drive scale: A self-report index of reward-based eating. *PLoS One*, 9(6), Article e101350.
- Fingeret, M. C., Warren, C. S., Cepeda-Benito, A., & Gleaves, D. H. (2006). Eating disorder prevention research: A meta-analysis. *Eating Disorders*, 14(3), 191–213. <https://doi.org/10.1080/10640260600638899>
- Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders*, 2(2), 15–34.
- Giuliani, N. R., Mann, T., Tomiyama, A. J., & Berkman, E. T. (2014). Neural systems underlying the reappraisal of personally craved foods. *Journal of Cognitive Neuroscience*, 26(7), 1390–1402.
- Gordon, R. S., Jr. (1983). An operational classification of disease prevention. *Public Health Reports*, 98(2), 107.
- Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors*, 7(1), 47–55. [https://doi.org/10.1016/0306-4603\(82\)90024-7](https://doi.org/10.1016/0306-4603(82)90024-7)
- Hedeker, D., & Gibbons, R. D. (1997). Application of random-effects pattern-mixture models for missing data in longitudinal studies. *Psychological Methods*, 2(1), 64.
- Higgins, J., & Green, S. (2011). *Cochrane handbook for systematic reviews of interventions*. The Cochrane Collaboration: Available from [www.handbook.cochrane.org](http://www.handbook.cochrane.org).
- 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(1), 12.
- Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology*, 3, 1–27. <https://doi.org/10.1146/annurev.clinpsy.3.022806.091432>
- Killen, J. D., Taylor, C. B., Hayward, C., Haydel, K. F., Wilson, D. M., Hammer, L., Strachowski, D., ... (1996). Weight concerns influence the development of eating disorders: A 4-year prospective study. *Journal of Consulting and Clinical Psychology*, 64(5), 936–940. <https://doi.org/10.1037/0022-006X.64.5.936>
- Le, L. K.-D., Barendregt, J. J., Hay, P., & Mihalopoulos, C. (2017). Prevention of eating disorders: A systematic review and meta-analysis. *Clinical Psychology Review*, 53, 46–58.
- Maassen, G. H. (2004). The standard error in the Jacobson and Truax reliable change index: The classical approach to the assessment of reliable change. *Journal of the International Neuropsychological Society*, 10(06), 888–893.
- Mann, T., Nolen-Hoeksema, S., Huang, K., Burgard, D., Wright, A., & Hanson, K. (1997). Are two interventions worse than none? Joint primary and secondary prevention of eating disorders in college females. *Health Psychology*, 16(3), 215–225. <https://doi.org/10.1037/0278-6133.16.3.215>
- Manwaring, J. L., Bryson, S. W., Goldschmidt, A. B., Winzelberg, A. J., Luce, K. H., Cunniff, D., Taylor, B. C., ... (2008). Do adherence variables predict outcome in an online program for the prevention of eating disorders? *Journal of Consulting and Clinical Psychology*, 76(2), 341–346.
- Mason, A. E., Epel, E. S., Aschbacher, K., Lustig, R. H., Acree, M., Kristeller, J., Daubenmier, J., ... (2016). Reduced reward-driven eating accounts for the impact of a mindfulness-based diet and exercise intervention on weight loss: Data from the SHINE randomized controlled trial. *Appetite*, 100, 86–93. <https://doi.org/10.1016/j.appet.2016.02.009>
- Mason, A. E., Jhaveri, K., Cohn, M., & Brewer, J. A. (2018). Testing a mobile mindful eating intervention targeting craving-related eating: feasibility and proof of concept. *Journal of Behavioral Medicine*, 41(2), 160–173. <https://doi.org/10.1007/s10865-017-9884-5>
- Mason, A. E., Laraia, B., Daubenmier, J., Hecht, F. M., Lustig, R. H., Puterman, E., Adler, N., Dallman, M., Kiernan, M., Gearhardt, A. N., & Epel, E. S. (2015). Putting the brakes on the “drive to eat”: Pilot effects of naltrexone and reward-based eating on food cravings among obese women. *Eating Behaviors*, 19, 53–56. <https://doi.org/10.1016/j.eatbeh.2015.06.008>
- McNeish, D. (2018). Thanks coefficient alpha, we'll take it from here. *Psychological Methods*, 23(3), 412–433. <https://doi.org/10.1037/met0000144>
- 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(3), 128–133. <https://doi.org/10.1016/j.brat.2012.12.001>

- Nye, S., & Cash, T. (2006). Outcomes of manualized cognitive-behavioral body image therapy with eating disordered women treated in a private clinical practice. *Eating Disorders, 14*(1), 31–40. <https://doi.org/10.1080/10640260500403840>
- Pinaquy, S., Chabrol, H., Simon, C., Louvet, J.-P., & Barbe, P. (2003). Emotional eating, alexithymia, and binge-eating disorder in obese women. *Obesity Research, 11*(2), 195–201.
- R Core Team, & R. C. (2013). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing. Retrieved from <http://www.R-project.org>.
- Rosen, J. C. (2013) (Vol. 1).
- Starkweather, J. (2012). Step out of the past: Stop using coefficient alpha; there are better ways to calculate reliability. *Research and Statistical Support, 6*, 6–12.
- Stice, E., Becker, C. B., & Yokum, S. (2013). Eating disorder prevention: Current evidence-base and future directions. *International Journal of Eating Disorders, 46*(5), 478–485.
- Stice, E., Johnson, S., & Turgon, R. (2019). *Psychiatric Clinics, 42*(2), 309–318.
- Stice, E., Mazotti, L., Weibel, D., & Agras, W. S. (2000). Dissonance prevention program decreases thin-ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms: A preliminary experiment. *International Journal of Eating Disorders, 27*(2), 206–217.
- Stice, E., Presnell, K., & Spangler, D. (2002). Risk factors for binge eating onset in adolescent girls: A 2-year prospective investigation. *Health Psychology, 21*(2), 131.
- Stice, E., Rohde, P., Durant, S., & Shaw, H. (2012). A preliminary trial of a prototype internet dissonance-based eating disorder prevention program for young women with body image concerns. *Journal of Consulting and Clinical Psychology, 80*(5), 907–916. <https://doi.org/10.1037/a0028016>
- Stice, E., & Shaw, H. (2004). Eating disorder prevention programs: A meta-analytic review. *Psychological Bulletin, 130*(2), 206–227. <https://doi.org/10.1037/0033-2909.130.2.206>
- Stice, E., Shaw, H., & Marti, C. N. (2007). A meta-analytic review of eating disorder prevention programs: Encouraging findings. *Annual Review of Clinical Psychology, 3*, 207–231. <https://doi.org/10.1146/annurev.clinpsy.3.022806.091447>
- Strachan, M. D., & Cash, T. F. (2002). Self-help for a negative body image: A comparison of components of a cognitive-behavioral program. *Behavior Therapy, 33*(2), 235–251. [https://doi.org/10.1016/S0005-7894\(02\)80027-2](https://doi.org/10.1016/S0005-7894(02)80027-2)
- Twisk, J., Bosman, L., Hoekstra, T., Rijnhart, J., Welten, M., & Heymans, M. (2018). Different ways to estimate treatment effects in randomised controlled trials. *Contemporary Clinical Trials Communications, 10*, 80–85.
- Watson, H. J., Joyce, T., French, E., Willan, V., Kane, R. T., Tanner-Smith, E. E., McCormack, J., Dawkins, H., Hoiles, K. J., & Egan, S. J. (2016). Prevention of eating disorders: A systematic review of randomized, controlled trials. *International Journal of Eating Disorders, 49*(9), 833–862. <https://doi.org/10.1002/eat.22577>
- Williamson, D. A., White, M. A., York-Crowe, E., & Stewart, T. M. (2004). Cognitive-behavioral theories of eating disorders. *Behavior Modification, 28*(6), 711–738. <https://doi.org/10.1177/0145445503259853>
- Wilson, G. T., Fairburn, C. C., Agras, W. S., Walsh, B. T., & Kraemer, H. (2002). Cognitive-behavioral therapy for bulimia nervosa: Time course and mechanisms of change. *Journal of Consulting and Clinical Psychology, 70*(2), 267–274. <https://doi.org/10.1037/0022-006X.70.2.267>