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Mediators of Two Selective Prevention Interventions Targeting Both Obesity and Eating Disorders

Paul Rohde,

Oregon Research Institute

Christopher Desjardins,

Oregon Research Institute

Danielle Arigo,

The University of Scranton

Heather Shaw,

Oregon Research Institute

Eric Stice

Oregon Research Institute

Abstract

The present study tested hypothesized mechanisms underlying the effects of two selective prevention interventions targeting both obesity and eating disorders (*Healthy Weight* and the newly developed *Project Health*), relative to video control. Tests examined mediation for the significant weight gain prevention and eating disorder symptom prevention effects previously reported. College students ($N = 364$; 72% women) with weight concerns were randomized to condition and assessed for 2-years post-intervention. *Project Health* participants had significant improvements in 2 of the 7 proposed mediators relative to comparisons (i.e., cognitive dissonance, the unhealthy Western dietary pattern) but change in these variables did not mediate its effect on long-term BMI change. Two variables emerged as full mediators of the eating disorder prevention effects for both experimental interventions: body dissatisfaction and negative affect. Analyses failed to support the exploratory hypothesis that change in eating disorder symptoms mediated the effects of condition on BMI gain. This report is the among the first to examine mediation for programs aimed at preventing both weight gain and eating disorders, particularly in mixed-gender groups. Mediational analyses are essential in identifying the mechanism of intervention action, which can inform improvements to prevention programs.

Keywords

Eating disorder; obesity; prevention; mediation; cognitive dissonance

Corresponding Author: Paul Rohde, Oregon Research Institute, 1776 Millrace Drive, Eugene, Oregon, 97403, USA. paulr@ori.org. Phone: (541) 484-2123. Fax: (541) 484-1108.

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Obesity and eating disorders are prevalent chronic conditions associated with functional impairment, distress, physical and psychiatric morbidity, and mortality (Flegal, Carroll, Kit, & Ogden, 2012; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), prompting the need for prevention programs that reduce the onset of these two major public health problems. Although several prevention programs have sought to reduce future onset of both obesity and eating disorders, until recently only one has affected both outcomes. Compared to assessment controls, the 3-hour *Healthy Weight* selective prevention program reduced weight gain, obesity onset, eating disorder symptoms, and eating disorder onset through 3-year follow-up (Stice, Marti, Spoor, Presnell, & Shaw, 2008; Stice, Shaw, Burton, & Wade, 2006). In *Healthy Weight* young women with body image concerns make small, lasting healthy changes to dietary intake and exercise that bring caloric intake and expenditure into balance, which should reduce weight gain, as well as risk for eating pathology. The participant-driven lifestyle change plan is intended to promote internalization of health goals and executive control over lifestyle choices. A refined version of *Healthy Weight* resulted in less weight gain through 6-month follow-up, greater weight gain prevention through 1-year follow-up for initially overweight youth, greater eating disorder symptom reductions, and a 60% reduction in eating disorder onset over 2-year follow-up, versus educational brochure controls (Stice, Rohde, Shaw, & Marti, 2012, 2013). An expanded 6-session version of *Healthy Weight* produced greater reductions in BMI and eating disorder symptoms than an active comparison control (cognitive reappraisal-based prevention; (Stice et al., 2015).

We subsequently tested whether the efficacy of *Healthy Weight* could be enhanced by adding activities designed to induce cognitive dissonance about consuming unhealthy foods, a sedentary lifestyle, and excess body fat, based on a large body of research supporting the efficacy and effectiveness of a dissonance-based eating disorder prevention program (e.g., Stice et al., 2006, 2008). According to cognitive dissonance theory (Festinger, 1957), discussing the costs of engaging in unhealthy behaviors prompts people to align their attitudes with their publically displayed behavior. Our novel dissonance-enhanced eating disorder/obesity prevention intervention is a 6-hour intervention (*Project Health*) that retained the participant-driven small, gradual healthy lifestyle modification plan, but added written and verbal exercises designed to create dissonance regarding lifestyle behaviors that contribute to excess weight gain, the costs of obesity, and the benefits of staying at a healthy weight.

The current randomized experimental therapeutics trial tested whether *Project Health* produced greater decreases in BMI and eating disorder symptoms, and lower future onset of overweight/obesity and eating disorders, than a 6-session version of the original *Healthy Weight* program (which lacks dissonance-induction activities) and an educational video control. College students ($N = 364$; 72% female) who were at risk for both outcomes due to weight concerns were randomized to condition and completed pretest, posttest, and 6, 12, and 24-month follow-up assessments (Stice, Rohde, Shaw, & Gau, in press). *Project Health* participants showed significantly smaller increases in BMI at 2-year follow-up than both *Healthy Weight* participants and controls (both $d = -.18$), and significantly lower onset of overweight/obesity over 2-year follow-up than *Healthy Weight* participants and controls (13% vs. 21% and 22%, respectively). *Project Health* and *Healthy Weight* participants also

showed significantly greater eating disorder symptom reductions ($d = -.15$ and $-.19$, respectively) and nonsignificantly lower rates of eating disorder onset (3% & 3% vs. 9% respectively; $p = .068$ & $.064$) than controls through 2-year follow-up. It is important to note that including men in this trial reduced the incidence of eating disorder onset, which might explain why these two effects did not reach significance.

These effects are encouraging, and it is important to further investigate the cognitive and/or behavioral processes that mediate these intervention effects, which is the goal of the present study. The aims were to test whether a set of hypothesized mediators accounted for (a) the significant weight gain prevention effects in *Project Health* relative to both the active comparison of *Healthy Weight* and education video controls, and (b) the significant eating disorder symptom prevention effects found in both *Project Health* and *Healthy Weight* relative to controls. An understanding of the mechanism(s) of intervention effects might guide improvements to existing prevention programs. For each of five potential mediators, we examined the degree of change for each proposed mediator during the intervention and whether that degree of change mediated the respective weight gain or eating disorder prevention effects.

The first potential mediator was cognitive dissonance about engaging in lifestyle behaviors that contribute to excess weight gain. Changes in attitudes about healthy behavior and related psychological constructs (e.g., self-efficacy for healthy behavior) have shown mediating effects for school-based obesity prevention programs (van Stralen et al., 2011), and prior eating disorder prevention research found that participants assigned to versions of an intervention that maximized versus minimized dissonance induction showed significantly greater reductions in eating disorder symptoms (Green, Scott, DiYankova, & Gasser, 2005; McMillan, Stice, & Rohde, 2011). Because of the dissonance-inducing activities unique to *Project Health*, including role-plays and written exercises regarding unhealthy foods and sedentary behavior, we anticipated that cognitive dissonance regarding engaging in behaviors that contribute to weight gain would mediate the observed weight gain prevention effects.

The second and third potential mediators were indices of dietary intake and physical activity. These two energy balance-related mechanisms are standard to almost all behaviorally based obesity prevention or treatment programs, and have been shown to mediate the effects of obesity treatments (Norman, Kolodziejczyk, Adams, Patrick, & Marshall, 2013; Teixeira, 2015) and weight gain prevention programs (Partridge, McGeechan, Bauman, Phongsavan, & Allman-Farinelli, 2016). These factors have been examined much less frequently in eating disorder prevention programs. Previous mediation research (Stice, Presnell, Gau, & Shaw, 2007) found that, relative to women in an expressive writing control condition, *Healthy Weight* participants showed significantly greater improvements in healthy eating and physical activity and that change in physical activity (but not dietary intake) correlated significantly with change in eating disorder symptoms. Controlling for either dietary intake or physical activity, however, did not account for the effects of *Healthy Weight* on eating disorder symptom reduction.

The fourth and fifth potential mediators were body dissatisfaction and negative affect, both of which have been previously found to mediate interventions targeting obesity and eating disorders. Positive body image and self-worth, which are inversely correlated with negative affect, mediated the effects of successful overweight/obesity treatment interventions in the majority of existing studies, both in terms of achieving weight loss and increased physical activity (Teixeira et al., 2015). Regarding eating pathology, body dissatisfaction has predicted future increases in negative affect, which in turn predicted increased bulimic symptoms (Stice, 2001). Furthermore, *Healthy Weight* was found to produce significant improvements in body satisfaction and negative affect (Stice et al., 2008), and improved body satisfaction mediated the eating disorder effects of a previous dissonance-based eating disorder prevention program (Seidel, Presnell, & Rosenfield, 2009; Stice, Marti, Rohde, & Shaw, 2011).

In sum, the present study had two aims. First, we tested whether *Project Health* participants, compared to both participants in a credible alternative intervention (*Healthy Weight*) and an obesity education video control intervention, experienced greater improvements in dietary intake, physical activity, negative affect, body dissatisfaction, and greater change in cognitive dissonance regarding engaging in unhealthy lifestyle behaviors, and the degree to which these changes account for the reductions in BMI for *Project Health* participants versus participants in the other conditions. Second, we tested whether the combined sample of *Project Health* and *Healthy Weight* participants, relative to education controls, experienced greater changes in the same potential mediators and the degree to which these changes account for the reductions in eating disorder symptoms found in *Project Health* and *Healthy Weight* versus video controls. These analyses aim to elucidate the mechanisms of action that account for the successful effects of both of these interventions, and inform future iterations of these programs as well as other public health prevention programs.

Methods

Participants and Procedure

Participants ($N = 364$) were 261 young women and 103 young men ($M_{\text{age}} = 19.2$, $SD = 1.2$); 72% were Caucasian, 15% Asian/Pacific Islander, 11% Hispanic/Latino, 3% Black/African American, 3% Alaskan native, and < 1% Hawaiian native. Students ages 17-23 with weight concerns from 3 universities were recruited August, 2012 to March, 2014. Informed written consent was obtained as was approval from Institutional Review Boards from the institute supporting this project and participating universities. Exclusion criteria included a current DSM-IV diagnosis of anorexia nervosa, bulimia nervosa, or binge eating disorder; an interviewer-assessed BMI < 18 or >30; or current participation in another eating disorder or obesity prevention research study.

Eligible participants were randomly assigned to the *Healthy Weight* intervention ($n = 122$), *Project Health* intervention ($n = 119$), or an educational video control condition ($n = 123$) via a random number table. Participants provided interview and survey data at pretest, posttest (intervention termination), and at 6, 12, and 24-month follow-ups, collected by female assessors masked to condition. Participants were financially compensated for completing each assessment. Both interventions consisted of 6 weekly 1-hr group sessions

with 6-10 participants and 2 group leaders. If a participant missed a session, a brief (10-15 min) individual session was conducted to review material when possible. Additional study details are provided in Stice et al. (in press).

Interventions

Healthy Weight.—The main goal of *Healthy Weight* is to make weekly small, incremental, sustainable changes to diet and exercise to achieve a balance between caloric intake and output. Sessions began with a brief review of previous material, presentation of educational handouts, in-session discussions, review of participant-selected behavior change goals, and the development of healthy behavior change plans for the next session. Home exercises consisted of following individualized diet and exercise goals, and keeping a food and exercise log to identify future change goals.

Project Health.—Each session began with a verbal commitment to participate, included discussions of completed home exercises and in-session writing/sharing exercises to create accountability, in-session activities designed to create dissonance regarding engaging in lifestyle behaviors that contribute to weight gain (e.g., discussing how society promotes overeating, roleplay practice refusing requests from others to have unhealthy food or beverages, discussing the importance of making lasting lifestyle changes rather than going on transient diets), and concluded with home exercises designed to create dissonance (e.g., write a letter on the costs of obesity and the benefits of living at a healthy weight, write a letter on costs of being sedentary, list the top 10 costs of unhealthy foods and sedentary behaviors). As in *Healthy Weight*, participants also commit to make small, gradually increasing improvements to their diet and activity levels.

Educational video (control).—Participants were sent an email with a link to a free 51-minute video, *The Weight of the World*, which focused on the costs of the obesity epidemic from various viewpoints.

Assessments

Body mass.—After removing shoes and coats, height was measured to the nearest millimeter using stadiometers and weight was assessed to the nearest 0.1 kg using digital scales. Two measures of each component of BMI (kg/m^2) were obtained and averaged to form our weight change outcome. BMI correlates with direct measures of body fat such as dual energy x-ray absorptiometry ($r = .80-.90$) and health measures (Pietrobelli et al., 1998).

Eating disorder symptoms and diagnosis.—The Eating Disorder Diagnostic Interview (Stice et al., 2006) assessed DSM-IV eating disorder symptoms on a month-to-month basis. Items assessing symptoms (e.g., frequency of binge eating, frequency of compensatory weight control behaviors, overvaluation of weight/shape, fear of weight gain) in the past month at each assessment were summed to form a composite, which served as the eating disorder symptom outcome. This composite has shown internal consistency ($\alpha = .92$), 1-week test-retest reliability ($r = .90$), sensitivity to intervention effects, and predictive validity (Stice et al., 2006). It showed internal consistency ($\alpha = .70$ at T1), inter-rater agreement (ICC $r = .90$), and 1-week test-retest reliability (ICC $r = .93$) in the present trial.

Cognitive dissonance.—A 14-item Dissonance Regarding Unhealthy Lifestyle Behaviors scale measured subjective experience of cognitive dissonance from engaging in unhealthy dietary practices and sedentary behaviors (sample items: “I feel bad when I eat high-fat foods, such as hamburgers and fries”, “Engaging in my typical workout puts me in a positive state of mind”, “I feel better about myself after eating a healthy serving of vegetables”). This scale showed internal consistency ($\alpha = .80$) and 1-month test-retest reliability ($r = .93$) in pilot work with college students. In the current trial the scale showed internal consistency ($\alpha = .84$), in addition to sensitivity to detecting *Project Health* intervention effects relative to video controls (Stice et al., in press).

Dietary Intake.—We used an adaptation of the Block Food Frequency Questionnaire to assess dietary intake (Block FFQ; Block, Hartman, & Naughton, 1990). Participants estimated the frequency of consumption of 78 specific food types over the past 2 weeks and indicated whether their typical serving was small, medium, or large (definitions provided). FFQs correlate (mean $r = .57$) with 4-day food record estimates for energy and most nutrients (Block & Subar, 1992), show 2-week test-retest reliability (mean $r = .69$; Klohe et al., 2005), and provided values within 3% of values obtained from 7-day weighed food records (Surrao et al. 1998). We focused on total caloric intake and two general patterns of food consumption that have been labeled Western and prudent (Hu et al., 1999). The Western pattern indicates an emphasis on processed and calorie-dense foods such as high-fat dairy products and desserts; the prudent pattern describes an emphasis on fruit, vegetables, whole grains, and lean meats/dairy products. The Western pattern has been associated with increased risk for type 2 diabetes and mortality, whereas the prudent pattern appears to have protective effects on these outcomes (Heidemann et al., 2008; Fung et al., 2004). These subscales of the Block FFQ have shown 1-year reproducibility via factor analysis and correlations with plasma biomarkers for micro- and macronutrients (e.g., folate, saturated fat), indicating convergent validity (Fung et al., 2001; Hu et al., 1999). In the present study, internal consistency ranged from .69 to .76 for the prudent pattern and .82 to .86 for the Western pattern across the 5 assessments.

Physical activity.—The Paffenbarger Activity Questionnaire (Paffenbarger, Wing, & Hyde, 1978) assessed physical activity with 20 items that subjectively measured participants’ amount of high, moderate, and low intensity physical activity during the past 2 weeks. Participants noted activity type, as well as its frequency and duration. Scores were combined to create a single measure of total physical activity related to recreational activities (Rubenstein et al., 2011). This scale has shown 1-month test-retest reliability ($r = .72$) and convergent validity with physical activity records ($r = .28 - .86$; Ainsworth, Leon, Richardson, Jacobs, & Paffenbarger, 1993).

Body dissatisfaction.—The 35-item Body Esteem Scale (Franzoi & Shields, 1984) assesses opinions about various body parts and functions, such as legs, face, and muscular strength. It has shown test-retest reliability (Franzoi, 1994); $\alpha = .91$ at baseline.

Negative affect.—The 20-item Negative Affect subscale of the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988) measures general negative affect. It has

shown high stability and test-retest validity ($r = .71$; Watson et al., 1988); $\alpha = .93$ at baseline.

Statistical Analysis

Modeling change in BMI and eating disorder symptoms.—To examine acute and long-term prevention effects on BMI and number of eating disorder symptoms, these effects were modeled separately. To examine the acute effect, we calculated change in outcome from pre-intervention to post-intervention (post – pre). To examine the long-term prevention effect, we modeled outcome change post-intervention by fitting a linear latent curve model (Bollen & Curran, 2006), as shown in Figure 1. We assessed whether the linear latent curve model fit well, based on the CFI, TLI, and RMSEA (e.g., Hu & Bentler, 1999). If the model did not fit well, we considered a quadratic latent curve model.

Condition coding.—For the BMI analyses, we created a contrast that compared *Project Health* participants to the average response of the *Healthy Weight* and control participants. For the eating symptom disorder analyses, we created a contrast that compared the average response of *Project Health* and *Healthy Weight* against control participants. This approach corresponded to the nature of change in these outcomes (Stice et al., in press).

Modeling change in the mediators.—Change scores from pre-intervention to post-intervention were calculated for each prospective mediator, as acute prevention effects are generally strongest (e.g., Stice, Shaw, & Marti, 2006). The change score for each mediator was regressed onto condition controlling for baseline levels of the mediator.

Assessing mediation.—The mediation analysis used the Baron & Kenny (1986) paradigm. First, we assessed whether condition significantly predicted change in the outcome during or after the intervention. Because we were not anticipating suppression or distal mediation effects (Shrout & Bolger, 2002), we considered the first step a necessary precondition to mediation. Change in the outcome during the intervention was regressed onto condition controlling for the outcome at baseline. For the linear latent curve, we regressed the slope of BMI or eating disorder symptoms (posttest to 2-year follow-up) onto condition, controlling for the outcome's intercept (von Soest & Hagtvet, 2011) (i.e., the BMI intercept latent factor), which is analogous to the analysis of covariance model performed when examining the change in the outcome during the intervention and for assessing prospective mediators.

If condition was significantly related to change in outcome, either during or after the intervention, we proceeded to test if condition was significantly related to change in the proposed mediators; if change in the mediators were significant predictors of change in the outcome; and whether mediation, either partial or complete, occurred by testing the relative indirect effects. These final steps were combined and allowed us to examine steps 2, 3, and 4 in the Baron & Kenny (1986) paradigm simultaneously.

Path analysis was used to assess the acute (i.e., pre-post) prevention effect and structural equation modeling was used to assess the long-term (i.e., follow-up) prevention effect. These models were fit in R (R Core Team, 2017) using the lavaan package (Rosseel, 2013).

Because of missing data, we used full information maximum likelihood, which has been shown to give unbiased parameter estimates when the data are at least missing at random (Schafer & Graham, 2002). To test the relative indirect effects, we used 95% bias-corrected bootstrapped confidence intervals based on 10,000 samples. Because we examined multiple mediators, we applied the Benjamini-Hochberg method (BH, Benjamini & Hochberg, 1995). The BH method works by controlling the false discovery rate, and relative to the Bonferroni's correction is more powerful, while still adequately protecting against Type I errors (Williams, Jones, & Tukey, 1999). In this study, we corrected for all tests associated with mediation performed across both outcomes (e.g., BMI and eating symptoms). This allowed us to correct for the inflated Type I error resulting from multiple testing. Confidence intervals that did not contain zero indicated that the proposed mediator mediated the effect of condition on outcome (Hayes & Preacher, 2014).

Results

Assessing the Mediation Precondition for BMI

Acute prevention effect.—Figure 2 shows the change in BMI during (the dark gray rectangle) and after the intervention. The points in Figure 2 correspond to the connected group means at each assessment window. Figure 2 shows that during the intervention, BMI values for *Project Health* participants decreased whereas values for *Healthy Weight* and control participants increased. However, these BMI changes during the intervention were not statistically significant ($z = -1.384$; $p = .166$). Therefore, we did not examine mediation of an acute prevention effect.

Long-term prevention effect.—Figure 2 shows that after the intervention, the combined group of *Healthy Weight* and control participants experienced greater BMI gains than *Project Health* participants. A linear latent growth curve modeled BMI change post-intervention. This model had good fit based on CFI (0.979) and TLI (0.974) but only adequate fit based on the RMSEA (0.111). A quadratic latent growth curve was considered but did not converge. However, a quadratic growth curve model and a linear growth curve model were fit in a mixed effects modeling framework using the lme4 package (Bates, Mächler, Bolker, & Walker, 2015) and the quadratic growth curve did not represent a significant improvement in model fit ($\chi^2[2] = 3.353$, $p = .187$). Inspection of residual plots gave no indication that the linear model was inappropriate. Thus, we modeled BMI using a linear latent growth curve model.

Condition was a significant predictor of BMI change post-intervention controlling for BMI intercept ($z = -2.479$, $p = .013$). *Project Health* participants had more gradual increases in BMI post-intervention relative to *Healthy Weight* and control. The slope for *Project Health* was expected to be .024 units lower than the slope for *Healthy Weight* and control. Because BMI change post-intervention was associated with condition, we examined mediation of the long-term prevention effect.

Mediation Analysis of the Long-term Prevention Effect for BMI

Table 1 shows the adjusted (controlling for baseline variable level) means of the proposed mediators by condition contrasts. Mediation analyses controlled for baseline levels of the proposed mediator. Table 2 shows the correlation matrix between the contrast variables and potential mediators.

Cognitive dissonance.—*Project Health* participants showed a greater increase in cognitive dissonance relative to the average of *Healthy Weight* and control participants ($z = 3.111$, adjusted $p = .008$). However, change in cognitive dissonance during the intervention did not predict BMI change post-intervention ($z = 0.438$, adjusted $p = .710$).

Caloric intake.—Because of a large positive skew, caloric intake was log transformed prior to calculating the difference score. *Project Health* participants did not show a significantly greater reduction in log-transformed calories consumed than the average of *Healthy Weight* and control participants ($z = -2.04$, adjusted $p = .072$).

Prudent pattern.—Change in prudent pattern did not differ between *Project Health* participants and the average of *Healthy Weight* and control participants ($z = 1.831$, adjusted $p = .102$).

Western pattern.—*Project Health* participants showed greater reductions in Western pattern relative to the average of *Healthy Weight* and control participants ($z = -3.129$, adjusted $p = .008$). However, change in Western pattern during the intervention did not predict BMI change post-intervention ($z = -0.113$, adjusted $p = .910$).

Physical activity.—Change in total physical activity related to engagement in recreational activities did not differ between *Project Health* participants and the average of *Healthy Weight* and control participants ($z = -0.482$, adjusted $p = .703$).

Body dissatisfaction.—Change in body dissatisfaction did not differ between *Project Health* participants and the average of *Healthy Weight* and control participants ($z = -0.987$, adjusted $p = .409$).

Negative affect.—Change in negative affect did not differ between *Project Health* participants and the average of *Healthy Weight* and control participants ($z = -0.340$, adjusted $p = .760$).

Assessing the Mediation Precondition for Eating Disorder Symptoms

Acute prevention effect.—Figure 3 shows the change in eating disorder symptoms during (the dark gray rectangle) and after the intervention. Figure 3 shows that during the intervention *Project Health* and *Healthy Weight* participants had a steeper decrease in eating disorder symptoms than controls. This change in eating symptoms during the intervention was significant ($z = -25.867$, $p < .001$) and we therefore examined mediation of the acute prevention effect.

Long-term prevention effect.—Figure 3 shows that after the intervention, the combined group of *Healthy Weight* and *Project Health* participants had similar trajectories in eating symptoms as control participants. A linear latent growth curve tested for a difference between the groups post-intervention. This model had good fit based on CFI (0.979), TLI (0.974) and acceptable fit based on the RMSEA (0.061). Condition was not a significant predictor of change in eating symptoms post-intervention controlling for the eating symptoms intercept ($z = -0.394$, $p = .694$), suggesting that improvements observed during the intervention persisted over the 2-year follow-up. Because change in eating symptoms post-intervention was not associated with condition, we did not examine mediation of the long-term prevention effect.

Mediation Analysis of the Acute Prevention Effect for Eating Disorder Symptoms

Table 3 shows the adjusted (controlling for baseline variable level) means of the proposed mediators by condition. Mediation analysis control for baseline levels of the proposed mediator.

Cognitive dissonance.—*Project Health* and *Healthy Weight* participants showed greater increases in cognitive dissonance relative to the control participants ($z = 3.874$, adjusted $p = .001$). However, change in cognitive dissonance during the intervention did not predict eating disorder symptom change during the intervention ($z = 1.105$, adjusted $p = .355$).

Caloric intake.—Because of a large positive skew, caloric intake was log transformed prior to calculating the difference. *Project Health* and *Healthy Weight* participants showed a greater reduction in caloric intake than control participants ($z = -2.461$, adjusted $p = .041$). However, change in log-transformed caloric intake during the intervention did not predict eating disorder symptom change during the intervention ($z = 0.887$, adjusted $p = .453$).

Prudent pattern.—*Project Health* and *Healthy Weight* participants showed a greater increase in prudent pattern relative to control participants ($z = 3.567$, adjusted $p = .001$). However, change in prudent pattern did not significantly predict change in eating disorder symptom ($z = -2.229$, adjusted $p = .058$).

Western pattern.—*Project Health* and *Healthy Weight* participants had greater decreases in Western pattern relative to control participants ($z = -3.747$, adjusted $p = .001$). However, change in Western pattern during the intervention did not predict eating disorder symptom change during the intervention ($z = 1.537$, adjusted $p = .180$).

Physical Activity.—Change in total physical activity did not differ between the average of *Project Health* and *Healthy Weight* and control participants ($z = 1.386$, adjusted $p = .229$).

Body dissatisfaction.—*Project Health* and *Healthy Weight* participants had greater reductions in body dissatisfaction than control participants ($z = -3.261$, adjusted $p = .003$). Change in body dissatisfaction predicted change in eating symptoms ($z = -2.385$, adjusted $p = .045$), such that a one-unit increase in body dissatisfaction during the intervention was associated with a 1.01 increase in the number of eating symptoms. The indirect effect of condition on change in eating symptoms through change in body dissatisfaction was

significant (-0.175 , 95% CI = -0.42 , -0.04), providing support for this second mediation effect. Relative to the control participants, *Project Health* and *Healthy Weight* participants decreased 0.18 eating symptoms because of a greater decrease in body dissatisfaction, which in turn decreased the number of eating symptoms. Condition became a nonsignificant predictor after controlling for body dissatisfaction ($z = -2.036$, adjusted $p = .072$), consistent with body dissatisfaction mediating the effect of condition on eating disorder symptom change.

Negative affect.—*Project Health* and *Healthy Weight* participants showed greater reductions in negative affect than controls ($z = -3.020$, adjusted $p = .011$). Change in negative affect predicted change in eating disorder symptoms ($z = 4.678$, adjusted $p < .001$), such that a one-unit increase in negative affect during the intervention was associated with a 1.68 increase in the number of eating disorder symptoms. The indirect effect of condition on change in eating disorder symptoms through change in negative affect was significant (-0.28 , 95% CI = -0.69 , -0.10), providing support for this third mediation effect. Relative to the control participants, *Project Health* and *Healthy Weight* participants decreased 0.28 eating disorder symptoms because of a greater decrease in negative affect, which in turn decreased the number of eating disorder symptoms. Condition became a nonsignificant predictor after controlling for negative affect ($z = -2.047$, adjusted $p = .072$), consistent with negative affect mediating the effect of condition on eating disorder symptom change.

Exploratory Analysis: Degree to Which Changes in Outcomes Mediate each Other

Given the rapid reduction of eating disorder symptoms and the more gradual effects noted for weight gain prevention, we explored the possibility that acute (pre-post) reductions in eating disorder symptoms might mediate the long-term effects found for *Project Health* on BMI (given the exploratory nature of these analyses, we did not correct for experiment-wide number of tests). Because *Healthy Weight* participants also showed acute effects in eating disorder symptoms, our comparison group was the video control. However, for completeness, we computed mediational tests of whether pre- to post-intervention change in eating disorder symptoms mediated any weight gain prevention effects of *Healthy Weight* vs. controls over follow-up, as *Healthy Weight* participants showed significant acute phase eating disorder symptom reductions. Given that there were no significant differences in BMI during the intervention and no significant differences in change in eating disorder symptoms from post to 2-year follow-up, we did not test whether pre- to post-intervention changes in BMI mediate the intervention effects on differential change in eating disorder symptoms after posttest.

The adjusted means for eating disorder symptoms for *Project Health*, *Healthy Weight*, and control participants are shown at the bottom of Table 3. Both *Project Health* ($z = -1.984$, $p = .047$) and *Healthy Weight* participants ($z = -2.247$, $p = .025$) had greater reductions in eating disorder symptoms than controls. Change in eating disorder symptoms during the intervention predicted change in BMI after the intervention ($z = -2.787$, $p = .005$), such that a one-unit increase in eating disorder symptoms during the intervention was associated with a decrease of .002 in the BMI slope after the intervention. However, the 95% bootstrapped

bias-corrected confidence intervals contained zero for both contrasts implying there was no mediation.

Discussion

The aims of this study were to test whether a set of hypothesized mechanisms (1) accounted for the significant weight gain prevention effects previously reported in *Project Health* relative to both the active comparison intervention, *Healthy Weight*, and the education video control, and (2) accounted for the significant eating disorder symptom prevention effects that were detected for both *Project Health* and *Healthy Weight* interventions relative to the video control. The present study is important because it is the first to examine potential mediators of two interventions that both aimed to simultaneously prevent unhealthy weight gain and the onset of eating pathology. A second unique feature of the present study is that participants included both women and men, as very little is known regarding successful weight gain/eating disorder prevention efforts with men.

We found a significant long-term (but not acute) prevention effect for weight gain in *Project Health* relative to the combined sample of *Healthy Weight* and video controls, and conversely, a significant acute (but not long-term) prevention effect for change in eating disorder symptoms for the combined *Project Health* and *Healthy Weight* conditions relative to video controls. The acute phase (pre- to post-intervention) BMI effect was in the same direction as the long-term BMI scores but may have not been significant due to the more gradual nature of change in weight compared to eating disorder symptoms. The greater reduction in BMI for *Project Health* versus *Healthy Weight* and control participants indicates that the former group entered had greater negative energy balance than participants in the other conditions, as BMI constitutes an objective measure. Regarding eating disorder symptom change, the acute (pre- to post-intervention) effect was significant, but then the two lines remained relatively stable and flat, indicating that the intervention effects for symptoms persisted over 2-year follow-up (Figure 3). Thus, we attempted to account for the long-term BMI changes and the more acute phase eating disorder symptom changes in relation to specific active conditions (*Project Health* and both interventions, respectively).

Regarding potential mediators of the long-term weight gain prevention effect, *Project Health* participants had significant improvements in two of the seven examined variables relative to the combined comparison group of *Healthy Weight* and video control participants, with significant increases in cognitive dissonance and reductions in the Western pattern of unhealthy food choices. However, change in these variables did not correlate with change in BMI, which was the next requirement for mediation. Condition differences for the remaining five variables – total caloric intake, prudent dietary pattern, physical activity, body dissatisfaction, and negative affect – were nonsignificant. It is possible that the measures we used were not sufficiently reliable and valid to provide an optimal test of mediation. For instance, self-report measures of dietary intake and physical activity have shown limited validity (Lichtman et al., 1992).

Regarding potential mediators of the acute prevention effect for eating disorder symptoms of the combined sample of *Project Health* and *Healthy Weight* participants relative to video

controls, two variables emerged as significant mediators of these prevention effects: reductions in body dissatisfaction and negative affect. The mediating effect of body dissatisfaction converges with prior evidence that body dissatisfaction has been found to mediate the effects of *Healthy Weight* (Stice et al., 2008) and other eating disorder prevention programs (Seidel, Presnell, & Rosenfield, 2009; Stice, Marti, Rohde, & Shaw, 2011) on reductions in eating disorder symptoms, but extends this body of research by including the new dissonance-based *Project Health* intervention and by examining mediational effects in mixed-gender groups. Regarding the second mediator in the present study, it is intriguing that reductions in negative affect appeared to play a role in mediating the effects of both interventions on reductions in eating disorder symptoms, particularly given that neither *Project Health* nor *Healthy Weight* specifically targeted negative affect. We previously reported that *Healthy Weight* produced significant improvements in negative affect (Stice et al., 2008) but it was not examined as an intervention mediator in that report. The present report is novel in its observation of mediational effects of negative affect for both interventions and for both women and men. The finding suggests that improving lifestyle behaviors linked to obesity can have a positive effect on emotional functioning, which subsequently reduce the onset of eating pathology. Clinically, the findings suggest that increasing the degree to which interventions produce stronger improvements in body dissatisfaction and negative affect will result in greater eating disorder prevention effects.

Given the nature of effects of *Project Health* and *Healthy Weight* on change in BMI and eating disorder symptoms relative to the educational video controls, one interpretation was that the reductions in eating disorder symptoms might mediate the effect of the intervention on BMI gain reductions. Thus, we examined whether change in eating disorder symptoms mediates the effects of condition on BMI gain effects as an exploratory analysis. Previous research provides some evidence that eating disorder symptoms increase the risk for future weight gain. For instance, we previously found that unhealthy weight control behaviors increased risk for future weight gain (Stice, Cameron, Killen, Hayward, & Taylor, 1999) and that binge eating predicted obesity onset in community adolescents (Stice, Presnell, & Spangler, 2002). It therefore is reasonable to test whether an intervention that reduces eating disorder symptoms is associated with a subsequent reduction in BMI gains. Although we found that change in eating symptoms during the intervention predicted change in BMI in the follow-up period, the 95% confidence intervals of the indirect effects of the conditions through eating disorder symptoms on BMI contained zero, thus failing to support this mediational hypothesis. Nonetheless, it is vital to capture the potential dynamic interplay between eating disorder symptoms and weight change, in part because this has been somewhat ignored in the literature.

Four additional variables were examined that showed significant condition effects in both the first (*Project Health* versus an average of the two other conditions) and the second (*Project Health* plus *Healthy Weight* versus the educational video control) contrasts but failed the third test of mediation (i.e., change in mediator predicting change in outcome): cognitive dissonance, total caloric intake, the prudent pattern, and the Western food pattern. The finding that cognitive dissonance was most impacted by *Project Health* confirms our intention in developing this intervention. To our knowledge, no prior research in obesity prevention has specifically examined cognitive dissonance as a mediator of intervention

effectiveness. As noted, prior eating disorder prevention research found that participants assigned to high-dissonance compared to low-dissonance eating disorder prevention interventions showed significantly greater reductions in eating disorder symptoms (Green et al., 2005; McMillan et al., 2011). Given the failure to find full support of mediation for the core *Project Health* concept of cognitive dissonance in this report, future iterations of *Project Health* should increase the components that have been found to induce cognitive dissonance (e.g., public accountability, voluntary nature of commitment, no external justification, greater effort) for lifestyle behaviors linked to excess weight gain and perhaps include a greater focus on costs of engaging in eating disordered behaviors.

It was reassuring that *Project Health* had specific intervention effects on the Western pattern since dietary intake is a primary contributor to future weight gain. Previous weight loss programs for overweight or obese adults provide evidence that changes in dietary intake partially mediate the effects of an intervention on weight loss (Norman, Kolodziejczyk, Adams, Patrick, and Marshall, 2013; Partridge, McGeechan, Bauman, Phongsavan, and Allman-Farinelli, 2016).

It was also encouraging that all three indices of dietary intake (total caloric intake, the Western dietary pattern, and the prudent pattern) had significant effects in the condition contrast of the two active interventions (i.e., *Project Health* and *Healthy Weight*) versus the educational video control, though all three failed the next test of mediation (i.e., that change in the mediator would correlate with change in outcome). The Western dietary pattern focuses on processed and calorie-dense foods including high-fat dairy products and desserts, whereas the prudent pattern emphasizes the consumption of fruit, vegetables, whole grains, and lean meats/dairy products. Although dietary intake has generally been examined as a risk factor for obesity, previous research has found that overeating unhealthy foods is a risk factor for eating disorder onset (Stice et al., 2016). In addition, Stice et al. (2007) previously reported that, relative to women in an expressive writing control condition, *Healthy Weight* participants showed significantly greater improvements in healthy eating, as assessed by a short checklist, but failed to find a mediational effect.

We found no intervention effects for changes in the physical activity, suggesting that either these programs are not significantly improving physical activity or the self-report measure was inaccurate. Self-report measures are known to be problematic but the selected measure has shown convergent validity with physical activity records ($r = .28 - .86$; Ainsworth, Leon, Richardson, Jacobs, & Paffenbarger, 1993). Given that our previous work focused on objectively assessed physical activity (via waistband accelerometry) in an obesity prevention trial for college students also showed no significant effect of condition on aerobic-intensity activity (Arigo, Rohde, Shaw, & Stice, 2017), it is possible that such programs are not yet optimally designed for increasing physical activity. For example, a qualitative analysis of participant feedback from the present study noted an under-emphasis of increased physical activity relative to improved diet (Shaw, Rohde, & Stice, 2017). Other research, however, has found significant effects for physical activity both in weight prevention (Hollis et al., 2014) and treatment studies (Lubans et al., 2012), though both studies used objective measures of physical activity.

Study Strengths and Limitations

This study had several strengths, including long-term follow-up after a prevention program, a sophisticated statistical approach, and the inclusion of men in the sample (for whom relatively little information is available with respect to eating disorder symptoms). We also had an opportunity to examine two interventions that have currently or previously been found to produce significant reductions in both weight gain and eating disorder symptoms. Only a minority of obesity prevention programs have produced weight gain prevention effects that persist over long-term follow-up (e.g., Plotnikoff et al., 2015) and those that have are intensive (i.e., averaging 52 hours in duration; Stice, Shaw, & Marti, 2006). Few studies have examined mediation effects of obesity prevention, partially because main effects have been difficult to obtain. That concern is less applicable for eating disorder prevention, though the vast majority of prevention programs have not significantly reduced eating disorder symptoms.

This study also had several limitations. First, all of our mediators were self-reported, which may have hindered our ability to accurately test our mediational hypotheses. Second, it is possible that there was greater support for mediators of eating disorder symptoms than for objectively measured BMI because there is shared method and reporter bias with the former, but not the latter. Third, although we previously found no evidence suggesting that gender moderated intervention effects for either outcome, we conducted mixed-gender groups and had considerably fewer male compared to female participants. Thus, we could not examine whether the mediational effects were gender-specific.

Conclusions and Future Directions

In addition to the next steps indicated above, there are other variables that need to be examined in future research. For example, a review of mediating mechanisms of school-based obesity prevention for younger students (children and adolescents) found strong evidence for self-efficacy and moderate evidence of intention as mediators of increased physical activity, and moderate support for changes in attitude, knowledge, and the strength of healthy eating habits as mediators of improved dietary behaviors (van Stralen et al, 2011). In a comprehensive review of mediators of weight loss interventions for adults (Teixeira, 2015), variables that significantly mediated medium- or long-term weight control included higher autonomous motivation, self-efficacy/barriers, self-regulation skills (e.g., self-monitoring), flexible eating restraint, and positive body image. Measures of perceived self-efficacy (i.e., a person's belief that she or he can produce a desired effect) should be included in obesity, and presumably eating disorder, prevention programs (e.g., Partridge, McGeechan, Bauman, Phongsavan, & Allman-Farinelli, 2017). Ideally, objective measures of potential mediators, especially physical activity and dietary intake, should be used in future trials.

Mediational analyses are essential in identifying the mechanism of action for an intervention. Mediation research provides an opportunity to improve intervention effects by enhancing the effective program components and eliminating or modifying those not found to be effective. Because we did not detect any mediation effects for obesity, we can make no definitive recommendations regarding improved weight gain prevention. Presumably,

however, efforts to increase cognitive dissonance and reduce consumption of foods in the Western dietary pattern appear to have the strongest effects in obesity prevention efforts. Regarding increased cognitive dissonance induction, *Project Health* could be enhanced by adding discussions of costs associated with engaging in disordered eating behaviors. We are on firmer ground regarding recommendations for improving the eating disorder prevention effects of both interventions: interventions targeting improved body dissatisfaction and negative affect should be most productive; furthermore, the findings that both *Project Health* and *Healthy Weight* achieved significant change on all three measures of dietary intake suggest that dietary intake may warrant continued study in the etiology of eating pathology.

Several effects in the present study were small in magnitude and one recommendation for future research is the need to design and evaluate prevention programs that produce larger reductions in both the mediators and outcomes. Increasing the size of the main effects might make it easier to detect mediators and the fact that we only affected some mediators suggests that we need to produce larger effects on our mediators, such as changes in physical activity. Lastly, future research needs to measure the mediators more frequently to better test whether change in the mediator occurs before change in the outcome. The present analyses provide encouraging initial support, especially for several mediators of eating disorder symptom effects, but also suggest that we measure our mediators and outcomes more frequently and with more reliable and valid measures to provide optimally sensitive tests of the mechanisms of action for evidence-based prevention interventions.

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Highlights

- Evaluated mediators of two weight gain/eating disorder prevention programs
- No evidence of mediation for observed weight gain prevention effect was detected
- Body dissatisfaction and negative affect fully mediated eating disorder effects
- Future eating disorder prevention programs should emphasize these mechanisms

Figure 1.

Conditional growth model of BMI and ED controlling for condition.

Note. *Project Health* is dummy variables and *Intercept* and *Slope* refer to the random intercept of BMI or ED and the random slope of BMI or ED, respectively.

Figure 2.
Observed means in BMI across the assessments by condition.
Note. The gray rectangle corresponds to the intervention.

Figure 3.
Observed means in eating disorder symptoms across the assessments by condition.
Note. The gray rectangle corresponds to the intervention.

Table 1.

Adjusted means (controlling for baseline levels of the mediator) for change during the intervention for the potential mediators comparing *Project Health* (PH) against *Healthy Weight* (HW) and control participants.

Mediator	Adjusted Means	
	<i>PH</i>	<i>HW + controls</i>
Cognitive dissonance	0.182	0.061
Calorie intake	−0.129	−0.064
Prudent pattern	0.095	0.029
Western pattern	−0.144	−0.069
Physical activity	−1.866	−0.498
Body dissatisfaction	−0.241	−0.187
Negative affect	−0.220	−0.201

Note. Negative values indicate decreases during the intervention.

Table 2.

Correlations between contrasts and changes in potential mediators.

	PH	PH + HW	CD	CI	PP	WP	PA	BD
PH	1							
PH + HW	0.498	1						
CD	0.152	0.194	1					
CI	-0.111	-0.13	-0.079	1				
PP	0.0650	0.136	0.053	0.350	1			
WP	-0.155	-0.159	-0.084	0.817	0.162	1		
PA	-0.033	0.049	0.054	0.073	0.154	-0.008	1	
BD	-0.067	-0.165	0.083	0.038	-0.128	-0.006	0.019	1
NA	0.011	-0.091	0.004	0.156	-0.076	0.163	-0.051	0.071

Note. PH - the contrast of *Project Health* against *Healthy Weight* and Control; PH + HW - the contrast of *Project Health* and *Healthy Weight* against Control; CD – cognitive dissonance; CI – calorie intake; PP – prudent pattern; WP – western pattern; PA – physical activity; BD – body dissatisfaction; and NA – negative affect.

Table 3.

Adjusted means (controlling for baseline levels of the mediator) for change during the intervention for the potential mediators comparing *Project Height* (PH) and *Healthy Weight* (HW) against control participants and for PH, HW, and controls participants for the exploratory analysis.

Mediator	Adjusted Means		
	<i>PH + HW</i>	<i>Controls</i>	
Cognitive dissonance	0.150	0.002	
Calorie intake	−0.111	−0.033	
Prudent pattern	0.092	−0.033	
Western pattern	−0.123	−0.035	
Physical activity	0.375	−3.490	
Body dissatisfaction	−0.263	−0.090	
Negative affect	−0.264	−0.096	
	<i>HW</i>	<i>PH</i>	<i>Controls</i>
Eating symptoms	−4.328	−4.208	−3.152

Note. Negative values indicate decreases during the intervention.