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Review

Meta-analytic review of dissonance-based eating disorder prevention programs: Intervention, participant, and facilitator features that predict larger effects

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HIGHLIGHTS

- Conducted meta-analytic review of dissonance-based eating disorder prevention programs.
- We identified 56 trials evaluating 68 prevention programs (7808 participants).
- Average effect sizes (*d*) relative to minimal and alternative control were .39 and .20.
- Effects were larger given more activities/sessions, higher risk status, leader training/supervision.
- Future directions for dissonance-based prevention programs are provided.

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ABSTRACT

Many trials have provided support for dissonance-based eating disorder prevention programs. This meta-analytic review characterized the average intervention effects and tested whether various intervention, participant, and facilitator features correlated with larger effects to guide implementation of optimally effective versions of this program. We identified 56 trials that evaluated 68 dissonance-based eating disorder prevention programs (7808 participants). Average intervention effect sizes (*d*) relative to minimal intervention control conditions and credible alternative interventions (respectively) were 0.57 and 0.31 for thin-ideal internalization, 0.42 and 0.18 for body dissatisfaction, 0.37 and 0.17 for dieting, 0.29 and 0.21 for negative affect, and 0.31 and 0.13 for eating disorder symptoms. As hypothesized, effects were larger for interventions with more dissonance-inducing activities, more group sessions, and larger group sizes, as well as when delivered in-person versus on-line, sessions were recorded, participation was voluntary, body dissatisfaction was required, participants were mid-adolescents or adults (versus older adolescence), there were more ethnic minority participants, groups were led by clinicians versus researchers and at least two facilitators, and when facilitators received more training and supervision. Unexpectedly from a dissonance-induction perspective, effects were larger when participants were compensated. Results offer directions for maximizing the benefits of implementation efforts with dissonance-based eating disorder prevention programs, and may hold lessons for preventing other public health problems with dissonance-based interventions.

Representative data from the USA and Australia suggest that 13% of females experience a threshold or subthreshold DSM-IV eating disorder by young adulthood, which are characterized by chronicity, distress, functional impairment, and risk for future obesity, depression, suicide, and mortality (Arcelus, Mitchell, Wales, & Nielsen, 2011; Stice, Marti, & Rohde, 2013). Indeed, eating disorders show stronger relations to mortality, suicide attempts, and functional impairment than other psychiatric disorders, including schizophrenia, major depression, and bipolar disorder (Crow & Smiley, 2010) and are the second leading cause of mental health disability for adolescent girls and young women (Begg et al., 2007). Yet 80% of individuals with eating disorders do not receive treatment (Swanson, Crow, le Grange, Swendsen, & Merikangas, 2011) and extant treatments only result in lasting remission from binge eating and compensatory behaviors for 47% of patients on average (Hay, 2013). Thus, a public health priority is to develop and broadly implement effective eating disorder prevention programs. Prevention

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programs may be particularly effective for eating disorders because the peak risk period for onset occurs around the ages of 16 to 19 (Hudson, Hiripi, Pope, & Kessler, 2007; Stice, Marti, & Rohde, 2013), suggesting that if efficacious prevention programs were broadly implemented during adolescence, they could reduce the population prevalence of eating disorders.

1. Empirical support for dissonance-based eating disorder prevention programs

The fact that several malleable sociocultural, attitudinal, and behavioral risk factors for eating disorders have been identified suggests that it should be viable to prevent these disorders (e.g., pursuit of the thin beauty ideal, body dissatisfaction, overeating; Stice, Gau, Rohde, & Shaw, 2017). Dozens of eating disorder prevention programs have been developed and evaluated, but only three have reduced eating disorder symptom composite measures (Atkinson & Wade, 2016; Stice, Marti, Spoor, Presnell, & Shaw, 2008; Stice, Rohde, Shaw, & Marti, 2013) and only three have reduced future eating disorder onset (Martinsen et al., 2014; Stice, Marti, Spoor, et al., 2008, Stice, Rohde, Shaw, & Gau, 2017a,Stice, Rohde, Shaw, & Gau, 2017b; Stice, Rohde, Shaw, & Marti, 2013). The most empirical support has emerged for dissonance-based eating disorder prevention programs (commonly referred to as the *Body Project*), which has included efficacy trials, effectiveness trials, comparative trials, and tests of the intervention theory conducted by multiple research teams (reviewed subsequently). In the *Body Project* young women voluntarily critique the thin beauty ideal in verbal, written, and behavioral exercises, which theoretically generates dissonance that prompts reduced pursuit of this unrealistic ideal because people align their attitudes with their publically displayed behaviors (Stice, Mazotti, Weibel, & Agras, 2000). Festinger (1957) proposed people are motivated to maintain consistency between their behaviors and attitudes, and that when an individual engages in a behavior that is inconsistent with an attitude, they experience psychological discomfort that causes them to align their attitudes with their behavior. For instance, in the *Body Project* participants verbally generate costs associated with pursuing the thin ideal in response to Socratic questions, complete role-plays in which they talk facilitators out of pursuing this ideal, write a letter to a younger self on how to avoid body image concerns, and engage in acts of body activism that challenge this ideal (intervention script provided at: www.bodyprojectsupport.org).

According to the dual pathway model (Stice, Chase, Stormer, & Appel, 2001), reduced subscription to the thin ideal should decrease body dissatisfaction, unhealthy weight control behaviors, negative affect, eating disorder symptoms, and risk for future eating disorder onset. In support, elevated pursuit of the thin ideal, body dissatisfaction, dieting, and negative affect predict futures onset of any eating disorder (Ghaderi & Scott, 2001; Jacobi et al., 2011; McKnight, 2003; Rohde, Stice, & Marti, 2015; Santonastaso, Friederici, & Favaro, 1999). Research has also provided support for the thesis that pursuit of the thin ideal typically emerges before body dissatisfaction, which typically emerges before dieting and negative affect, which typically emerges before eating disorders, for those who eventually show onset of an eating disorder (Stice & Van Ryzin, 2019).

Efficacy trials have found that the *Body Project* produced greater reductions in eating disorder risk factors (thin-ideal internalization, body dissatisfaction, dieting, negative affect), eating disorder symptoms, and functional impairment in adolescent girls and young women with body image concerns relative to assessment-only control conditions, and often relative to alternative interventions, with several effects persisting through 3-year follow-up (Stice et al., 2000; Stice et al., 2001; Stice, Marti, Spoor, et al., 2008; Stice, Shaw, Burton, & Wade, 2006; Stice, Trost, & Chase, 2003). It is important to note that although the *Body Project* produced significantly greater reductions in outcomes than the *Healthy Weight* eating disorder prevention program from pretest to posttest, the latter program produced greater reductions in eating disorder risk factors and symptoms than control conditions over longer-term follow-up (Stice, Marti, Spoor, et al., 2008). That trial also found that future onset of threshold/subthreshold eating disorders over 3-year follow-up was 6% in *Body Project* participants and 6.5% in *Healthy Weight* participants, versus 15% in the assessment-only condition, representing a statistically significant 60% reduction in future ED onset for both programs. Efficacy trials conducted by independent teams also found that the *Body Project* produced greater reductions in risk factors and eating disorder symptoms versus assessment-only control conditions (Halliwell & Diedrichs, 2014; Matusek, Wendt, & Wiseman, 2004; Mitchell, Mazzeo, Rausch, & Cooke, 2007) and a media advocacy prevention program (Becker, Smith, & Ciao, 2005), though not all of these trials targeted young women with body image concerns. Further, the *Body Project* reduced eating disorder symptoms and electrocardiogram (ECG)-assessed cardiac risk indices among women with subclinical and clinical eating disorders (Green et al., 2016), which is important because cardiac problems are a leading cause of mortality among such individuals. Thus, the *Body Project* has shown efficacy when implemented universally to young women who were not screened for body image concerns, when implemented selectively to young women with body image concerns, and when implemented in an indicated fashion to young women with subclinical eating pathology.

Effectiveness trials have confirmed that the *Body Project* produces similar effects, relative to educational brochure and educational video control conditions, when high school and college counselors deliver the intervention under ecologically valid conditions, with several effects persisting through 3-year follow-up (Stice, Butryn, Rohde, Shaw, & Marti, 2013; Stice, Durant, Rohde, & Shaw, 2014; Stice, Stice, Rohde, Butryn, Shaw, & Marti, 2015; Stice, Rohde, Durant, & Shaw, 2012; Stice, Rohde, Gau, & Shaw, 2009; Stice, Rohde, Shaw, & Gau, 2011). However, this program did not reduce future eating disorder onset over 3-year follow-up in these effectiveness trials, potentially because the facilitators implemented the *Body Project* only a few times compared to facilitators in efficacy trials. Effectiveness trials also found that when undergraduate peer educators deliver the *Body Project* to other college students it produces larger reductions in risk factors and eating disorder symptoms than educational brochure and educational video controls (Stice, Rohde, Durant, Shaw, & Wade, 2013; Stice, Rohde, et al., 2017a,Stice, Rohde, et al., 2017b), the *Healthy Weight* prevention program (Becker et al., 2010), and the Internet-delivered *eBody Project* prevention program (Stice, Rohde, et al., 2017a,Stice, Rohde, et al., 2017b). However, other effectiveness trials did not find that it significantly outperformed the *Healthy Weight* prevention program or a media advocacy prevention program (Becker, Bull, Schaumberg, Cauble, & Franco, 2008; Becker, McDaniel, Bull, Powell, & McIntyre, 2012; Becker, Smith, & Ciao, 2006). Undergraduate-led *Body Project* groups also produced greater reductions in risk factors in high school students than assessment-only controls (Halliwell, Jarman, McNamara, Risdon, & Jankowski, 2015). An effectiveness trial that provided more intensive training and supervision found that young women assigned to peer-led *Body Project* groups showed a significant 74% reduction in eating disorder onset over 7-month follow-up relative to young women assigned to the *eBody Project* and a (nonsignificant) 64% reduction in eating disorder onset relative to an educational video control condition (2.2% versus 8.4% and 6.1% respectively; Stice, Rohde, et al., 2017a,Stice, Rohde, et al., 2017b). By 3-year follow-up, participants assigned to peer-led *Body Project* groups showed a statistically significant 56% reduction in eating disorder onset than participants assigned to the educational video control condition and a statistically significant 53% reduction in eating disorder onset than participants assigned to clinician-led *Body Project* groups (7% versus 16% and 15% respectively). The Internet-delivered *eBody Project* has likewise produced significantly greater reductions in risk factors and eating disorder symptoms than educational brochure and educational video controls, with some effects persisting through the final 2-year follow-up (Stice, Durant, et al., 2014; Stice, Rohde, Durant, & Shaw, 2012). Thus, at least

some effects persisted through 3-year follow-up in each of the four trials that collected data over that duration, and at least some effects persisted through 2-year follow-up in one trial that collected data over that duration.

In support of the intervention theory, reductions in thin-ideal internalization mediated the effects of the *Body Project* on reduction in eating disorder symptoms (Seidel, Presnell, & Rosenfield, 2009; Stice, Presnell, Gau, & Shaw, 2007). Consistent with the notion that dissonance induction contributes to intervention effects, completing versions of this intervention designed to maximize dissonance versus versions designed to minimize dissonance, but with the same general intervention content, produced greater symptom reductions (Green, Scott, Diyankova, Gasser, & Pederson, 2005; McMillan, Stice, & Rohde, 2011). Although those trials imply that both session content and non-specific factors contribute to intervention effects, participants who completed the Internet delivered *eBody Project,* which involves no interactions with facilitators or other participants, showed greater reductions in eating disorder risk factors and symptoms than educational video controls, suggesting that effects are not solely due to non-specific factors (Stice, Durant, et al., 2014; Stice, Rohde, et al., 2017a, 2017b; Stice, Rohde, Durant, & Shaw, 2012). And as noted, the *Body Project* has produced stronger reductions in outcomes than other group-based prevention programs, implying that effects are not solely due to non-specific factors inherent to group-based interventions. Further, the *Body Project* produced larger effects for participants with initial elevations in thin-ideal internalization, consistent with the thesis that they should experience the greatest dissonance (Stice, Marti, Shaw, & O'Neil, 2008). Completing the *Body Project* also offset the risk conveyed by the most potent eating disorder risk factor in one trial - denial of the costs of pursuing the thin ideal (Stice, Rohde, Gau, & Shaw, 2012): participants with this risk factor who completed the *Body Project* showed an eating disorder incidence of 0% over 3-year follow-up, versus 18% for those who completed two alternative interventions and 50% for assessment-only controls. Completing the *Body Project* also eliminated the adverse effects of exposure to thin models on preadolescent girls (Halliwell & Diedrichs, 2014). Further, participants who completed the *Body Project* showed a larger pre-to-post reduction in fMRI-assessed reward region (caudate) response to thin models compared to educational brochure controls, providing objective evidence that participants may not perceive the thin ideal as a desirable goal after completing the *Body Project* (Stice, Yokum, & Waters, 2015). The evidence that this intervention affects objective biological outcomes (neural response to thin models and cardiac functioning) is reassuring because these outcomes are immune to the demand characteristics inherent in randomized trials that can contribute to greater reductions in self-reported outcomes for participants in intervention versus control conditions.

The fact that the *Body Project* has produced effects that replicate across independent trials is noteworthy because many published findings do not replicate (Ioannidis, Munafò, Fusar-Poli, Nosek, & David, 2014). It is the only eating disorder prevention program that has produced effects that have been independently replicated. A recent meta-analytic review found that dissonance programs produced the largest reductions in eating disorder symptoms versus any other type of selective or indicated prevention program, with an average $d = 1.06$ when compared against assessment-only control conditions and an average $d = 0.30$ when compared against non-specific control conditions (Watson et al., 2016).

2. Meta-analytic review of dissonance-based eating disorder prevention program trials

A benefit of meta-analytic reviews is that they involve effect sizes based on observed changes across groups over time and report average effect sizes. The focus on a standardized effect size metric, such as change in standard deviation (*SD*) units (*d*), facilitates comparisons across studies that used different measures. Cohen (1988) defined $d = 0.20$, 0.50, and 0.80 as small, medium, and large effects, respectively. In contrast, narrative reviews focus on statistical significance, which is potentially problematic because sample size plays a major role in determining whether an effect is significant. In trials with small samples, larger effects may not be statistically significant, whereas in trials with very large samples, even trivial effects can be statistically significant. Thus, the first aim of this report is to characterize the average intervention effects of the *Body Project* on four primary eating disorder risk factors (thin-ideal internalization, body dissatisfaction, dieting, and negative affect) and on eating disorder symptoms.

A second benefit of meta-analytic reviews is that they permit an examination of moderators that correlate with effect size magnitude. This is key because there has been variation in effects, with some trials indicating that dissonance prevention programs produce large effects (e.g., Becker et al., 2010; Brown & Keel, 2015; Stice et al., 2006; Stice, Rohde, Durant, et al., 2013) and others suggesting that they produce small effects (e.g., Smith & Petrie, 2008; Wade, George, & Atkinson, 2009; Wiseman, Sunday, Bortolotti, & Halmi, 2004; Wolfe, 1992). Because it appeared that the trials that generated large effects differed systematically from those that generated small effects in terms of intervention, participant, and facilitator features, we tested hypotheses regarding factors that might correlate with larger effects. Thus, the second aim is to test whether certain intervention, participant, and facilitator features correlate with larger effects on eating disorder risk factors and symptoms. The fact that trials have evaluated various dissonance eating disorder prevention programs affords a unique opportunity to investigate finer details regarding the effects of these features. Improved understanding of features associated with larger effects should inform implementation of optimally effective versions of this prevention program, which is timely because organizations are increasingly implementing dissonance eating disorder prevention programs. Given pressures to deliver versions of this prevention program that are as short or inexpensive as possible, it is crucial to determine whether delivering versions of the intervention with fewer sessions, fewer dissonance-induction activities, using abbreviated training, omitting supervision, or working with undergraduate peer educators to deliver the intervention is associated with smaller effects. An understanding of features that correlate with larger effects may also inform implementation of prevention programs for other mental health and health problems.

3. Intervention features that may moderate intervention effect sizes

We investigated several variables hypothesized to moderate intervention effects based on theory, prior findings, and clinical experience. We focused on the following intervention features: (1) number of dissonance-inducing activities, (2) number of group sessions, (3) average number of group participants, (4) whether the intervention was delivered on-line or in-person, and (5) whether sessions were video-recorded. We hypothesized effects would be larger in trials in which participants engaged in more dissonance-inducing activities during or between sessions, as this should maximize dissonance-induction. We coded whether interventions included 12 dissonance-induction activities: (1) verbally agreeing to voluntarily participate in group discussions, (2) critiquing the thin ideal in a group discussion, (3) arguing facilitators out of pursuing the thin ideal in role-plays, (4) writing a letter to a younger girl about costs of the thin-ideal, (5) recording positive personal physical features while looking in a mirror, (6) writing a "rewind response" letter to someone in the past who led you to pursue the thin ideal; (7) engaging in body activism (e.g., like placing a love-your-body poster in the women's bathroom), (8) generating verbal "quick comebacks" to thin-ideal statements (9) engaging in a behavioral challenge to refute body image concerns, and (10) writing a letter to one's younger self about why she shouldn't pursue the thin-ideal; (11) discussing the benefits of the group; (12) committing to a self-

affirmation exercise (e.g., like keeping a journal of all of the positive things your body does). Theoretically, each of these activities generates dissonance regarding pursuit of the thin ideal, which should contribute to larger effects. Establishing a dose-response relation is a critical test of the intervention theory for a prevention program.

We hypothesized that interventions delivered over more sessions would produce larger effects because completing more home exercises should produce greater dissonance, based on the evidence that when participants put more effort into a goal, they show greater attitudinal shift towards valuing that goal (Cooper & Axsom, 1982). In addition, having time to reflect on the intervention content between sessions may aid in the consolidation of learning, which may contribute to larger effects. Further, it is also possible that meeting multiple times may contribute to a stronger development of group support, which *Body Project* participants state is one of the most valuable elements of this intervention (Shaw, Rohde, & Stice, 2016).

We hypothesized that intervention effects would be larger when the prevention program is delivered in larger groups. Theoretically, larger groups should maximize public accountability, which is key to dissonance-induction (Festinger & Carlsmith, 1959). Experiments have found that composing and giving a speech or writing an essay about a perspective produces increased endorsement of that perspective if participants think that the speech or essay will be heard or viewed by others, but produces little attitudinal change otherwise (Aronson, Fried, & Stone, 1991). The fact that there is typically no attitudinal change in these studies when participants believe that the speech or essay will not be heard or viewed by others illustrates that public accountability is critical for dissonance-induction. To our knowledge, no study has tested for a linear relation between group size and degree of dissonance-induced attitudinal change in dissonance-based group-delivered interventions.

In addition, we hypothesized intervention effects would be significantly larger if the dissonance eating disorder prevention programs was delivered in-person versus on-line. This is because in-person delivery should maximize public accountability, which has been shown to maximize dissonance induction and consequent attitudinal change (Festinger & Carlsmith, 1959).

Further, we hypothesized the intervention effects would be significantly larger when sessions were video-recorded. This is because recording sessions should increase perceived accountability, which should theoretically contribute to larger effects.

4. Participant features that may moderate intervention effect sizes

We focused on the following participant features: (1) whether participants were required to have body image concerns, (2) whether participants were mandated to complete the prevention program, (3) whether participants were compensated, (4) mean age of the sample, and (5) the percentage of the sample that was European-American. We tested the hypothesis that the effects of dissonance eating disorder prevention programs would be stronger for samples in which participants were required to have body image concerns versus samples without this requirement because meta-analytic reviews have found that effects were stronger for selective prevention programs that target samples at elevated risk for eating disorders versus universal prevention programs that target unselected populations (e.g., Stice, Shaw, & Marti, 2007). Body dissatisfaction is one of the most potent predictors of future escalation in symptoms and onset of eating disorders (e.g., Johnson & Wardle, 2005; Stice, Gau, et al., 2017). However, it is unclear whether this pattern of findings emerged because all prevention programs are more effective for populations at risk for eating disorders due to the fact that they are more motivated to engage in the intervention and have more room for improvement, or because the prevention programs that have been evaluated in selective trials are more effective than those evaluated in universal trials. As the present meta-analysis focused on a more homogenous set of prevention programs, it may better discern whether the risk status of participants is associated with larger effects, holding intervention content more constant. We hypothesize that dissonance-based eating disorder prevention programs will produce larger effects in trials that focused on participants with versus without body dissatisfaction because they have more motivation to engage in the intervention activities and more room for improvement in the outcomes.

We hypothesized that effects for the dissonance eating disorder prevention programs would be larger if participants volunteered to complete the intervention versus being mandated to do so. Many universities require that students complete interventions to prevent alcohol abuse and sexual victimization before they are permitted to register for classes, illustrating that some schools mandate participation in prevention programs. Further, some sororities have mandated that pledges complete dissonance-based eating disorder prevention programs (Becker & Stice, 2008). Research indicates that when participants *voluntarily* engage in a counter-attitudinal behavior (e.g., writing an essay that supports a perspective they do not initially endorse) they subsequently show attitudinal shift towards this new perspective, but that this does not occur if they are *required* to write the counter-attitudinal essay (Baumeister & Tice, 1984). Theoretically, engaging in a counter-attitudinal behavior prompts people to adjust their attitudes to conform to their behavior only if they feel that they engaged in that behavior voluntarily. It is important to note that in the studies in which individuals were mandated to complete a dissonance-based eating disorder prevention program the individuals were given the option of deciding not to provide data for the evaluation (i.e., participation in the research study was voluntary).

For similar theoretical reasons, we hypothesized that dissonance eating disorder prevention programs would produce weaker effects if individuals were compensated for participating in the study. Experiments indicate that when participants engage in a behavior that is inconsistent with their attitude, they only change that attitude if they have been paid a small amount of money rather than a large amount (Cohen, 1962), presumably because in the latter case they attribute their inconsistent behavior to the large payment. Participant compensation has ranged from a low of no financial or other type of compensation to payment of up to $165 USA dollars for completing seven assessments over a 4-year follow-up (i.e., participants were typically compensated for completing assessment, rather than the prevention program).

We hypothesized that intervention effects would be larger for trials with older versus younger participants because meta-analytic reviews have found that eating disorder prevention programs produced larger effects for older adolescents and young adults ~~versus younger adolescents~~ than for participants aged 9–14; Stice, Shaw, & Marti, 2007). Participants in trials of dissonance-based eating disorder prevention programs have ranged from 11 to 64, which includes trials that evaluated versions adapted for middle school girls (Halliwell & Diedrichs, 2014; Rohde et al., 2014). It is unclear whether this is because most eating disorder prevention programs are more effective if delivered immediately before or during the peak period of risk for eating disorder onset (ages 15–19; Stice, Rohde, Durant, et al., 2013) or because the interventions that have been evaluated with older participants are more effective than those evaluated with younger participants. Our meta-analysis should be able to differentiate between these two possibilities because the prevention programs included herein are more similar than those included in past meta-analytic reviews of eating disorder prevention trials. We hypothesized that intervention effects would be significantly smaller for early adolescents versus older adolescents and adults.

We investigated the effects of the ethnic composition of the sample because some data suggested that on average European-Americans subscribe more strongly to the thin beauty ideal than certain ethnic minority groups (Warren, Gleaves, Cepeda-Benito, Fernandez, & Rodriguez, 2005) and the basis of dissonance eating disorder

prevention programs is to critique this ideal. Thus, we tested the hypothesis that dissonance eating disorder prevention programs would produce larger effects in trials with higher percentages of European-Americans. However, a more recent study with a larger sample found that European-American young women did not report significantly higher endorsement of the thin beauty ideal than did Hispanic, Asian American, of African American young women (Cheng, Perko, Fuller-Marashi, Gau, & Stice, 2018).

5. Facilitator features that may moderate intervention effect sizes

We focused on the following facilitator features: (1) whether group facilitators were researchers, clinicians, or peer leaders, (2) the number of facilitators who led the groups, (3) the number of training hours provided to facilitators, and (4) whether facilitators received supervision. We hypothesized that intervention effects would be larger for groups facilitated by research clinicians (including the investigators and their graduate students) versus any type of clinician who naturally provide services to the population (e.g., to high school or college students) or undergraduate peer leaders based on a prior meta-analytic review (Stice, Shaw, & Marti, 2007). One trial found that effect sizes were larger for clinicians versus peer-leaders (Stice, Rohde, Durant, et al., 2013), though this did not occur after doubling the training time for peer-leaders versus clinicians (Stice, Rohde, et al., 2017a,Stice, Rohde, et al., 2017b).

We hypothesized that effects would be smaller for groups led by a single facilitator versus more facilitators. Theoretically, multiple facilitators are better positioned to manage the session activities and monitor whether participants are fully engaged in the discussions.

Finally, we hypothesized that effects would be larger when facilitators received more training and supervision. Greater initial training and supervision should maximize intervention fidelity and therapist competence, which should theoretically translate into larger effects. It should be noted that we had to focus on the amount of training and supervision, rather than intervention fidelity and therapist competence ratings, because the latter variables were not routinely reported.

6. Methods

6.1. Sample of studies

A database search to retrieve published articles was performed on *PsychInfo, MedLine, Dissertation Abstracts, and Cumulative Index to Nursing and Allied Health* for the years January 1980 – October 2018 using the following keywords: cognitive, dissonance, prevention, preventive, intervention, body dissatisfaction, body acceptance, and eating disorder. We also examined the reference sections of identified articles and reviews. With regard to unpublished studies, we asked established prevention researchers (e.g., Drs. Becker and Halliwell) if they had or knew of any results from unpublished trials. Further, we contacted investigators (e.g., Ms. Daníelsdóttir) who had requested the intervention script for the *Body Project* from Dr. Stice. We included randomized and non-randomized controlled trials that allowed estimation of between-condition effect sizes relative to either assessment-only/waitlist control conditions or alternative interventions. We also included studies that used a single-group repeated measures design without a comparison condition if it was possible to calculate adjusted within-condition effect sizes. We would have included studies written in other languages, but did not locate any published or unpublished studies in languages other than English.

6.2. Statistical methods

6.2.1. Effect size estimation

An effect size in the metric of mean difference in standard deviation (*SD*) units (equivalent to Cohen's *d*) was derived for each study. Effect sizes were computed using the R metafor package (Viechtbauer, 2010) which implemented recommendations from Morris (2008) for computing effect sizes for meta-analyses that combine studies from repeated measures, independent-groups, and independent-group repeated measures designs. Effect sizes were adjusted with a small sample bias-correction function (Hedges, 1982) because effects from small samples are more likely to be biased. Effect sizes and sampling variance estimates were computed in a raw-score *SD* metric, based on raw scores, as opposed to change scores. Sampling variances for effect sizes based on repeated measures require a pretest-posttest correlation that is typically unreported. Thus, for all repeated measures studies we used an estimate of the pretest-posttest correlation that was based on raw data from two studies conducted by the authors (Stice, Rohde et al., 2015). Studies with a single-group repeated measures design ($n = 10$; see Appendix Table 1) potentially overestimate effect size because the effect does not contain an adjustment for longitudinal change that would have occurred in the control group. We followed Becker (1988), in which single-group repeated measures effect sizes are adjusted by first conducting a meta-analysis using only control groups, then subtracting the average control group change from single-group repeated measures effect sizes. For studies with multiple follow-ups, we used only the assessment that most closely followed the intervention, because all trials collected data at posttest (only 5 of the 56 identified trials collected follow-up data over 2- to 3-year follow-up). Further, because intervention effects are typically strongest at posttest, focusing on pretest to posttest effects would afford the greatest sensitivity to detecting moderators that predict variation in effect sizes. When effect sizes could not be derived from the report, this information was requested from the authors and was obtained in all but one case. *SD*s were obtained from the standard error when only the latter was provided ($n = 5$; Appendix Table 1).

6.2.2. Selection of outcomes

Trials in this meta-analysis evaluated interventions designed to reduce eating disorder risk factors and symptoms. We identified five outcomes commonly reported in these trials: eating disorder symptoms and four risk factors (thin-ideal internalization, body dissatisfaction, dieting, negative affect), the latter four variables have been found to predict future onset of eating disorders in multiple independent prospective studies (e.g., Allen, Byrne, Oddy, & Crosby, 2013; Stice, Gau, et al., 2017), but are not diagnostic symptoms of eating disorders per DSM-IV or V. Effect sizes were reverse coded if a decrease in the outcome indicated improvement so that positive effect sizes reflect improvement in the outcome.

6.2.3. Intervention group contrasts

Effect sizes were computed for dissonance prevention programs versus assessment-only/educational brochure control conditions and for dissonance interventions versus credible alternative comparison interventions, which included group interventions and educational video conditions. This is because effect sizes are typically larger when an intervention is compared to a minimal intervention control condition versus when compared to a credible alternative comparison intervention, presumably because expectancies, demand characteristics, and non-specific effects inherent to any credible intervention contribute to greater reductions in the outcomes. Assessment-only and educational brochure control conditions were both coded as minimal-intervention control conditions for the analyses, as both tended to be associated with similar change in outcomes. Trials with neither of the above comparison conditions were treated as within-group repeated measures studies and were combined with effect sizes from trials that used minimum intervention control conditions because the effect sizes from the uncontrolled trials were adjusted for the typical reduction in minimal intervention control conditions (see above). We compared effect sizes for the dissonance interventions relative to each of these two classes of comparison conditions to assess the impact of type of comparison

Table 1
Operationalization and descriptive statistics for moderators.

Moderator	Coding description and criteria	Descriptive statistics
Intervention features		
Number of dissonance inducing activities	Continuous variable reflecting the number of dissonance-inducing activities completed.	*M* = 8.74, *SD* = 3.54 *Range* = (0, 12)
Number of intervention sessions	Continuous variable reflecting the total number of sessions.	*M* = 3.31, *SD* = 1.71 *Range* = (1, 12)
Average group size	Continuous variable reflecting the number of participants in intervention groups.	*M* = 9.75, *SD* = 6.15 *Range* = (1, 31)
Online intervention	Dichotomous variable reflecting whether intervention was delivered online 1 = *Yes*; 0 = *No*	*Yes* = 5; *No* = 63
Sessions video or audio recorded	Dichotomous variable reflecting whether sessions were video-recorded 1 = *Yes*; 0 = *No*	*Yes* = 29; *No* = 39
Participants compensated financially or otherwise (e.g., course credit) for study participation	Dichotomous variable reflecting whether participants were compensated. 1 = *Yes*; 0 = *No*	*Yes* = 38; *No* = 29
Participant Features		
Body image concerns required	Dichotomous variable reflecting whether participants were required to have body image concerns 1 = *Yes*; 0 = *No*	*Yes* = 43; *No* = 25
Mandatory participation	Dichotomous variable reflecting whether participation was mandatory 1 = Yes; 0 = No	Yes = 10; No = 58
Mean age of sample	Continuous variable reflecting mean age of participants at baseline	*M* = 19.60, *SD* = 4.10 Range = (12.07, 43.20)
Percentage of European-American participants	Continuous variable reflecting percentage of the sample that was European-American.	*M* = 74.26, *SD* = 16.64 Range = (33, 100)
Facilitator features		
Facilitator type	Categorical variable reflecting whether facilitator was a researcher, clinician, or peer	Researcher = 27; Clinician = 15; Peer = 20
Number of facilitators	Continuous variable reflecting the number of facilitators who led groups	*M* = 1.90, *SD* = 0.85 *Range* = (0, 4)
Number of training hours	Continuous variable reflecting the number of training hours	*M* = 6.00, *SD* = 4.21 *Range* = (0, 16)
Facilitators supervised	Dichotomous variable reflecting whether facilitators were supervised 1 = *Yes*; 0 = *No*	*Yes* = 43; *No* = 18

condition on effect sizes in preliminary analyses. Because results confirmed that dissonance prevention programs produced larger effect when compared to minimal intervention control conditions versus when compared to credible alternative comparison conditions we examined moderators separately for these two comparison conditions.

6.2.4. Operationalization and coding of effect size moderators

Table 1 lists the operational definition of each moderator. The reliability of moderator coding was assessed by examining agreement between the third and fourth authors. Cohen's κ were computed for categorical moderators and intraclass correlations (ICC) were computed for continuous moderators. In a randomly selected subset of 10 studies included in this meta-analytic review, the mean κ = 0.84 and the mean ICC = 0.95 for the moderators. Thus, inter-rater reliability was high for moderator coding.

6.2.5. Moderator models

We investigated moderators in a mixed-effects regression model in which effect size heterogeneity is represented through an additional error term that allows for variance in the true effect size and study characteristics were treated as fixed effect moderators. We implemented multilevel meta-analysis models to analyze moderators of effect sizes using the meta3 function from the metaSEM R package (Cheung, 2015b). Multilevel meta-analysis is an application of variance-known multilevel models in which effect sizes are nested in higher-level units, such as papers, and the variance of the effect sizes is treated as known based on standard formulas for effect sizes variance, but estimation for all other model parameters is conducted in a multilevel model framework (Hox, 2010), which is ideal for situations in which there are multiple effect sizes within a study (Maas, Hox, & Lensvelt-Mulders, 2004). Many studies had more than two conditions that could be used to compute effect sizes, resulting in multiple effect sizes per study. Prior to examining moderators, effect size heterogeneity was estimated using the *Q* statistic (Cochran, 1954), which tests the null hypotheses that effect size variance is zero and can be applied to both the mixed-effects and multilevel models (Cheung, 2015a).

We fit a multilevel regression model for each moderator. In moderator models in which random effects were not estimable (i.e., near zero values) or optimization did not succeed, the models were refit as mixed-effect models using the meta function from the metaSEM R package (Cheung, 2015b). Facilitator type contained three levels (clinician, researcher, peer) and we thus dummy-coded for researcher and peer, using clinicians as the reference group. Continuous variables were standardized in a *z* score format prior to fitting models to facilitate interpretation of the coefficients which effectively placed continuous moderators on the same scale (i.e., *M* = 0, *SD* = 1). For continuous moderators we tested for both linear and quadratic effects; in the event of a significant quadratic effect, we report the linear and quadratic terms. For models that did not exhibit a significant quadratic effect, we report only the linear model. Most quadratic models exhibited an inverted-U (i.e., effect sizes increased to an inflection point and then began to decrease). We computed fitted values for 2 SDs above and below the means to determine the point where the maximum effect size was achieved. In the few instances in which a U-shaped quadratic effect was observed, we note this pattern and report the value of the moderator with the smallest effect. When we observed significant moderator effects, we probed the effects using model-based marginal means (Bauer & Curran, 2005). For categorical moderators, we estimated marginal effects for each level of the moderator; for continuous, mean-centered moderators, we estimated marginal effects at 1 *SD* above and below the mean.

7. Results

The literature search identified 59 published or unpublished reports from which we extracted data for 71 dissonance eating disorder prevention programs. Appendix Table 1 provides an overview of each study and the results. We excluded Wiseman et al. (2004) because we

Table 2
Effect sizes (d) for dissonance-based eating disorder prevention programs versus controls and uncontrolled repeated measures designs.

Study	Thin-Ideal Internalization	Body Dissatisfaction	Dieting	Negative Affect	Eating Disorder Symptoms
Dissonance-based programs versus controls and uncontrolled repeated measures designs					
Amaral, Stice, and Ferreira (2018)	1.95	1.00	1.09	0.68	0.30
Atkinson & Wade (2015) (dissonance v. classes as usual)	−0.01	0.18	0.24	0.19	0.44
Atkinson and Wade (2016) (dissonance v. control)	0.50	0.38	0.60	0.34	0.72
Becker et al. (2005) (dissonance v. waitlist)	0.26	0.33	0.21	–	0.33
Brown, et al. (2017)	0.47	–	–	–	0.31
Brown and Keel (2015) (dissonance v. waitlist)	0.73	0.77	0.58	–	0.55
Casasnovas et al. (2018)	0.16	0.19	−0.14	0.24	−0.09
Chithambo & Huey (2016) (dissonance v. no intervention)	0.94	0.24	0.29	0.46	0.15
Ciao et al. (2015) (peer-led dissonance v. waitlist)	0.96	0.47	0.22	–	0.43
Corning, et al. (2010) (intervention v. control)	0.16	0.24	–	–	–
Cruwys, Haslam, Fox, & McMahon (2015) (no comparison group)	1.55	0.71	1.49	–	–
Danielsdottir et al., (2012) (study 1) (body project intervention v. waitlist control)	0.76	0.19	0.23	–	0.39
Danielsdottir et al., (2012) (study 2) (no comparison group)	0.37	0.82	0.10	–	0.19
Green et al., (2016) (dissonance v. control)	–	–	–	–	1.02
Green, Kroska, and Herrick (2018)	0.21	0.40	–	0.20	0.64
Green et al. (2005) (high dissonance) (high level dissonance v. control)	–	–	–	–	0.13
Green et al. (2005) (low dissonance) (low level dissonance v. control)	–	–	–	–	−0.25
Green et al. (2017)	0.59	0.92	–	–	1.02
Greif, Becker, & Hildebrandt (2015) (no comparison group)	0.91	0.23	–	0.17	0.39
Grossman, et al. (2018)	0.75	0.20	–	–	–
Halliwell & Diedrichs (2013) (intervention v. control)	0.49	0.40	0.14	–	–
Halliwell et al. (2015) (dissonance v. control)	0.12	0.32	–	–	–
Jilka (2002) (cognitive dissonance v. no treatment)	1.03	0.48	0.14	–	0.26
Jilka (2002) (cognitive dissonance v. psychoeducational control)	1.31	0.68	−0.01	–	0.44
Kilpela et al. (2016) (female-only) (Female only dissonance v. Waitlist)	0.12	0.81	–	0.36	0.56
Kilpela et al. (2016) (mixed sex) (Mixed sex dissonance v. Waitlist)	0.47	0.56	–	0.28	0.60
Kilpela et al. (2015) (2-session dissonance) (no comparison group)	0.55	0.48	0.36	0.25	0.22
Kilpela et al. (2015) (4-session dissonance) (no comparison group)	0.24	0.07	0.14	0.11	0.17
Kilpela et al., 2014 (no comparison group)	0.81	0.24	–	0.25	0.19
Krishna (2011, Honors Thesis) (dissonance v. control)	0.87	–	–	–	0.72
Machin (2008, Dissertation) (cognitive dissonance v. control)	0.31	0.17	−0.04	–	0.83
Matusek et al. (2004) (dissonance v. control)	0.69	0.18	–	0.25	0.43
McMillan et al. (2011) (high dissonance) (high dissonance v. control)	1.21	0.64	0.70	0.29	0.47
McMillan et al. (2011) (low dissonance) (low dissonance v. control)	0.87	0.67	0.55	0.17	0.10
Mitchell et al. (2007) (dissonance v. control)	0.15	0.33	−0.01	0.43	0.72
Pennesi and Wade (2017) (dissonance v. control)	–	–	–	−0.29	0.33
Perez, Becker, and Ramirez (2010) (no comparison group)	0.53	0.30	0.15	–	0.13
Presnell, Seidel, and Madeley (2008) (dissonance only v. assessment only control) Ramirez, Perez, and Taylor (2012)	0.18	0.34	0.45	0.38	0.07
(Prevention Group v. Comparison Group)	0.25	0.60	–	0.03	0.14
Roehrig, Thompson, Brannick, and van den Berg (2006) (no comparison group)	0.46	0.33	0.54	0.80	0.63
Rohde et al. (2014) (study 1) (Body Project v. Control)	0.32	0.81	0.44	0.15	0.22
Rohde et al. (2014) (study 2) (Body Project v. Control)	0.23	0.36	0.59	0.55	0.84
Serdar, 2006 Group dissonance (Group dissonance v. Control)	0.11	0.08	0.23	0.26	0.26
Serdar, 2006 Internet dissonance (Internet dissonance v. Control)	0.03	0.17	0.06	0.14	0.16
Smith and Petrie (2008) (cognitive dissonance v. control)	0.47	−0.25	–	0.30	0.20
Stice, Butryn, Rohde, et al. (2013) (Dissonance v. Control)	0.74	0.61	0.50	0.41	0.38
Stice et al. (2006) (dissonance v. assessment only)	0.87	0.61	0.43	0.39	0.30
Stice et al. (2009) (dissonance v. Brochure controls)	0.50	0.43	0.30	0.21	0.30
Stice et al. (2000) (dissonance v. waitlist)	0.49	0.46	0.22	0.76	0.50
Stice, Durant, et al. (2014) (Body Project) (Body Project v. Brochure controls)	0.75	0.87	0.50	0.42	0.49
Stice, Durant, et al. (2014) (eBody) (eBody Project v. Brochure controls)	0.72	0.84	0.60	0.61	0.39
Stice, Rohde, Durant, et al. (2013) (clinician-led groups Study 1) (Clinician-led intervention v. Brochure controls)	1.06	0.90	0.77	0.57	0.52
Stice, Rohde, Durant, et al. (2013) (peer-led groups Study 1) (Peer-led intervention v. Brochure controls)	0.81	0.61	0.43	0.26	0.33
Stice, Rohde, Durant, et al. (2013) (peer-led groups Study 2) (Peer-led intervention v. Delayed controls)	1.06	0.85	0.86	0.64	0.38
Stice et al. (2003) (dissonance v. waitlist)	0.52	0.34	0.27	−0.23	0.31
Trost (2006) (HIP intervention v. waitlist)	0.24	0.15	0.12	0.14	0.16
Van Diest & Perez, 2013 (no comparison group)	0.73	0.37	–	–	0.25
Wade et al. (2009) (cognitive dissonance v. control)	–	0.37	–	–	–
Wolfe (1992, Dissertation) (high dissonance v. minimal dissonance)	−0.12	−0.04	–	–	–
Dissonance-based programs versus alternative interventions					
Atkinson and Wade (2016) (dissonance v. mindfulness)	−0.08	0.14	0.07	0.00	0.31
Atkinson and Wade (2016) (dissonance v. mindfulness)	−0.95	−0.58	−0.11	−0.45	−0.11
Becker et al. (2008) (dissonance v. media advocacy)	0.22	0.02	0.13	–	0.11
Becker et al. (2012) (athlete-modified dissonance v. athlete-modified healthy weight)	0.18	0.17	0.15	0.22	0.12

(*continued on next page*)

Table 2 (*continued*)

Study	Thin-Ideal Internalization	Body Dissatisfaction	Dieting	Negative Affect	Eating Disorder Symptoms
Becker et al. (2006) (dissonance v. media psychoeducation)	0.16	0.11	0.13	–	0.18
Becker et al. (2006) (dissonance v. media advocacy)	0.40	−0.07	0.11	–	0.01
Becker et al. (2010) (dissonance v. modified healthy weight)	0.44	0.25	0.18	0.32	0.33
Chithambo and Huey (2017) (internet dissonance v. internet CB)	0.37	−0.20	−0.19	0.14	−0.31
Krysanski (2005, Dissertation) (dissonance v. psychoeducation control)	0.28	−0.20	0.04	−0.07	−0.11
Linville et al. (2015) (dissonance v. video control)	0.52	0.24	0.43	0.45	0.34
Machin (2008, Dissertation) (cognitive dissonance v. healthy weight)	0.47	0.42	−0.09	–	−0.56
Matusek et al. (2004) (dissonance v. healthy weight)	0.16	−0.02	–	0.29	0.12
Mitchell et al. (2007) (dissonance v. yoga)	0.01	0.40	−0.13	0.14	0.24
Pennesi and Wade (2017) (dissonance v. imagery rescripting)	–	–	–	0.02	−0.70
Smith and Petrie (2008) (cognitive dissonance v. healthy weight)	0.54	0.58	–	0.51	0.27
Stice et al. (2001) (dissonance v. early version of the healthy weight)	0.62	0.31	0.16	0.09	0.04
Stice et al., (2006) (dissonance v. expressive writing)	0.73	0.71	0.42	0.40	0.41
Stice et al., (2006) (dissonance v. healthy weight)	0.39	0.31	0.38	0.19	0.16
Stice, Durant, et al. (2014) (Body Project) (Body Project v. Video controls)	0.54	0.50	0.35	0.34	0.35
Stice, Durant, et al. (2014) (eBody) (eBody Project v. Video controls)	0.52	0.37	0.40	0.43	0.14
Stice, Rohde, et al. (2017a),Stice, Rohde, et al., 2017b), (eBody Project v. Video controls)	0.41	0.27	0.32	0.20	0.29
Stice, Rohde, et al. (2017a),Stice, Rohde, et al., 2017b), Peer (Body Project v. Video controls)	0.67	0.64	0.49	0.34	0.28
Stice, Rohde, et al. (2017a),Stice, Rohde, et al., 2017b), Clinician (Body Project v. Video controls)	0.74	0.60	0.57	0.37	0.43
Stice et al. (2003) (dissonance v. healthy weight)	0.03	0.10	0.16	−0.09	−0.14
Wade et al. (2009) (cognitive dissonance v. acceptance)	–	−0.01	–	–	–
Wade et al. (2009) (cognitive dissonance v. distraction)	–	−0.21	–	–	–
Wade et al. (2009) (cognitive dissonance v. ruminative attention control)	–	0.30	–	–	–

could not obtain means and *SD*s. We excluded Magnuson (2012), which evaluated a single intervention that contained dissonance-based activities as well as cognitive behavioral and media literacy activities, because it was impossible to determine whether it was the dissonance-induction content that produced intervention effects. We excluded Ciao (2012), study 1 due to its small sample size ($n = 9$); Study 2 from Ciao, Latner, Brown, Ebneter, and Becker (2015) was included. This resulted in a total of 56 papers that evaluated 68 dissonance eating disorder prevention programs (total $N = 7808$ participants) that were included. There was an average of 1.26 unique condition contrasts per dissonance programs, resulting in 86 possible effect sizes per outcome. Observed effect sizes for dissonance interventions versus control conditions and versus alternative interventions are reported in the upper and lower portions of Table 2, respectively.

7.1. Average effect size and effect size heterogeneity

Average effect sizes were significantly different from zero for all five examined outcomes: thin-ideal internalization ($d = 0.50$, $z = 9.20$, $p < .001$), body dissatisfaction ($d = 0.36$, $z = 9.63$, $p < .001$), dieting ($d = 0.31$, $z = 5.97$, $p < .001$), negative affect ($d = 0.25$, $z = 8.46$, $p < .001$), and eating disorder symptoms ($d = 0.26$, $z = 9.26$, $p < .001$). There was significant heterogeneity in effect sizes for thin-ideal internalization (Q [76] = 358.51, $p < .001$), body dissatisfaction (Q [78] = 231.84, $p < .001$), dieting (Q [58] = 272.08, $p < .001$), negative affect (Q [55] = 89.53, $p = .002$), and eating disorder symptoms (Q [75] = 133.16, $p < .001$).

The magnitude of effect sizes for dissonance interventions versus control conditions and pre-post only studies was larger than those for dissonance interventions versus alternative interventions for thin-ideal internalization ($d = 0.28$, $z = 3.66$, $p < .001$), body dissatisfaction ($d = 0.23$, $z = 3.69$, $p < .001$), dieting ($d = 0.19$, $z = 3.03$, $p = .002$), and eating disorder symptoms ($d = 0.18$, $z = 3.05$, $p = .002$) but not for negative affect ($d = 0.08$, $z = 1.55$, $p = .121$). Thus, we conducted separate analyses for the average effect sizes and moderators of effect size magnitude for dissonance prevention programs versus control conditions and for dissonance prevention programs versus alternative interventions.

Average dissonance eating disorder prevention programs versus control condition effect sizes were significantly different from zero for all outcomes: thin-ideal internalization ($d = 0.57$, $z = 9.83$, $p < .001$), body dissatisfaction ($d = 0.42$, $z = 10.78$, $p < .001$), dieting ($d = 0.37$, $z = 5.88$, $p < .001$), negative affect ($d = 0.29$, $z = 8.65$, $p < .001$), and eating disorder symptoms ($d = 0.31$, $z = 9.89$, $p < .001$). Similarly, average dissonance eating disorder prevention programs versus alternative intervention condition effect sizes were significantly different from zero, though smaller in magnitude, for thin-ideal internalization ($d = 0.31$, $z = 4.48$, $p < .001$), body dissatisfaction ($d = 0.18$, $z = 2.90$, $p = .004$), dieting ($d = 0.17$, $z = 3.12$, $p = .002$), negative affect ($d = 0.21$, $z = 5.35$, $p < .001$), and eating disorder symptoms ($d = 0.13$, $z = 2.31$, $p = .021$).

7.2. Moderators of eating disorder prevention program effects

7.2.1. Thin-ideal internalization

Table 3 reports moderators of effects for dissonance prevention programs versus control conditions. Three moderators were significant. Number of facilitators was a significant predictor of effect sizes, wherein effect sizes were larger for interventions with more facilitators ($z = 2.04$, $p = .041$); simple slope estimates of effect sizes were $d = 0.46$ at 1 *SD* below the mean (1.0) and $d = 0.73$ at 1 *SD* above the mean (2.8). Number of training hours exhibited a non-significant positive linear effect ($z = 1.06$, $p = .288$) and a significant negative quadratic effect ($z = -2.55$, $p = .011$); in the inverted U function, effects sizes peaked at 7.3 h of training. Effect sizes were significantly larger when facilitators were supervised ($d = 0.68$) than when they were not ($d = 0.37$) ($z = 2.52$, $p = .012$).

Among dissonance intervention versus alternative intervention contrasts, three moderators were significant (Table 4). Group size exhibited a non-significant linear effect ($z = 0.27$, $p = .790$) and a significant negative quadratic effect ($z = -2.05$, $p = .040$); effects sizes peaked at group sizes of 10.4. Effect sizes were significantly larger when participants were compensated financially or otherwise ($d = 0.51$) than when they were not ($d = 0.12$) ($z = 3.86$, $p < .001$). Effect sizes were significantly larger when facilitators were supervised ($d = 0.39$) than when they were not ($d = 0.01$) ($z = 2.44$, $p = .015$).

Table 3
Moderator models for effect sizes from dissonance-based eating disorder prevention programs versus control conditions.

Moderator	Thin-ideal internalization	Body dissatisfaction	Dieting	Negative affect	Eating disorder symptoms
Unconditional means model	0.57***	0.42***	0.37***	0.29***	0.31***
Number of dissonance-inducing activities	0.06	0.12***	0.13	0.08*	0.05
Number of intervention sessions					
Linear	−0.09	0.05	−0.01	0.13*	0.07
Quadratic		−0.03*		−0.06*	
Average group size					
Linear	−0.02	0.00	−0.09	0.00	−0.02
Quadratic				−0.12*	
Delivered online	−0.13	−0.03	−0.12	0.02	−0.05
Sessions video-recorded	0.10	0.12	0.18	0.10	0.03
Body image concerns required	0.22	0.16*	0.25	0.13*	0.13*
Mandatory participation	−0.11	−0.21*	−0.33	−0.09	−0.14*
Compensated financially or otherwise	0.01	0.13	0.09	0.03	0.07
Mean age of sample	−0.01	−0.02	−0.01	0.00	0.03
Percentage of European-American participants					
Linear	−0.06	−0.05	−0.13*	−0.06*	−0.08**
Quadratic			−0.09*		−0.06**
Type of facilitator (ref = clinician)					
Peer	0.07	−0.10	−0.05	−0.09	−0.01
Researcher	−0.06	−0.14	−0.03	−0.02	0.12
Number of facilitators	0.14*	0.06	0.04	0.04	0.03
Number of training hours					
Linear	0.06	0.04	−0.02	0.00	−0.01
Quadratic	−0.11*				
Facilitators supervised	0.31*	0.21*	0.22	0.10	0.07

7.2.2. Body dissatisfaction

Among dissonance intervention versus control condition contrasts, five moderators were significant predictors of effect size. Number of dissonance-inducing activities was a significant predictor of effect sizes, wherein effects were larger for studies with a greater number of dissonance-inducing activities ($z = 3.37$, $p < .001$); effect sizes were $d = 0.30$ at 1 *SD* below the mean (5.2) and $d = 0.54$ at 1 *SD* above the mean (12.3). It should be noted that sometimes the value of the moderator at 1 *SD* above or below the mean was slightly outside the observed range of the moderator (extrapolation), which can occur if the moderator has a skewed distribution (Hays, 1994). Intervention duration exhibited a non-significant positive linear effect ($z = 0.98$, $p = .328$) and a significant negative quadratic effect ($z = -2.11$, $p = .035$); effects sizes peaked at 4.9 intervention sessions. Effect sizes were significantly larger when body image concerns were required ($d = 0.50$) than when they were not ($d = 0.34$) ($z = 2.25$, $p = .025$).

Table 4
Moderator models for effect sizes from dissonance-based eating disorder prevention programs versus alternative interventions.

Moderator	Thin-Ideal Internalization	Body Dissatisfaction	Dieting	Negative Affect	Eating Disorder Symptoms
Unconditional means model	0.31***	0.18**	0.17**	0.21***	0.13*
Number of dissonance-inducing activities	0.13	0.14*	0.24***	0.07	0.19***
Number of intervention sessions					
Linear	0.09	0.17*	0.00	0.08	0.07
Quadratic					−0.17*
Average group size					
Linear	0.04	−0.09	−0.11	0.00	0.25**
Quadratic	−0.36*			−0.16*	−0.21*
Delivered online	−0.11	−0.26*	−0.08	−0.01	−0.10
Sessions video-recorded	0.20	0.24*	0.28**	0.16*	0.23
Body image concerns required	0.21	0.16	0.07	0.07	−0.08
Mandatory participation	−0.15	−0.12	−0.06	−0.12	0.09
Compensated financially or otherwise	0.39***	0.29**	0.27**	0.16	−0.02
Mean age of sample					
Linear	0.18	0.02	0.06	0.06	−0.00
Quadratic			0.29*		0.39**
Percentage of European-American participants					
Linear	−0.09	−0.12	−0.06	−0.01	0.10
Quadratic		−0.12**	−0.10*		
Type of facilitator (ref = clinician)					
Peer	−0.11	−0.09	−0.25	−0.07	−0.18
Researcher	−0.28	−0.13	−0.32**	−0.25*	−0.21*
Number of facilitators					
Linear	0.01	0.05	0.08*	0.06	0.12***
Quadratic		−0.09**	−0.08***		−0.08***
Number of training hours					
Linear	0.09	−0.10	−0.07	0.08	−0.10
Quadratic		−0.42**	−0.35***		−0.40***
Facilitators supervised	0.38*	0.37*	0.32**	0.23**	0.26

Effect sizes were significantly larger for studies with voluntary participation ($d = 0.47$) versus mandatory participation ($d = 0.26$) ($z = -2.42$, $p = .015$). Effect sizes were significantly larger when facilitators were supervised ($d = 0.48$) than when they were not ($d = 0.28$) ($z = 2.47$, $p = .013$).

Among dissonance intervention versus alternative intervention contrasts, nine moderators were significant. Number of dissonance-inducing activities exhibited a significant positive linear effect ($z = 2.50$, $p = .012$); effect sizes were $d = 0.06$ at 1 *SD* below the mean (5.2) and $d = 0.34$ at 1 *SD* above the mean (12.3). Total intervention duration was a significant predictor of effect sizes, wherein effects were larger for studies that evaluated interventions of longer durations ($z = 2.03$, $p = .042$); effect sizes were $d = 0.05$ at 1 *SD* below the mean (1.6) and $d = 0.38$ at 1 *SD* above the mean (5.0). Effect sizes were significantly smaller for online interventions ($d = -0.06$) than in-person interventions ($d = 0.21$) ($z = -2.15$, $p = .031$). Effect sizes were significantly larger when sessions were video-recorded ($d = 0.39$) than when they were not ($d = 0.14$) ($z = 2.06$, $p = .039$). Effect sizes were significantly larger when participants were compensated ($d = 0.38$) than when they were not ($d = 0.09$) ($z = 3.07$, $p = .002$). Percentage of European-American participants exhibited a non-significant negative linear effect ($z = -1.85$, $p = .065$) and a significant negative quadratic effect ($z = -2.82$, $p = .005$); effects sizes were largest at 65.9% European-American. Number of facilitators exhibited a non-significant positive linear effect ($z = 1.10$, $p = .271$) and a significant negative quadratic effect ($z = -3.01$, $p = .003$); effects sizes were largest at 2.2 facilitators. Number of training hours exhibited a non-significant negative linear effect ($z = -0.98$ $p = .333$) and a significant negative quadratic effect ($z = -2.85$, $p = .004$); effects sizes peaked at 5.6 h of training. Effect sizes were significantly larger when facilitators were supervised ($d = 0.27$) than when they were not ($d = -0.10$) ($z = 2.46$, $p = .014$).

7.2.3. Dieting

Among dissonance intervention versus control condition contrasts, only percentage of European-American participants was a significant predictor of effect size. The percentage of European-American participants exhibited a significant negative linear effect ($z = -2.40$, $p = .016$) and a significant negative quadratic effect ($z = -2.27$, $p = .023$); effects sizes were largest at 62.6% European-American.

Among dissonance intervention versus alternative intervention contrasts, eight moderators were significant. Number of dissonance-inducing activities was a significant predictor of effect sizes, wherein effects were larger for studies with a greater number of dissonance-inducing activities ($z = 3.71$, $p < .001$); effect sizes were $d = -0.10$ at 1 *SD* below the mean (5.2) and $d = 0.37$ at 1 *SD* above the mean (12.3). Effect sizes were significantly larger when sessions were video-recorded ($d = 0.44$) than when they were not ($d = 0.16$) ($z = 2.99$, $p = .003$). Effect sizes were significantly larger when participants were compensated ($d = 0.37$) than when they were not ($d = 0.10$) ($z = 3.28$, $p = .001$). Mean age of sample exhibited a non-significant positive linear effect ($z = 0.62$, $p = .533$) and a significant positive quadratic effect ($z = 2.01$, $p = .045$) that resulted in a U-shaped curve with the minimal effect size at age 19.2. The percentage of European-American participants exhibited a non-significant negative linear effect ($z = -0.82$, $p = .410$) and a significant negative quadratic effect ($z = -2.39$, $p = .017$); effects sizes were largest at 69.3% European-American. Effect sizes were significantly larger for clinician-led groups ($d = 0.49$) versus researcher-led groups ($d = 0.17$; $z = -2.72$, $p = .007$); peer-led groups ($d = 0.24$) did not differ from clinician-led groups ($z = -1.94$, $p = .053$). Number of facilitators exhibited a significant positive linear effect ($z = 2.22$, $p = .026$) and a significant negative quadratic effect ($z = -3.71$, $p < .001$); effects sizes peaked at 2.3 facilitators. Number of training hours exhibited a non-significant negative linear effect ($z = -0.92$, $p = .357$) and a significant negative quadratic effect ($z = -3.33$, $p < .001$); effects sizes peaked at 5.6 h of training. Effect sizes were significantly larger when facilitators were supervised ($d = 0.26$) than when they were not ($d = -0.06$) ($z = 2.60$, $p = .009$).

7.2.4. Negative affect

Among dissonance intervention versus control condition contrasts, five moderators were significant. Number of dissonance-inducing activities was a significant predictor of effect sizes, wherein effects were larger for studies with a greater number of dissonance-inducing activities ($z = 2.15$, $p = .031$); effect sizes were $d = 0.18$ at 1 *SD* below the mean (5.2) and $d = 0.35$ at 1 *SD* above the mean (12.3). Intervention duration exhibited a significant positive linear effect ($z = 2.60$, $p = .009$) and a significant negative quadratic effect ($z = -2.11$, $p = .035$); effects sizes peaked at 5.0 intervention sessions. Group size exhibited a non-significant negative linear effect ($z = -0.04$, $p = .970$) and a significant negative quadratic effect ($z = -2.13$, $p = .033$); effects sizes peaked at group sizes of 9.8. Effect sizes were significantly larger when body image concerns were required ($d = 0.34$) than when they were not ($d = 0.20$) ($z = 2.19$, $p = .029$). The percentage of European-American participants was a significant moderator of effect sizes, wherein effects were lower in interventions with a greater percentage of European-American participants ($z = -2.26$, $p = .024$); effect sizes were $d = 0.31$ at 1 *SD* below the mean (57.6) and $d = 0.20$ at 1 *SD* above the mean (90.9).

Among dissonance intervention versus alternative intervention contrasts, four moderators were significant. Group size exhibited a non-significant linear effect ($z = 0.01$, $p = .994$) and a significant negative quadratic effect ($z = -2.32$, $p = .020$); effects sizes peaked at group sizes of 9.8. Effect sizes were significantly larger for video-recorded studies ($d = 0.33$) versus non-recorded studies ($d = 0.17$) ($z = 2.15$, $p = .032$). Effect sizes were significantly larger for clinician-led groups ($d = 0.37$) versus researcher-led groups ($d = 0.30$; $z = -2.54$, $p = .011$); peer-led groups ($d = 0.12$) did not differ from clinician-led groups ($z = -0.60$, $p = .548$). Effect sizes were significantly larger when facilitators were supervised ($d = 0.27$) than when they were not ($d = 0.04$) ($z = 2.64$, $p = .008$).

7.2.5. Eating disorder symptoms

Among dissonance intervention versus control condition contrasts, there were three significant moderators. Effect sizes were significantly larger when body image concerns were required ($d = 0.36$) than when they were not ($d = 0.23$) ($z = 2.42$, $p = .016$). Effect sizes were significantly smaller when interventions were mandated ($d = 0.20$) than when they were not ($d = 0.34$) ($z = -2.53$, $p = .011$). Percentage of European-American participants exhibited a significant negative linear effect ($z = -3.03$, $p = .002$) and a significant negative quadratic effect ($z = -2.61$, $p = .009$); effects were largest at 62.6% European-American.

Among dissonance intervention versus alternative intervention contrasts, seven moderators were significant. Number of dissonance-inducing activities was a significant predictor of effect sizes, wherein effects were larger for studies with more dissonance-inducing activities ($z = 3.92$, $p < .001$); effect sizes were $d = -0.09$ at 1 *SD* below the mean (5.2) and $d = 0.29$ at 1 *SD* above the mean (12.3). Intervention duration exhibited a non-significant positive linear effect ($z = 0.89$, $p = .375$) and a significant negative quadratic effect ($z = -2.27$, $p = .023$); effects sizes peaked at 3.7 intervention sessions. Group size exhibited a significant positive linear effect ($z = 2.86$, $p = .004$) and a significant negative quadratic effect ($z = -2.51$, $p = .012$); effects sizes peaked at group sizes of 13.4. Mean age of sample exhibited a non-significant negative linear effect ($z = -0.05$, $p = .964$) and a significant positive quadratic effect ($z = 2.76$, $p = .006$) that resulted in a U-shaped curve with the minimal effect size at age 19.6. Effect sizes were significantly larger for clinician-led groups ($d = 0.38$) versus researcher-led groups ($d = 0.17$; $z = -2.03$, $p = .043$); peer-led groups ($d = 0.19$) did not differ from clinician-led groups ($z = -1.68$, $p = .093$). Number of facilitators exhibited a significant positive linear

effect ($z = 3.44$, $p < .001$) and a significant negative quadratic effect ($z = -4.14$, $p < .001$); effects sizes peaked at 2.5 facilitators. Number of training hours exhibited a non-significant negative linear effect ($z = -0.92$, $p = .357$) and a significant negative quadratic effect ($z = -3.33$, $p < .001$); effects sizes peaked at 5.6 h.

8. Discussion

8.1. Summary of average effect sizes

We identified 56 published and unpublished trials from which we extracted 68 dissonance-based eating disorder prevention programs involving data from 7808 participants. As hypothesized, dissonance-based prevention programs produced significantly larger reductions in thin ideal internalization ($d = 0.57$), body dissatisfaction ($d = 0.42$), dieting ($d = 0.37$), negative affect ($d = 0.29$), and eating disorder symptoms ($d = 0.31$) in contrast to minimal-intervention control conditions, the most common type of control condition in the eating disorder prevention literature. These effects are medium to small effects. Results establish that dissonance-based prevention programs produced larger reductions in these outcomes than occur in response to the passage of time (regression to the mean) or from measurement of the outcomes, which is often associated with reductions in pathological outcomes. These analyses averaged across all dissonance-based eating disorder prevention programs, which included some that did not produce significant reductions in outcomes (see below), suggesting that these average effect sizes are a conservative estimate of efficacy. Nevertheless, the magnitude of these average effects is noteworthy because the interventions were brief and because prevention programs and treatments for these outcomes often produce only small effects relative to minimal-intervention comparison conditions (Corrieri et al., 2014; Scott-Sheldon, Carey, Elliott, Garey, & Carey, 2014; Turk et al., 2009).

Also as hypothesized, dissonance-based eating disorder prevention programs produced significantly larger reductions in thin-ideal internalization ($d = 0.31$), body dissatisfaction ($d = 0.18$), dieting ($d = 0.17$), negative affect ($d = 0.21$) and eating disorder symptoms ($d = 0.13$) in contrast to alternative interventions. These findings establish that these interventions also produce larger reductions in the outcomes than occur in response to expectancies and demand characteristics that operate in any credible active intervention condition, as well as non-specific factors that operate in any group-based intervention in which participants interact with a facilitator and other group members (e.g., emotional support). Although the effects from these more stringent comparisons are smaller, we think it vital to show that a particular intervention produces superior effects than alternative credible interventions because this establishes that the effects are not solely due to demand characteristics, expectances, and non-specific effects. These findings are encouraging because no other eating disorder prevention program has produced significantly greater reductions than active credible alternative interventions. Again, it is important to note that some of the alternative interventions are efficacious (e.g., the *Healthy Weight* eating disorder and obesity prevention program; Stice et al., 2006, Stice, Marti, Spoor, et al., 2008; Stice, Rohde, Shaw, & Marti, 2012; Stice, Rohde, Shaw, & Marti, 2013). When interpreting these average effect sizes, it is important to note that the trials in this meta-analysis included females and males, participants who ranged in age from preadolescent to later adulthood, and a wide range of ethnic and racial groups, including studies conducted in countries other than the USA (e.g., Brazil).

One noteworthy pattern in the findings was that the effects were stronger for thin-ideal internalization than for body dissatisfaction, dieting, negative affect, and eating disorder symptoms compared to both types of comparison conditions. This might be interpreted as providing additional support for the intervention theory for dissonance-based eating disorder prevention programs because the intervention activities center on exploring the costs of pursuing the thin ideal.

Effect sizes shown in Table 2 reveal that the trials that observed the largest effects ($d > 0.70$) for eating disorder symptoms, arguably the most critical outcome, emerged for the prevention programs evaluated by Atkinson and Wade (2016), Green et al. (2016), Green et al. (2017), Krishna (2011), Machin (2008), Mitchell et al. (2007), and Rohde et al., 2014) relative to minimal intervention control conditions. Given that the studies by Machin, Mitchell, and Green, and Rohde evaluated interventions that added unique exercises to the standard *Body Project* the pattern of findings suggest that these versions of dissonance-based eating disorder prevention programs might be good candidates for implementation. For instance, Green and associates added a focus of the costs of social comparisons, particularly via social media, to the standard *Body Project*, and Rohde and associates expanded the *Body Project* from 4 to 6 sessions, adding novel interactive exercises to increase engagement (e.g., groups produce a video news report on the costs of the thin beauty ideal). Adding additional dissonance-induction activities theoretically contributed to greater reductions in thin-ideal internalization, body dissatisfaction, and eating disorder symptoms, which is substantiated by the evidence discussed below that dissonance-based prevention programs that contain more dissonance-induction activities and more sessions were associated with larger intervention effects.

Some trials found very small effects for dissonance-based eating disorder prevention programs relative to control conditions (e.g., Green et al., 2005; McMillan et al., 2011; Serdar et al., 2014; Wolfe, 1992). One possible explanation for the small effect sizes for the high-dissonance prevention program evaluated by Green et al. (2005) is that it is the only trial that used a posttest-assessment design, which is not as sensitive as the pretest-posttest repeated-measures design in detecting intervention effects. The small effect sizes for the low-dissonance prevention programs evaluated by Green et al. (2005) and McMillan et al. (2011) would be expected because those interventions were expressly designed to minimize dissonance-induction, a key mechanism of effect for dissonance-based prevention programs. The most likely explanation for the small effects from Serdar et al. (2014) is that it evaluated a simple Internet-based intervention in which participants typed responses to written versions of the Socratic questions posed by facilitators to participants in *Body Project* groups, which would not be expected to induce much dissonance because of the low public accountability and level of effort required. In contrast, in the more effective *eBody Project* participants create personalized profile pages with pictures of the participant and post their anti-thin-ideal letters and videos on the Internet to increase accountability and complete additional dissonance-induction exercises to increase the level of required effort. The most likely explanation for the small effects from Wolfe (1992) is that the intervention evaluated in that study did not contain 8 out of the 9 dissonance-induction activities contained in the *Body Project*. As such, the small effects in the Green, McMillan, Serdar and Wolfe trials can be interpreted as providing additional support for the intervention theory of dissonance-based prevention programs.

Seven out of 27 trials did not find that dissonance-based prevention programs produced significantly larger reductions in outcomes that alternative interventions (Atkinson & Wade, 2016; Becker et al., 2006; Becker et al., 2008; Pennesi & Wade, 2017; Stice et al., 2001; Stice et al., 2003; Wade et al., 2009). Atkinson and Wade (2016) found that the *Body Project* did not produce significantly larger reductions in outcomes compared to a mindfulness-based eating disorder prevention program, suggesting that it would be worthwhile for future trials to evaluate such prevention program. Stice et al. (2001, 2003) found that the *Body Project* did not produce superior effects than the *Healthy Weight* eating disorder prevention program, likely because the latter has been found to produce reductions in eating disorder risk factors, eating disorder symptoms, and future eating disorder onset (Stice et al., 2006; Stice, Marti, Spoor, et al., 2008; Stice, Rohde, Durant, et al., 2013; Stice, Rohde, Gau, & Shaw, 2012). Becker et al. (2006, 2008) found that the dissonance-based eating disorder prevention program they evaluated

did not produce markedly larger reductions in eating disorder symptoms than a prevention program promoting media literacy and media advocacy, suggesting that the latter may also be efficacious. In addition, the fact that Becker and associates evaluated a streamlined version of the *Body Project* that omitted several dissonance-induction activities may also have attenuated effects, based on the evidence from moderator analyses that prevention programs with more dissonance-induction activities and more intervention sessions produce larger effects (discussed below). Pennesi and Wade (2017) found that a very brief dissonance-based prevention program did not produce larger reductions in eating disorder symptoms relative to an imagery rescripting prevention program and Wade et al. (2009) found that a very brief dissonance-based prevention program did not produce larger reductions in body dissatisfaction than a brief acceptance-based intervention or a distraction-based intervention, most likely because the dissonance-based interventions were extremely brief and did not contain most of the dissonance-induction activities used in other dissonance-based prevention programs.

8.2. Moderators of dissonance-based eating disorder prevention programs

The second aim of this meta-analysis was to test whether certain intervention, participant, and facilitator features correlate with larger effects on eating disorder risk factors and symptoms, which should be useful in guiding implementation of the most effective version of this prevention program to the optimal population and in the optimal manner by facilitators. Before interpreting the moderating effects, it is important to acknowledge that we conducted a large number of inferential tests. The fact that 34% of the moderation tests were statistically significant is reassuring because only 5% would have been expected to be significant based on chance alone. We considered using a more conservative significance threshold, however given that fewer than 60 studies contributed effect sizes to most moderator analyses, we were more concerned about false-negative findings than false-positive findings. Nonetheless, given the possibility of chance findings, it is probably best to prioritize features that moderated effects for at least two outcomes, as we have more confidence that those effects are reliable.

Interestingly, moderator analyses appeared more sensitive when the effects of dissonance-based eating disorder prevention programs were compared to active alternative interventions (43% of the moderator effects were significant) versus minimal-intervention control conditions (25% of the moderator effects were significant). This suggests that there was less “noise” in the data when dissonance-based interventions are compared to alternative interventions, potentially because alternative interventions better equate conditions on expectancy effects and demand characteristics inherent to randomized trials. We also think that the analyses comparing dissonance-based prevention programs to alternative interventions is more important because research suggests that assessment-only conditions are typically not associated with clinically meaningful reductions in outcomes and the results from the comparative trials should be more informative to clinicians attempting to decide which eating disorder prevention program to implement.

With regard to intervention features, we hypothesized that effects would be larger in trials in which participants engaged in a greater number of dissonance-inducing activities, as this should maximize dissonance-induction. This feature significantly moderated the effects on four of the five outcomes (body dissatisfaction, dieting, negative affect, and eating disorder symptoms), making it one of our most consistent effect size moderators. These results suggest that it would be optimal to implement versions of the *Body Project* that contain all of the dissonance-induction activities contained in the 4-session *Body Project*, rather than versions that omit some of these activities. Indeed, these moderator findings imply that adding addition in-session or home practice dissonance-induction exercises, such as was done for the 6-session version of the *Body Project* that has been implemented in the USA, might produce the largest reductions in outcomes and is worth evaluating further.

We had also hypothesized that dissonance-based prevention programs with more sessions would produce larger effects because they typically contain more in-session and between-session dissonance-inducing activities, which should produce more dissonance due to the increased level of effort. Further, we theorized that the increased opportunity to reflect on intervention content between sessions might promote improved consolidation of learning that contributes to larger effects. In addition, meeting multiple times as a group may contribute to a stronger development of social support, which *Body Project* participants state is one of the most valuable elements of this intervention. Effect sizes were significantly larger for two out of the five outcomes (body dissatisfaction and negative affect) for dissonance-based eating disorder prevention programs with more versus fewer sessions, with two quadratic effects suggesting that the optimal effects occur when there are 4 to 5 sessions. Results suggest that dissonance-based eating disorder prevention programs that have at least 4 sessions should be implemented if the goal is to maximize intervention effects. The evidence that programs with more dissonance-induction activities and more sessions produce larger effects than those with fewer activities and sessions provide evidence of a dose-response relation for this prevention program, which is a key test of the intervention theory for a prevention program.

We had hypothesized that intervention effects would be larger for dissonance-based eating disorder prevention programs implemented in larger versus smaller groups, as the former would theoretically increase public accountability that increases dissonance-induction. This moderator was associated with a significantly larger effect for three of five the outcomes (thin-ideal internalization, negative affect and eating disorder symptoms). The quadratic relations suggested that the largest effect size for this outcome emerged when groups contained 10 participants. This is consistent with practice guidelines for effective group psychotherapy, which recommend 7–10 participants per group (American Group Psychotherapy Association, 2007; Bernard et al., 2008).

In addition, we had hypothesized the group-based dissonance-based eating disorder prevention programs would produce larger effects than versions implemented on-line, reasoning that the former would promote greater public accountability and consequent dissonance-induction. However, group versus on-line delivery only moderated the effects of one of the five outcomes (body dissatisfaction), suggesting that this moderator may have limited impact. It is important to note that there was substantial heterogeneity of the effects for on-line delivery of dissonance-based eating disorder prevention programs. One involved only one of the 12 dissonance-induction activities from the *Body Project* (Chithambo & Huey, 2017) and another evaluated a simple Internet-based intervention in which participants typed responses to written versions of the Socratic questions posed by facilitators to participants in *Body Project* groups (Serdar et al., 2014). In contrast, we evaluated the Internet-delivered *eBody Project* that conserved each of the dissonance-induction activities from the group-based *Body Project* and included posting of writing and video home exercises on the Internet to promote public accountability. The former two programs produced a much smaller average effect than the latter program ($d = 0.04$ versus 0.43).

With regard to the last intervention feature we examined, we had hypothesized that dissonance-based eating disorder prevention programs would produce larger effects if the sessions were video-recorded because it should theoretically increase public accountability and contribute to larger effects. As expected, intervention effects were significantly larger when dissonance-based eating disorder prevention groups were video-recorded versus not video-recorded for three of the five outcomes (body dissatisfaction, dieting, and negative affect). It is also possible that videotaping sessions improves intervention fidelity because facilitators have greater accountability to supervisors, assuming the recordings are reviewed on at least a random basis, which

could also contribute to larger effects. These results imply that if interventionists are interested in maximizing intervention effects, the group-based sessions should be video-recorded, and perhaps also reviewed by supervisors.

We investigated five participant features hypothesized to moderate the effects of dissonance-based eating disorder prevention programs. Results provided support for the hypothesis that intervention effects of dissonance-based eating disorder prevention programs would be larger if implemented to body dissatisfied young women at high risk for eating disorders; this moderator affected three of the five outcomes (body dissatisfaction, negative affect, and eating disorder symptoms). Results converge with prior meta-analytic reviews that found that a broad array of eating disorder prevention programs produce larger effects when delivered to high-risk versus unselected participants (Stice, Shaw, & Marti, 2007) and with evidence that several universal prevention programs were more effective for subgroups of high-risk participants than for the full sample (e.g., Buddeberg-Fischer, Klaghofer, Gnam, & Buddeberg, 1998; Stewart, Carter, Drinkwater, Hainsworth, & Fairburn, 2001; Weiss & Wertheim, 2005). Collectively these data imply that the larger effects may occur because young women with body image concerns are more motivated to engage in the intervention, find the material more pertinent to their lives, and have more room for improvement because of their elevated risk. Results suggest that larger effects will typically occur for dissonance-based eating disorder prevention programs if they are implemented in a selective fashion to high-risk individuals. It is important to acknowledge that some participants who enroll in body acceptance eating disorder prevention programs due to body image concerns also have threshold or subthreshold eating disorders (Stice, Rohde, et al., 2017a,Stice, Rohde, et al., 2017b), which blurs the line between selected and indicated prevention. Further, it is possible that effects for dissonance-based eating disorder prevention programs will be even larger if they are implemented in an indicated fashion to young women with subclinical eating pathology versus when implemented universally to unselected populations, but only two trials evaluated a dissonance-based eating disorder prevention program when delivered in an indicated fashion (Green et al., 2016; Green, Kroska, & Herrick, 2018), preventing us from comparing the effects sizes from indicated versus selective and universal dissonance-based prevention programs. An added benefit of this approach is that the intervention can be described as a body acceptance class, which facilitates recruitment. Focusing only on high-risk individuals would make dissemination more manageable and less expensive because there are fewer high-risk individuals. However, one potential downside of indicated prevention programs is that they miss low-risk individuals who may still show onset of eating disorders.

Results provided support for the hypothesis that intervention effects would be larger if participants completed the dissonance-based eating disorder prevention programs on a voluntary versus mandated basis, though this moderator was significant for only two outcomes (body dissatisfaction and eating disorder symptoms). Theoretically, engaging in dissonance-induction activities on a voluntary basis results in greater dissonance-induction (Baumeister & Tice, 1984). These findings suggest it might be optimal to implement dissonance-based eating disorder prevention programs on a voluntary basis. However, several trials have observed significant reductions in risk factors and eating disorder symptoms when the dissonance-based prevention programs are mandated (e.g., Becker et al., 2010), suggesting that implementing the prevention programs on a mandated basis can benefit participants.

Counter to our hypothesis based on dissonance theory that intervention effects would be smaller when participants are compensated for participating in the prevention trial (typically for completing assessments) versus when they are not, moderator analyses revealed that effects were significantly *stronger* when participants were compensated for three of the five outcomes (thin-ideal internalization, body dissatisfaction, and dieting). It is possible that effects were larger when participants were compensated because they were more likely to attend more intervention sessions, which could explain the larger effects. This null finding might be interpreted as suggesting that external justification for engaging in counter-attitudinal activities does not reduce dissonance-induction, as suggested by much more tightly controlled trials (e.g., Cohen, 1962). However, we would argue that experiments that manipulate external justification for engaging in counter-attitudinal activities permit firmer inferences. For instance, it is possible that trials that compensated participants tended to require that participants have body image concerns, which might have contributed to the observed moderating effects. With regard to implementation implications, findings suggest that compensating participants for attending dissonance-based eating disorder prevention programs does not attenuate effects, and indeed that it might be optimal to do so if the goal is to maximize intervention effects.

We hypothesized that intervention effects for dissonance-based eating disorder prevention programs would be larger for older versus younger participants, based on findings from a meta-analytic review of a broader range of eating disorder prevention programs (Stice, Presnell, et al., 2007). There were significant moderating effects for two of the five outcomes (dieting and eating disorder symptoms). The significant quadratic effects for these outcomes suggested that the effects were smallest for 19-year old participants, but larger for mid-adolescent females and young-adult women. Results imply that it might be optimal to implement dissonance-based eating disorder prevention programs to mid-adolescent females and with adult women versus older-adolescent females. However, the fact that randomized trials have found that dissonance-based eating disorder prevention programs have produced significant reductions in eating disorder risk factors and symptoms for female middle school students (Halliwell et al., 2015; Rohde et al., 2014), high-school students (Stice et al., 2009, 2011), college students (Becker et al., 2010; Stice, Rohde, Durant, et al., 2013), and young adult women (Rohde et al., 2017) implies that even though effects may be smaller for 19 year olds, they are still clinically meaningful.

Results indicated that dissonance-based eating disorder prevention programs produced significantly larger reductions four of the five outcomes (body dissatisfaction, dieting, negative affect, and eating disorder symptoms) in trials that involved samples with a lower percentage of European-Americans. The main effects indicated that intervention effects tended to be larger for samples that were more ethnically diverse, though there were some quadratic effects that suggested that effects were greatest when 30%-40 of the sample were from ethnic minority groups. These findings are consistent with a prior studies that found that a dissonance-based eating disorder prevention program produced non-significantly larger reductions in eating disorder risk factors and symptoms for Asian-American, Hispanic, and African-American versus European-American participants (Rodriguez, Marchand, Ng, & Stice, 2008; Stice, Marti, & Cheng, 2014). Moreover, more recent findings from a large data set revealed that European-American young women did not report significantly higher endorsement of the thin beauty ideal than did Hispanic, Asian American, of African American young women (Cheng et al., 2018); indeed, Asian American young women reported significantly greater thin-ideal internalization than both European-American and African-American young women. Results from that large ethnic differences study and the present study suggest that pursuit of the thin beauty ideal may be a trans-ethnicity risk factor for eating disorders, which may explain why dissonance-based eating disorder prevention programs work well with multiple ethnic groups. Thus, results imply that it may not be necessary to develop eating disorder prevention programs that are tailored to specific ethnic groups, which would complicate implementation. Dissonance-based interventions, due to their use of Socratic questioning and greater engagement of participant involvement for change, may adapt more naturally to female appearance ideals associated with various cultures compared to interventions that are more focused on presenting factual information or are more proscriptive in their model of change. This suggests that dissonance-based prevention and treatment

interventions for other applied outcomes may likewise be effective for a wide variety of racial and ethnic groups.

We investigated four facilitator factors hypothesized to predict larger intervention effects for dissonance-based eating disorder prevention programs. Results provided no support for the hypothesis that clinician-led groups would produce larger intervention effects than peer educator-led groups. This finding dovetails with results from the only randomized trial to compare the effectiveness of clinician-led to peer-led *Body Project* groups (Stice, Rohde, et al., 2017a,Stice, Rohde, et al., 2017b). Collectively, these findings suggest that it should be equally valuable to implement dissonance-based eating disorder prevention programs with peer educators or clinicians. Of course, given that there are typically many more peer-educators at colleges than clinicians, and peer-educators often deliver interventions as part of their coursework (though some are paid), they represent a very cost-effective method of broad implementation of interventions (Becker et al., 2008). Results provided evidence that intervention effects were significantly larger for three of the five outcomes for clinician-led groups versus researcher-led groups (dieting, negative affect, and eating disorder symptoms). The finding that clinicians produced stronger effects than closely trained and supervised research therapists is encouraging for broader implementation of these programs. Clinicians may have produced superior effects because they have more experience than research clinicians, consistent with one study found that clinicians reported an average of 8 years of experience working with young adults in a professional mental health role, all had experience delivering prevention programs, and 89% had experience conducting group interventions (Stice, Rohde, Durant, et al., 2013).

We tested the hypothesis that dissonance-based eating disorder prevention programs delivered by more facilitators would produce larger intervention effects, based on the assumption that a greater number of facilitators would be able to conduct more effective sessions and adhere to the curriculum more closely. There was evidence that effects were significantly larger when more versus fewer individuals facilitated dissonance-based eating disorder prevention programs for four of the five outcomes (thin-ideal internalization, body dissatisfaction, dieting, and eating disorder symptoms), making this one of the moderators that received the greatest empirical support. However, there were quadratic effects for three of these outcomes that suggested that implementation with 2–3 facilitators might be ideal for producing maximal effects. The implementation implication is that it would be ideal to have 2–3 individuals facilitate dissonance-based eating disorder prevention programs.

We also evaluated the hypothesis that facilitators who received more hours of training in implementing dissonance-based eating disorder prevention programs would produce larger effects than those who received less training. This moderator produced significant effects for four of the five outcomes (thin-ideal internalization, body dissatisfaction, dieting, and eating disorder symptoms), suggesting it is also one of the most critical moderators. There was a quadratic component to this effect for all outcomes, which indicated that the largest effects for these outcomes occurred when training took 5.5 to 7.2 h, suggesting that 6–7 h might be optimal for implementation efforts.

Finally, we tested the hypothesis that providing supervision to facilitators implementing dissonance-based eating disorder prevention programs would be associated with larger intervention effects versus when supervision was not provided. Results indicated that providing supervision was associated with significantly larger effects for four of the five outcomes (thin-ideal internalization, body dissatisfaction, dieting, and negative affect), making this another of the most critical moderators identified in this meta-analysis. Theoretically, supervision based on video-recorded groups allows careful monitoring of both fidelity and competence after the initial training has been completed and provides an opportunity for corrective feedback when indicated, though other types of supervision are also beneficial. Results provide very strong evidence that providing supervision will optimize intervention effects from dissonance-based eating disorder prevention programs.

8.3. Caveats of moderator analyses

First, evidence that a moderator is associated with intervention effect sizes does not establish a causal relation. It will be necessary to experimentally manipulate the moderators in a trial to permit firmer causal inferences regarding the effects of the moderators. Second, many of the conclusions regarding moderators of intervention effects are based on theoretical considerations rather than on direct empirical evidence. For instance, although we posit that selective programs delivered to youth with body image concerns produce large effects because the distress that may characterize high-risk samples leads them to engage more effectively in the program content, most trials did not systematically report attendance, homework completion, or session engagement and participation, which would be useful for confirming this speculation. Third, it is possible that some of the moderator effects are due to chance because the moderator analyses involved numerous inferential tests. However, 34% of the moderator effects were statistically significant, versus the 5% that would be expected based on chance alone. The fact that the number of moderator effects was over 7 times the number that would be expected based on chance suggests that most of the effects are reliable. Moreover, the fact that many of the moderator findings converge with findings from lab-based experiments on factors that maximize dissonance-induction and with results from prior meta-analytic reviews increases the confidence that can be placed in the findings.

9. Conclusions and directions for future research

In sum, this meta-analytic review found that dissonance-based eating disorder prevention programs produced significant reductions in each of the four examined risk factors and in eating disorder symptoms that correspond to small to medium effect sizes relative to control conditions. The average effects for dissonance-based eating disorder prevention programs relative to alternative interventions were smaller, but the most commonly evaluated alternative intervention has itself been shown to reduce eating disorder risk factors and symptoms relative to control conditions and alternative interventions and to reduce eating disorder onset in multiple trials, which suggests this may be a high standard. As noted, no other eating disorder prevention program has been found to produce significantly larger intervention effects than alternative interventions.

The moderator findings suggest that effects should be largest if interventionists implement versions of the dissonance-based eating disorder prevention program that include all dissonance-induction activities contained in the *Body Project* and use the 4- or 6-session versions of the prevention program. Effects should likewise be largest when dissonance-based eating disorder prevention programs are delivered to groups of approximately 10 young women and when sessions are video-recorded, as both factors increase accountability, which increases dissonance-induction. Effects should also be larger when participants complete the prevention programs voluntarily, have body image concerns, and are compensated. Moderator analyses also suggested that effects should be larger if dissonance-based eating disorder prevention programs are implemented to adolescent girls and young adult women, and to more ethnically diverse groups. In addition, intervention effects should be larger when clinicians or peer educators implement groups, there are 2–3 facilitators, and when facilitators received between 6 and 7 h of training and, perhaps most importantly, are supervised.

In terms of future research directions, it would be useful to conduct randomized experiments that manipulate the enumerated moderators to confirm that they are causally related to larger intervention effects. It might be most logical to start with the moderators that received the strongest support in this meta-analysis, which include the number of dissonance-based activities in the prevention program, the number of

facilitators, the number of training hours, and whether supervision is provided. For instance, one trial directly compared the effects of the *Body Project* when implemented by clinicians versus peer educators, providing evidence that peer-led groups produced significantly greater reductions in future onset of eating disorders over 3-year follow-up compared to clinician-led groups (+ +Stice, Rohde, et al., 2017a,Stice, Rohde, et al., 2017b). It would be particularly useful to have experimental support for the hypothesis that combining these moderators should produce larger intervention effects. It would also be helpful if future trials of dissonance-based eating disorder prevention programs systematically report average attendance of the intervention sessions, homework completion, intervention fidelity, therapist competence, and cohesion of groups, as this would allow an investigation of the effects of these variables on outcomes and potentially elucidate the mechanism of effect for the moderators. It might be also be useful to identify the types of young women who do not respond well to dissonance-based eating disorder prevention programs (i.e., who subsequently develop eating disorders despite completing this type of prevention program) so that alternative interventions can be developed for these individuals. Although the evidence-base for dissonance-based eating disorder prevention programs is encouraging, we think it vital to develop additional prevention programs that address distinct risk factors for eating disorders (e.g., psychosocial impairment and negative affect) if we are to realize the goal of reducing the population prevalence of eating disorders, which represent a major mental health problem confronting young women. More broadly, results suggest that it would be potentially useful to develop dissonance-based prevention programs for other health and mental health problems, such as depression and suicidality, substance misuse, and obesity, as dissonance-induction appears to represent a robust method of reducing attitudinal and behavioral risk factors for public health problems.

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Appendix A. Supplementary data

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References

Allen, K., Byrne, S., Oddy, H., & Crosby, R. (2013). Eating disorders in adolescents: Prevalence, stability, and psychosocial correlates in a population-based sample of male and female adolescents. *Journal of Abnormal Psychology, 122*, 720–732.

Amaral A., Stice E. and Ferreira M., Pilot trial of a dissonance-based eating disorder prevention program with Brazilian girls, 2018, (Manuscript submitted for publication).

American Group Psychotherapy Association (2007). *Practice guidelines for group psychotherapy. Science to service task force report*.

Arcelus, J., Mitchell, A. J., Wales, J., & Nielsen, S. (2011). Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. *Archives of General Psychiatry, 68*(7), 724–731.

Aronson, E., Fried, C., & Stone, J. (1991). Overcoming denial and increasing the intention to use condoms through the induction of hypocrisy. *American Journal of Public Health, 81*, 1636–1637.

Atkinson, M., & Wade, T. (2016). Does mindfulness have potential in eating disorder prevention? A preliminary controlled trial with young adult women. *Early Intervention in Psychiatry, 10*, 234–245.

Bauer, D. J., & Curran, P. J. (2005). Probing interactions in fixed and multilevel regression: Inferential and graphical techniques. *Multivariate Behavioral Research, 40*, 373–400.

Baumeister, R. F., & Tice, D. M. (1984). Role of self-presentation and choice in cognitive dissonance under forced compliance: Necessary or sufficient causes? *Journal of Personality and Social Psychology, 46*(1), 5–13.

Becker, B. J. (1988). Synthesizing standardized mean-change measures. *British Journal of Mathematical and Statistical Psychology, 41*, 257–278. https://doi.org/10.1111/j.2044-8317.1988.tb00901.x.

Becker, C., Smith, L. M., & Ciao, A. C. (2005). Reducing eating disorder risk factors in sorority members: A randomized trial. *Behavior Therapy, 36*, 245–253.

Becker, C. B., Bull, S., Schaumberg, K., Cauble, A., & Franco, A. (2008). Effectiveness of peer-led eating disorders prevention: A replication trial. *Journal of Consulting and Clinical Psychology, 76*, 347–354.

Becker, C. B., McDaniel, L., Bull, S., Powell, M., & McIntyre, K. (2012). Can we reduce eating disorder risk factors in female college athletes? A randomized exploratory investigation of two per-led interventions. *Body Image, 9*, 31–42.

Becker, C. B., Smith, L. M., & Ciao, A. C. (2006). Peer facilitated eating disorders prevention: A randomized effectiveness trial of cognitive dissonance and media advocacy. *Journal of Counseling Psychology, 53*, 550–555.

Becker, C. B., & Stice, E. (2008). *Sorority body image program: Group leader guide.* New York: Oxford University Press.

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

Begg, S., Vos, T., Baker, B., Stevenson, C., Stanley, L., & Lopez, A. (2007). *The burden of disease and injury in Australia 2003.* Canberra: Australian Institute of Health and Welfaire.

Bernard, H., Burlingame, G., Flores, P., Greene, L., Joyce, A., Kobos, J., et al. (2008). Clinical practice guidelines for group psychotherapy. *International Journal of Group Psychology, 58*, 455–542.

Brown, T., & Keel, P. (2015). A randomized controlled trial of a peer co-led dissonance-based eating disorder prevention program for gay men. *Behaviour Research and Therapy, 74*, 1–10.

Buddeberg-Fischer, B., Klaghofer, R., Gnam, G., & Buddeberg, C. (1998). Prevention of disturbed eating behaviour: A prospective intervention study in 14- to 19-year old Swiss students. *Acta Psychiatrica Scandinavica, 98*, 146–155.

Cheng, Z., Perko, V., Fuller-Marashi, L., Gau, J., & Stice, E. (2018). *Ethnic differences in eating disorder prevalence, risk factors, and predictive effects of risk factors among young women.* (Under review as a revise and resubmit).

Cheung, M. W. L. (2015a). *Meta-analysis: A structural equation modeling approach.* New York, NY: Wiley.

Cheung, M. W. L. (2015b). metaSEM: An R package for meta-analysis using structural equation modeling. *Frontiers in Psychology, 5*. https://doi.org/10.3389/fpsyg.2014.01521. Retrieved from http://journal.frontiersin.org/Journal/10.3389/fpsyg.2014.01521/abstract.

Chithambo, T. P., & Huey, S. J. (2017). Internet-delivered eating disorder prevention: A randomized controlled trial of dissonance-based and cognitive-behavioral interventions. *International Journal of Eating Disorders, 50*, 1142–1151.

Ciao, A. (2012). Effectiveness and feasibility of peer-delivered dissonance-based eating disorder prevention in high school girls. Unpublished doctoral thesis. *University of Hawaii*. Honolulu: HI.

Ciao, A., Latner, J., Brown, K., Ebneter, D., & Becker, C. (2015). Effectiveness of a peer-delivered dissonance-based program in reducing eating disorder risk factors in high school girls. *International Journal of Eating Disorders, 48*, 779–784.

Cochran, W. G. (1954). The combination of estimates from different experiments. *Biometrics, 10*, 101–129. https://doi.org/10.2307/3001666.

Cohen, J. (1962). The statistical power of abnormal-social psychological research: a review. *Journal of Abnormal and Social Psychology, 65*, 145–153.

Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences* (2nd ed.). Hillsdale, NJ: Lawrence Erlbaum Associates, Publishers.

Cooper, J., & Axsom, D. (1982). Effort justification in psychotherapy. In G. Weary, & H. Mirels (Eds.). *Integrations of clinical and social psychology* (pp. 98–121). New York, NY: Oxford.

Corrieri, S., Heider, D., Conrad, I., Blume, A., Konig, H., & Riedel-Heller, S. (2014). School-based prevention programs for depression and anxiety in adolescence: A systematic review. *Health Promotion International, 29*, 427–441.

Crow, S., & Smiley, N. (2010). Costs and cost-effectiveness in eating disorders. In W. S. Agras (Ed.). *The Oxford handbook of eating disorders* (pp. 480–485). Oxford: Oxford University Press.

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

Festinger, L., & Carlsmith, J. M. (1959). Cognitive consequences of forced compliance. *The Journal of Abnormal and Social Psychology, 58*(2), 203–210.

Ghaderi, A., & Scott, B. (2001). Prevalence, incidence and prospective risk factors for eating disorders. *Acta Psychiatrica Scandinavica, 104*, 122–130.

Green, M., Scott, N., Diyankova, I., Gasser, C., & Pederson, E. (2005). Eating disorder prevention: An experimental comparison of high-level dissonance, low level dissonance, and no-treatment control. *Eating Disorders, 13*, 157–169.

Green, M., Willis, M., Fernandez-Kong, K., Reyes, S., Linkhart, R., Johnson, M., et al. (2016). Dissonance-based eating disorder program reduces cardiac risk: A preliminary trial. *Health Psychology, 36*, 346–355.

Green, M. A., Kroska, A., & Herrick, A. B. (2018). B., Sage, E., Miles, L., Ravet, M., Powers, M., Whitegoat, W., Linkhart, R., & King. *A preliminary trial of an online dissonance-based eating disorder intervention. Eating Behaviors, 31*, 88–98.

Green, M. A., Willis, M., Fernandez-Kong, K., Reyes, S., Linkhart, R., Johnson, M., et al. (2017). A controlled randomized preliminary trial of a modified dissonance-based

eating disorder intervention program. *Journal of Clinical Psychology, 73*, 1612–1628.
Halliwell, E., & Diedrichs, P. (2014). Brief report: Testing a dissonance body image intervention among young girls. *Healthy Psychology, 33*, 201–204.
Halliwell, E., Jarman, H., McNamara, A., Risdon, H., & Jankowski, G. (2015). Dissemination of evidence-based body image interventions: A pilot study into the effectiveness of using undergraduate students as interventionists in secondary schools. *Body Image, 14*, 1–4.
Hay, P. (2013). A systematic review of evidence for psychological treatments in eating disorders: 2005-2012. *International Journal of Eating Disorders, 46*, 462–469.
Hays, W. L. (1994). *Statistics.* Fort Worth, TX: Harcourt Brace College Publishers.
Hedges, L. V. (1982). Estimation of effect size from a series of independent experiments. *Psychological Bulletin, 92*, 490–499. https://doi.org/10.1037/0033-2909.92.2.490.
Hox, J. J. (2010). *Multilevel analysis: Techniques and applications* (2nd ed.). New York, NY: Routledge.
Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry, 61*(3), 348–358.
Ioannidis, J. P. A., Munafò, M. R., Fusar-Poli, P., Nosek, B. A., & David, S. P. (2014). Publication and other reporting biases in cognitive sciences: Detection, prevalence and prevention. *Trends in Cognitive Sciences, 18*(5), 235–241. https://doi.org/10.1016/j.tics.2014.02.010.
Jacobi, C., Fittig, E., Bryson, S. W., Wilfley, D., Kraemer, H. C., & Taylor, C. B. (2011). Who is really at risk? Identifying risk factors for subthreshold and full syndrome eating disorders in a high-risk sample. *Psychological Medicine, 41*, 1939–1949.
Jilka, K. (2002). *The effects of a cognitive dissonance-based eating disorder prevention program in a sorority population* (Honors thesis)Trinity University.
Johnson, F., & Wardle, J. (2005). Dietary restraint, body dissatisfaction, and psychological distress: A prospective analysis. *Journal of Abnormal Psychology, 114*, 119–124. https://doi.org/10.1037/0021-843X.114.1.119.
Kilpela, L., Blomquist, K., Verzijl, C., Wilfred, S., Beyl, R., & Becker, C. (2016). The Body Project 4 All: A pilot randomized controlled trial of a mixed-gender dissonance-based body image program. *International Journal of Eating Disorders, 49*(6), 591–602.
Kilpela, L., DeBoer, L., Alley, M., Presnell, K., McGinley, J., & Becker, C. B. (2015). Distributed and condensed versions of a cognitive dissonance program: Comparative effects on eating disorder risk factors and symptoms. *Advances in Eating Disorders, 3*, 34–47.
Kilpela, L., Hill, K., Kelly, M., Elmquist, J., Ottoson, P., Keith, D., et al. (2014). Reducing eating disorder risk factors: A controlled investigation of a blended task-shifting/train-the-trainer approach to dissemination and implementation. *Behaviour Research and Therapy, 63*, 70–82.
Krishna, M. (2011). *The effectiveness of peer-led eating disorder prevention programs within the Dartmouth College sorority system* (Senior honors thesis)Dartmouth College.
Krysanski, V. L. (2005). *Investigation of a cognitive dissonance intervention versus a psychoeducational placebo control: testing the effectiveness of prevention programming for eating disorders*. Doctoral dissertation, University of North Dakota, Grand Forks, ND.
Linville, D., Cobb, E., Lenee-Bluhm, T., Lopez-Zeron, G., Gau, J., & Stice, E. (2015). The effectiveness of an eating disorder prevention intervention in primary care medical settings. *Behaviour Research and Therapy, 75*, 32–39.
Maas, C. J., Hox, J. J., & Lensvelt-Mulders, G. J. (2004). Longitudinal meta-analysis. *Quality and Quantity, 38*, 381–389.
Machin, A. L. (2008). *Reducing the risk of disordered eating among female college students: A test of alternative interventions* (Doctoral dissertation)University of North Texas.
Magnuson, A. (2012). Evaluation of an eating disorders prevention curriculum on eating attitudes and behaviors of female college students. *Dissertation Abstracts International, 72*.
Martinsen, M., Bahr, R., Børresen, R., Holme, I., Pensgaard, A. M., & Sundgot-Borgen, J. (2014). Preventing eating disorders among young elite athletes: a randomized controlled trial. *Medical Science Sports and Exercise, 46*(3), 435–447.
Matusek, J. A., Wendt, S. J., & Wiseman, C. V. (2004). Dissonance thin-ideal and didactic healthy behavior eating disorder prevention programs: Results from a controlled trial. *International Journal of Eating Disorders, 36*, 376–388.
McKnight, I. (2003). Risk factors for the onset of eating disorders in adolescent girls: Results of the McKnight longitudinal risk factor study. *American Journal of Psychiatry, 160*, 248–254.
McMillan, W., Stice, E., & Rohde, P. (2011). High- and low-level dissonance-based eating disorder prevention programs with young women with body image concerns: An experimental trial. *Journal of Consulting and Clinical Psychology, 79*, 129–134.
Mitchell, K. S., Mazzeo, S. E., Rausch, S. M., & Cooke, K. L. (2007). Innovative interventions for disordered eating: Evaluating dissonance-based and yoga interventions. *International Journal of Eating Disorders, 40*, 120–128.
Morris, S. B. (2008). Estimating effect sizes from pretest-posttest-control group designs. *Organizational Research Methods, 11*, 364–386. https://doi.org/10.1177/1094428106291059.
Pennesi, J., & Wade, T. D. (2017). Imagery rescripting and cognitive dissonance: A randomized controlled trial of two brief online interventions for women at risk of developing an eating disorder. *International Journal of Eating Disorders, 51*, 439–448.
Perez, M., Becker, C. B., & Ramirez, A. (2010). Transportability of an empirically supported dissonance-based prevention program for eating disorders. *Body Image, 7*(3), 179–186.
Presnell, K., Seidel, A., & Madeley, M. C. (2008). *Comparison of dissonance-only vs. dissonance plus healthy weight management eating disorder prevention interventions*. Poster presentation at The Association for Behavioral and Cognitive Therapies annual convention, New York City, NY
Ramirez, A. L., Perez, M., & Taylor, A. (2012). Preliminary examination of a couple-based eating disorder prevention program. *Body Image, 9*(3), 324–333.
Rodriguez, R., Marchand, E., Ng, J., & Stice, E. (2008). Effects of a cognitive-dissonance-based eating disorder prevention program are similar for Asian American, Hispanic, and white participants. *International Journal of Eating Disorders, 41*, 618–625.
Roehrig, M., Thompson, K., Brannick, M., & van den Berg, P. (2006). MA Dissonance-Based Eating Disorder Prevention Program:A Preliminary Dismantling Investigation. *International Journal of Eating Disorders, 39*, 1–10.
Rohde, P., Auslander, B. A., Shaw, H., Raineri, K. M., Gau, J. M., & Stice, E. (2014). Dissonance-based prevention of eating disorder risk factors in middle school girls: Results from two pilot trials. *International Journal of Eating Disorders, 47*, 483–494.
Rohde, P., Stice, E., & Marti, N. (2015). Development and predictive effects of eating disorder risk factors during adolescence: Implications for prevention efforts. *International Journal of Eating Disorders, 48*, 187–198.
Rohde, P., Stice, E., Shaw, H., Ohls, O. C., & Gau, J. M. (2017). Age effects in eating disorder baseline risk factors and prevention intervention effects. *International Journal of Eating Disorders, 50*, 1273–1280.
Santonastaso, P., Friederici, S., & Favaro, A. (1999). Full and partial syndromes in eating disorders: A 1-year prospective study of risk factors among female students. *Psychopathology, 32*, 50–56.
Scott-Sheldon, L., Carey, K., Elliott, J., Garey, L., & Carey, M. (2014). Efficacy of alcohol interventions for first-year college students: A meta-analytic review of randomized controlled trials. *Journal of Consulting and Clinical Psychology, 82*, 177–188.
Seidel, A., Presnell, K., & Rosenfield, D. (2009). Mediators in the dissonance eating disorder prevention program. *Behaviour Research and Therapy, 47*, 645–653.
Serdar, K., Kelly, N. R., Palmberg, A. A., Lydecker, J. A., Thornton, L., Tully, C. E., & Mazzeo, S. E. (2014). Comparing online and face-to-face dissonance-based eating disorder prevention. *Eating Disorders, 22*(3), 244–260.
Shaw, H., Rohde, P., & Stice, E. (2016). Participant feedback from peer-led, clinician-led, and Internet-delivered eating disorder prevention interventions. *International Journal of Eating Disorders, 49*, 1087–1092.
Smith, A., & Petrie, T. (2008). Reducing the risk of disordered eating among female athletes: A test of alternative interventions. *Journal of Applied Sport Psychology, 20*, 392–407.
Stewart, D. A., Carter, J. C., Drinkwater, J., Hainsworth, J., & Fairburn, C. G. (2001). Modification of eating attitudes and behavior in adolescent girls: A controlled study. *International Journal of Eating Disorders, 29*, 107–118.
Stice, E., Butryn, M., Rohde, P., Shaw, H., & Marti, N. (2013). An effectiveness trial of a new enhanced dissonance eating disorder prevention program among female college students. *Behaviour Research and Therapy, 51*, 862–871.
Stice, E., Chase, A., Stormer, S., & Appel, A. (2001). A randomized trial of a dissonance-based eating disorder prevention program. *International Journal of Eating Disorders, 29*, 247–262.
Stice, E., Durant, S., Rohde, P., & Shaw, H. (2014). Effects of a prototype internet dissonance-based eating disorder prevention program at 1- and 2-year follow-up. *Health Psychology, 33*, 1558–1567. https://doi.org/10.1037/hea0000090.
Stice, E., Gau, J., Rohde, P., & Shaw, H. (2017). Risk factors that predict future onset of each DSM-5 eating disorder: Predictive specificity in high-risk adolescent females. *Journal of Abnormal Psychology, 126*, 38–51.
Stice, E., Marti, C. N., & Cheng, Z. H. (2014). Effectiveness of a dissonance-based eating disorder prevention program for ethnic groups in two randomized controlled trials. *Behaviour Research and Therapy, 55*, 54–64.
Stice, E., Marti, C. N., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *Journal of Abnormal Psychology, 122*, 445–457.
Stice, E., Marti, C. N., Spoor, S., Presnell, K., & Shaw, H. (2008). Dissonance and healthy weight eating disorder prevention programs: Long-term effects from a randomized efficacy trial. *Journal of Consulting and Clinical Psychology, 76*, 329–340.
Stice, E., Marti, N., Shaw, H., & O'Neil, K. (2008). General and program-specific moderators of two eating disorder prevention programs. *International Journal of Eating Disorders, 41*, 611–617.
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*, 206–217.
Stice, E., Presnell, K., Gau, J., & Shaw, H. (2007). Testing mediators of intervention effects in randomized controlled trials: An evaluation of two eating disorder prevention programs. *Journal of Consulting and Clinical Psychology, 75*, 20–32.
Stice, E., Rohde, P., Butryn, M. L., Shaw, H., & Marti, C. N. (2015). Effectiveness trial of a selective dissonance-based eating disorder prevention program with female college students: Effects at 2-and 3-year follow-up. *Behaviour Research and Therapy, 71*, 20–26. https://doi.org/10.1016/j.brat.2015.05.012.
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*, 907–916.
Stice, E., Rohde, P., Durant, S., Shaw, H., & Wade, E. (2013). Effectiveness of peer-led dissonance eating disorder prevention groups: Results from two randomized trials. *Behaviour Research and Therapy, 51*, 197–206.
Stice, E., Rohde, P., Gau, J., & Shaw, H. (2009). An effectiveness trial of a dissonance-based eating disorder prevention program for high-risk adolescent girls. *Journal of Consulting and Clinical Psychology, 77*, 825–834.
Stice, E., Rohde, P., Gau, J., & Shaw, H. (2012). Effect of a dissonance-based prevention program on risk for eating disorder onset in the context of eating disorder risk factors. *Prevention Science, 13*, 129–139.
Stice, E., Rohde, P., Shaw, H., & Gau, J. (2011). An effectiveness trial of a selected dissonance-based eating disorder prevention program for female high school students: Long-term effects. *Journal of Consulting and Clinical Psychology, 79*, 500–508.
Stice, E., Rohde, P., Shaw, H., & Gau, J. (2017a). An experimental therapeutics test of whether adding dissonance-induction activities improves the effectiveness of a

selected obesity and eating disorder prevention program. *International Journal of Obesity, 42*, 462–468.
Stice, E., Rohde, P., Shaw, H., & Gau, J. (2017b). Clinician-led, peer-led, and Internet-delivered dissonance-based eating disorder prevention programs: Acute effectiveness of these delivery modalities. *Journal of Consulting and Clinical Psychology, 85*, 883–895.
Stice, E., Rohde, P., Shaw, H., & Marti, C. N. (2012). Efficacy trial of a selected prevention program targeting both eating disorder symptoms and unhealthy weight gain among female college students. *Journal of Consulting and Clinical Psychology, 80*, 164–170.
Stice, E., Rohde, P., Shaw, H., & Marti, C. N. (2013). Efficacy trial of a selective prevention program targeting both eating disorders and obesity among female college students: 1- and 2-year follow-up effects. *Journal of Consulting and Clinical Psychology, 81*, 183–189.
Stice, E., Shaw, H., Burton, E., & Wade, E. (2006). Dissonance and healthy weight eating disorder prevention programs: A randomized efficacy trial. *Journal of Consulting and Clinical Psychology, 74*, 263–275.
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*, 233–257.
Stice, E., Trost, A., & Chase, A. (2003). Healthy weight control and dissonance-based eating disorder prevention programs: Results from a controlled trial. *International Journal of Eating Disorders, 33*, 10–21.
Stice, E., & Van Ryzin, M. (2019). A prospective test of the temporal sequencing of risk factor emergence in the dual pathway model of eating disorders. *Journal of Abnormal Psychology, 128*, 119–128.
Stice, E., Yokum, S., & Waters, A. (2015). Dissonance-based eating disorder prevention program reduces reward region response to thin models: How actions shape valuation. *PLoS One*. https://doi.org/10.1371/journal.pone.0144530.
Swanson, S. A., Crow, S. J., le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlates of eating disorders in adolescents: Results from the National Comorbidity Survey Replication Adolescent Supplement. *Archives of General Psychiatry, 68*, 714–723.
Trost, A. S. (2006). *The Healthy Image Partnership (HIP) Parents Program: The role of parental involvement in eating disorder prevention.* (doctoral dissertation).
Turk, M. W., Yang, K., Hravnak, M., Sereika, S. M., Ewing, L. J., & Burke, L. E. (2009). Randomized clinical trials of weight loss maintenance: A review. *Journal of Cardiovascular Nursing, 24*, 58–80.
Van Diest, A. M. K., & Perez, M. (2013). Exploring the integration of thin-ideal internalization and self-objectification in the prevention of eating disorders. *Body Image, 10*, 16–25.
Viechtbauer, W. (2010). Conducting meta-analyses in R with the metafor package. *Journal of Statistical Software, 36*, 1–48. Retrieved from http://www.jstatsoft.org/v36/i03/.
Wade, T., George, W., & Atkinson, M. (2009). A randomized controlled trial of brief interventions for body dissatisfaction. *Journal of Consulting and Clinical Psychology, 77*, 845–854.
Warren, C., Gleaves, D., Cepeda-Benito, A., Fernandez, M., & Rodriguez, R. S. (2005). Ethnicity as a protective factor against internalization of a thin ideal and body dissatisfaction. *International Journal of Eating Disorders, 37*, 241–249.
Watson, H. J., Joyce, T., French, E., Willan, V., Kane, R. T., Tanner-Smith, E., ... Egan, S. J. (2016). Prevention of eating disorders: A systematic review of randomized, controlled trials. *International Journal of Eating Disorders. Advance*. https://doi.org/10.1002/eat.22577 on-line publication.
Weiss, K. R., & Wertheim, E. H. (2005). An evaluation of a primary prevention program for disordered eating in adolescent girls: Examining responses of high- and low-risk girls. *Eating Disorders, 13*, 143–156.
Wiseman, C. V., Sunday, S. R., Bortolotti, F., & Halmi, K. A. (2004). Primary prevention of eating disorders through attitudinal change: A two country comparison. *Eating Disorders, 12*, 241–250.
Wolfe, J. L. (1992). *Applying cognitive dissonance within a personal health curriculum to reduce risk factors associated with the development of eating disorders in a university population.* Doctoral Dissertation)University of Georgia.

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A meta-analytic review of trials that tested whether eating disorder prevention programs prevent eating disorder onset

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ABSTRACT

This report provides a review of randomized controlled trials that tested whether an eating disorder prevention program significantly reduced future onset of eating disorders, which is important because eating disorders are common and result in marked functional impairment. We identified 15 trials involving 5080 participants (mean ages ranging from 14.5 to 22.3) that reported 19 tests of whether selective eating disorder prevention programs significantly reduced future onset of eating disorders relative to some type of minimal control condition or a credible alternative intervention. Healthy lifestyle modification prevention programs, dissonance-based prevention programs, and a self-esteem/self-efficacy prevention program significantly reduced future onset of eating disorders, though the later was only evaluated in one trial. Psychoeducational, cognitive behavioral, behavioral weight gain, interpersonal, and family-therapy-based prevention programs did not significantly reduce future onset of eating disorders. The average prevention effect size was statistically significant ($OR = 1.64$, 95% CI = [1.09, 2.46], $t = 2.54$, $p = .020$) and there was heterogeneity in effect sizes (Q [18] = 35.96, $p = .007$). Prevention effects were significantly larger for trials that recruited participants with elevations on a single risk factor versus with elevations in multiple risk factors and for healthy lifestyle modification prevention programs versus cognitive behavioral prevention programs, though the remaining examined factors did not moderate intervention effect sizes (e.g., risk of bias).

The fact that lifestyle modification and dissonance-based prevention programs significantly reduced future onset of eating disorders in multiple trials, producing a 54% to 77% reduction in future eating disorder onset implies that broadly implementing these prevention programs could reduce the population prevalence of eating disorders.

1. Introduction

Representative data from the US, Italy, and Australia suggest that 13% of females and 6% of males experience anorexia nervosa, bulimia nervosa, binge eating disorder and other specified and unspecified feeding and eating disorders, such as subthreshold levels of these disorders and purging disorder (Allen, Byrne, Oddy, & Crosby, 2013; Dakanalis et al., 2017; Stice, Marti, & Rohde, 2013). For both females and males eating disorders are characterized by functional impairment, poorer academic performance, emotional distress, lower mental health quality of life, and risk for future obesity, depression, suicide, and mortality (Allen et al., 2013; Arcelus, Mitchell, Wales, & Nielsen, 2011; Claydon & Zullig, 2019; Stice, Marti, & Rohde, 2013). Indeed, eating disorders show stronger relations to mortality, suicide attempts, and functional impairment than other psychiatric disorders, including schizophrenia, major depression, and bipolar disorder (Crow & Smiley, 2010) and are the second leading cause of mental health disability for adolescent girls and young women (Begg et al., 2007). Unfortunately, 80% of individuals with eating disorders do not receive treatment (Swanson, Crow, le Grange, Swendsen, & Merikangas, 2011) and evidence-based treatments produce symptom remission for only 30–40% of patients (Fairburn et al., 2015; McIntosh et al., 2016; Wonderlich et al., 2014). Thus, broadly implementing effective prevention programs might be a more effective way to address this public health problem. Prevention programs may be particularly effective for eating disorders because the peak period for onset occurs between the ages of 16 to 19 (Hudson, Hiripi, Pope, & Kessler, 2007; Stice, Marti, & Rohde, 2013), suggesting that if effective prevention programs were broadly

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implemented during adolescence, they could reduce the population prevalence of eating disorders.

The goal of this report is to review randomized controlled trials that tested whether an eating disorder prevention program significantly reduced future onset of eating disorders. To our knowledge, no previous review has summarized findings from trials that tested for such prophylactic prevention effects, potentially because few trials were designed to detect a reduction in future onset of eating disorders. These trials are difficult to conduct because it is necessary to (1) recruit a large sample, (2) conduct diagnostic interviews with all participants with assessors who are blinded to the condition to which participants were assigned, and (3) assess the cohort over a long follow-up period to permit a high enough incidence of eating disorder onset in the control condition to detect a significant reduction in the intervention condition. Large sample sizes and long follow-ups are necessary for sufficient power because inferential tests for detecting intervention effects on dichotomous outcomes, such as onset of eating disorders, have less power than inferential tests for detecting change in continuous outcomes, such as an eating disorder symptom composite measure and because the low incidence of eating disorders reduces sensitivity (Cohen, 1988). One meta-analysis sought to summarize the effects of eating disorder prevention programs on future onset of eating disorders at universities (Harrer et al., 2019; Harrer, Cuijpers, Furukawa, & Ebert, 2019), but did not include all studies involving college students that met the stated inclusion criteria, did not include several studies that included younger adolescents, and was not able to include several recently published trials.

We first review findings from prospective studies that have identified risk factors that predicted future onset of eating disorders because this knowledge should inform the optimal content of prevention programs. We then describe the methods we used to conduct this literature review and the variables that were hypothesized to correlate with the magnitude of the effects of the prevention programs on eating disorder onset (i.e., moderators). Next, we provide a narrative review of randomized trials that tested for a reduction in future onset of eating disorders, noting strengths and limitations of each trial. We then report the results of the meta-analysis that characterized the average eating disorder prevention effect achieved in these trials and examined moderators that correlate with larger eating disorder prevention effects, which should inform efforts to broadly implement the most effective prevention programs. Because the small number of identified trials limited power to identify moderators that correlate with larger prevention effects, the moderator analyses should be considered preliminary. Last, we discuss implications of these findings for efforts to prevent eating disorders and important research directions.

1.1. Risk factors that predict future onset of eating disorders

Several prospective studies have identified risk factors that predict future onset of any eating disorder. We focus on studies that used diagnostic interviews, as this is the gold-standard method of diagnosing eating disorders. We conducted a systematic review of several electronic databases (e.g., *PsycInfo, Web of Science, and MedLine*) to identify prospective studies that have sought to identify variables that predicted future onset of any eating disorder or one of the four specific types of eating disorders and reviewed findings from published reviews that focused on this topic (e.g., Stice, 2016). All of the identified studies appeared to have had sufficient power based on the sample size and the incidence of eating disorder onset over follow-up, as each study was able to identify baseline variables that did significantly predict future onset of eating disorders. Social pressure for thinness, pursuit of the thin beauty ideal, self-objectification, body dissatisfaction, parental overweight, dieting, negative affect, social support deficits, alcohol use, and female sex predicted future onset of any eating disorder (Allen, Byrne, Oddy, Schmid, & Crosby, 2014; Dakanalis et al., 2017; Ghaderi & Scott, 2001; Jacobi et al., 2011; McKnight, 2003; Rohde, Stice, & Marti, 2015; Santonastaso, Friederici, & Favaro, 1999). Risk factor findings are summarized in Table 1. (See Fig. 1.)

Only a few studies were powered to identify risk factors that predicted future onset of specific eating disorders, which include anorexia nervosa, bulimia nervosa, binge eating disorder, and purging disorder. Low body mass index (BMI), weight suppression, weight and shape overvaluation, fear of weight gain, and impaired psychosocial functioning predicted future onset of threshold or subthreshold anorexia nervosa (Stice, Desjardins, Rohde, & Shaw, in press; Stice, Gau, Rohde, & Shaw, 2017; Stice, Rohde, Shaw, & Desjardins, 2020). It is critical to note that several variables that are often considered established risk factors for anorexia nervosa were found to not predict onset of this eating disorder in these prospective studies, including early puberty, perceived pressure for thinness, thin-ideal internalization, body dissatisfaction, dieting, fasting, excessive exercise, compensatory behaviors, binge eating, feeling fat, and social support deficits. It is tempting to attribute these null findings to insufficient power, but the prospective studies that generated these null findings identified several factors that did predict future onset of anorexia nervosa, suggesting adequate power. An alternative possibility is that the measures used to assess those constructs were not sufficiently reliable, but most of those variables have predicted future onset of other eating disorders (as reviewed below). Thus, a more parsimonious interpretation of these null findings is that these variables do not increase risk for anorexia nervosa. Perpetuating beliefs about putative risk factors for specific eating disorders that have not received empirical support in prospective studies

Table 1
Eating disorder risk factors.

Eating disorder risk factors	Eating disorder type				
	Anorexia nervosa	Bulimia nervosa	Binge eating disorder	Purging disorder	Any eating disorder
Social					
Social pressure for thinness		x			x
Pursuit of the thin beauty ideal		x	x	x	x
Social support deficits		x			x
Impaired psychosocial functioning	x	x	x	x	
Psychological					
Overvaluation of weight and shape	x	x		x	
Self-objectification					x
Body-dissatisfaction		x	x	x	x
Feeling fat		x	x	x	
Fear of weight gain	x	x	x	x	
Negative affect		x	x	x	x
Low interoceptive awareness		x			
Biological					
Parental overweight					x
Overeating		x	x	x	
Binge eating		x	x	x	
Dieting		x	x	x	x
Weight suppression	x	x		x	
Low BMI	x				
Compensatory behaviors		x		x	
Alcohol use		x			x
Female sex					x

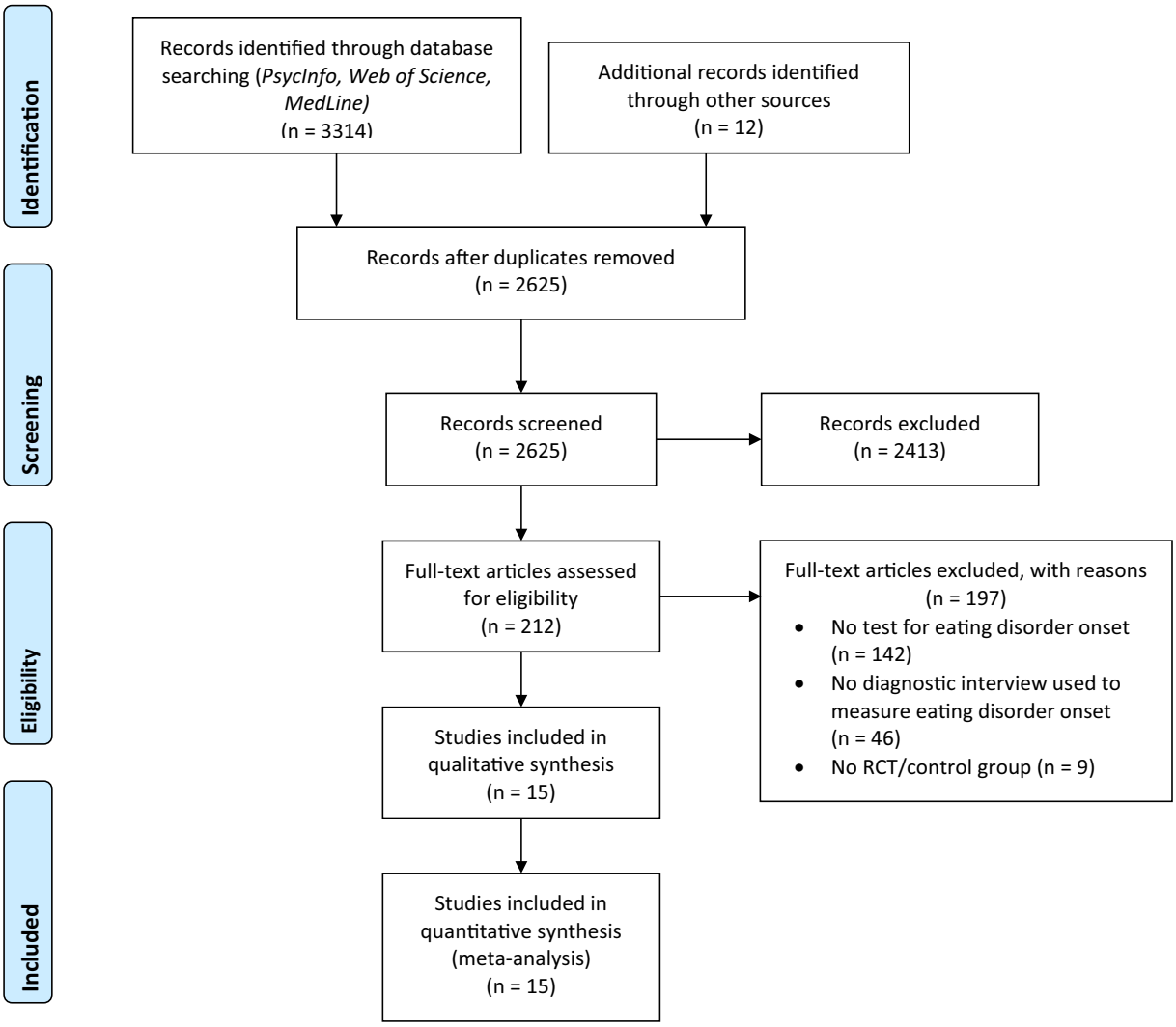


Fig. 1. Flow diagram.

has the potential to hinder needed etiologic studies and can result in prevention and treatment interventions that are ineffective.

Social pressure for thinness, pursuit of the thin beauty ideal, weight and shape overvaluation, fear of weight gain, body dissatisfaction, feeling fat, overeating, binge eating, dieting, weight suppression, compensatory behaviors, negative affect, social support deficits, impaired psychosocial functioning, alcohol use, and low interoceptive awareness predicted future onset of threshold or subthreshold bulimia nervosa (Killen et al., 1996; Patton, Johnson-Sabine, Wood, Mann, & Wakeling, 1990; Patton, Selzer, Coffey, Carlin, & Wolfe, 1999; Stice, Davis, Miller, & Marti, 2008; Stice et al., in press; Stice et al., 2017; Stice, Marti, & Durant, 2011; Stice, Rohde, Shaw, & Desjardins, 2020). Again, it is important to note variables that did not predict future onset of bulimia nervosa in these studies, which included perfectionism, maturity fears, interpersonal distrust, BMI, peer dieting, exercise, and parental separation.

Thin-ideal internalization, weight and shape overvaluation, fear of weight gain, body dissatisfaction, feeling fat, dieting, overeating, binge eating, negative affect, and impaired psychosocial functioning predicted future onset of threshold or subthreshold binge eating disorder (Stice et al., 2017; Stice et al., in press; Stice, Rohde, Shaw, & Desjardins, 2020). BMI, exercise, weight suppression, fasting, and compensatory weight control behaviors did not predict future onset of binge eating disorder in these studies.

Thin-ideal internalization, weight and shape overvaluation, fear of weight gain, body dissatisfaction, feeling fat, dieting, weight suppression, overeating, binge eating, compensatory behaviors, negative affect, and impaired social functioning predicted future onset of purging disorder (Stice et al., 2017; Stice et al., in press; Stice, Rohde, Shaw, & Desjardins, 2020). Social pressure to be thin, BMI, and exercise did not predict future onset of purging disorder in these studies.

Thus, emerging data suggest that the risk factors for the specific eating disorders are somewhat distinct. Nonetheless, pursuit of the thin ideal, weight and shape overvaluation, body dissatisfaction, fear of weight gain, dieting, weight suppression, feeling fat, overeating, binge eating, negative affect, and impaired psychosocial functioning predicted future onset of eating disorders in at least three prospective studies or have predicted future onset of at least three specific eating disorders. Results suggest that prevention programs that reduce risk factors with the greatest evidence-base, such as these, may prove effective in decreasing future eating disorder onset. However, these data also suggest that many prevention programs target variables that are not established risk factors for anorexia nervosa and that it might be necessary to target different risk factors and at-risk populations to prevent this particular eating disorder. Although targeting risk factors that have been found to predict onset of all four specific types of eating disorders might be optimal for preventing all of these disorders, which include negative affect, impaired psychosocial functioning, weight and shape overvaluation, and fear of weight gain, none of the prevention programs evaluated in trials that we identified target these risk factors.

2. Methods

2.1. Search strategy

To identify relevant trials, the first two authors conducted a database search to retrieve articles was performed on *PsycInfo, Web of Science, and MedLine* for the years January 1980 – May 2020 using the following keywords: eating disorder, anorexia, bulimia, binge eating, prevention, intervention, and onset. Further, two previously published systematic reviews by Stice, Shaw, and Marti (2007) and Watson et al. (2016) were examined for reports not captured in the database search. We also examined reference lists of relevant literature for additional reports and contacted active eating disorder prevention researchers for any relevant, unpublished data. Studies written in other languages would have been included, but no published or unpublished studies that met our inclusion criteria were found in languages other than English.

Studies were included in this review if they met the following criteria: (1) the study aimed to evaluate an eating disorder prevention program, (2) the program was evaluated in a randomized controlled trial (RCT) that contained a minimal intervention control condition (e.g., assessment-only or waitlist control condition) or a credible alternative intervention (e.g., expressive writing or a health education comparison condition), and (3) eating disorder incidence over follow-up in each condition was assessed using diagnostic interviews.

A total of 3326 studies were identified from the literature search as potentially eligible for inclusion. The abstracts and measures sections of each article were screened, articles that met at least two points of eligibility (RCT and tested an eating disorder prevention program) were read to further determine if all criteria were met. Studies that met the outlined eligibility criteria were then reviewed to finalize the sample of studies.

2.2. Data extraction

Data was extracted from each report to describe study and sample characteristics (e.g., intervention theory, delivery modality, duration, length of follow up). A coding manual was used for data extraction. Data was extracted and coded by the second author and carefully reviewed and checked by the first author. When discrepancies arose in coding, the authors reviewed each study together and arrived at a consensus (consulting with the authors of the retrieved articles in some instances). Inter-rater agreement was 100% after the consensus discussions.

2.3. Research quality assessment

The Cochrane Collaboration Risk of Bias criteria were used to evaluate the risk of bias for each trial. The Risk of Bias criteria measures research quality in five domains: randomization process, deviations from intended interventions, missing outcome data, outcome measurement, and selective outcome reporting (Higgans et al., 2011). Within these domains, specific factors such as randomization sequence, allocation concealment, blinding of assessor, and statistical analyses, are assessed. For each domain, the risk of bias was categorized as 'high', 'low', or 'some concerns'. Under the 'Deviations from Intended Intervention' domain, criteria related to blinding of participants and intervention facilitators were not included, as this type of blinding was not possible for the interventions included in this meta-analysis. Risk of Bias in this domain was determined by use of appropriate statistical analyses (i.e. intention-to-treat analyses). Intention-to-treat analyses was defined as the inclusion of all randomized participants in the final outcome analysis. Missing outcome data were considered 'low risk' in cases when attrition was below 20%, participant flowchart provided clear reporting of drop-outs, and reasons for dropout were not related to the study condition to which they were randomized, or trials reported no significant differences in baseline characteristics of study drop-outs versus non-drop-outs. Studies were given a categorization as High Risk of Bias if at least one domain was considered "high risk" and another domain was considered "some concerns". Risk of Bias was assessed by the second author and carefully reviewed by the first author. When discrepancies arose, the authors reviewed the published reports together and arrived at a consensus.

2.4. Meta-analytic statistical methods

2.4.1. Effect size estimation

Effect sizes were based on odds ratios from the final assessment point that were transformed into their natural log for analysis in the models described below. Odds ratios were coded so that a positive value reflects improvement in the outcome (i.e., interventions resulted in fewer eating disorders than control groups). Odds ratios of 1.68 (0.60), 3.47 (0.29), and 6.71 (0.15) are benchmarks for small, medium, and large effect sizes respectively (Chen, Cohen, & Chen, 2010). Effect sizes and effect size sampling variance estimates were computed using the R metafor package (Viechtbauer, 2010) which implements methods described in Lipsey

Table 3
Odds ratio and number needed to treat for prevention programs versus controls/alternative interventions.

Authors (intervention vs. control/alternative intervention) and year	Odds Ratio	Number needed to treat
Favaro, Zanetti, Huon, Sanosanto (Intervention vs. control) 2005	2.22	17.46
Jacobi, Volker, Trockel, & Taylor (Student bodies+ vs control) 2012	7.92	16.00
Stice, Rohde, Shaw, & Gau (Body project vs control) 2011	1.38	61.77
Stice, Rohde, Butyrn, Shaw, & Marti (Body project vs control) 2015	1.08	108.34
Stice, Rohde, Shaw, & Gau (Clinician-led *Body Project* vs control) 2020	0.88	53.72
Stice, Rohde, Shaw, & Gau (Peer-led *Body Project* vs control) 2020	2.36	10.85
Stice, Rohde, Shaw, & Gau (Ebodyproject vs control) 2020	1.18	44.41
Stice, Rohde, Shaw, & Gau (Clinician-led *Body Project* vs peer-led *Body Project*) 2020	0.37	9.03
Stice, Rohde, Shaw, & Gau (Clinician-led *Body Project* vs Ebodyproject) 2020	0.75	24.31
Stice, Rohde, Shaw, & Gau (Peer-led *Body Project* vs Ebodyproject) 2020	2.00	14.36
Jacobi et al. (Parents Act Now vs control) 2018	0.74	–
Martinsen et al. (Intervention vs control) 2014	22.80	24.00
Stice, Marti, Spoor, Presnell, & Shaw (Body Project vs control) 2008	2.50	11.05
Stice, Marti, Spoor, Presnell, & Shaw (Healthy Weight vs control) 2008	2.88	10.05
Stice, Marti, Spoor, Presnell, & Shaw (Body Project vs Expressive Writing) 2008	1.28	55.27
Stice, Marti, Spoor, Presnell, & Shaw (Healthy Weight vs Expressive Writing) 2008	1.47	36.90
Stice, Marti, Spoor, Presnell, & Shaw (Body Project vs Healthy Weight) 2008	0.87	111.00
Stice, Marti, Spoor, Presnell, & Shaw (Expressive Writing vs control) 2008	1.96	13.80
Taylor et al. (Student Bodies vs control) 2006	0.69	20.06
Taylor et al. (Image and Mood vs control) 2016	1.40	14.98
Ghaderi, Stice, Andersson, Persson, & Allzén (Virtual Body Project vs control) 2020	4.73	7.93
Tanofsky-Kraff et al. (Intervention vs control) 2014	4.27	16.33
Stice, Rohde, Shaw, & Marti (Healthy Weight vs control) 2013	2.79	18.72
Stice, Rohde, Shaw, & Gau (Project Health vs Healthy Weight) 2018	0.96	1063.75
Stice, Rohde, Shaw, & Gau (Healthy Weight vs control) 2018	3.17	19.17
Stice, Rohde, Shaw, & Gau (Project Health vs control) 2018	3.06	19.52
Lowe, Stice, Butryn, Sawrer (intervention vs. assessment control)	0.54	8.78

and Wilson (2001). (See Table 3.)

2.4.2. Intervention group contrasts

Effect sizes were computed for prevention programs versus minimal intervention control conditions and for prevention programs versus credible alternative interventions. In a preliminary analysis, we compared effect sizes for the interventions versus minimal intervention control conditions and interventions versus credible alternative interventions and did not find a significant difference. Thus, we limited the effect sizes included in the meta analytic models to prevention programs versus minimal intervention control conditions with the exception of two studies that did not have a minimal intervention control condition (Ghaderi, Stice, Andersson, Enö Persson, & Allzén, 2020; Tanofsky-Kraff et al., 2014) and thus included effect sizes based on prevention programs versus alternative interventions.

2.4.3. Operationalization and coding of effect size moderators

Table 2 defines each moderator that we extracted from the studies and characterizes the distribution of the moderators in the identified studies. We attempted to code average attendance and homework completion rates, but the identified studies did not report these data in a consistent form, preventing analyses of those moderators. There were missing data on several moderators and homework assigned had only one instance of one of the levels of that moderator, prompting us to omit it as well. We hypothesized that prevention effects would be larger for trials that evaluated longer versus shorter interventions and included home exercises, as both should result in greater uptake of the concepts and skills taught in the prevention program. We hypothesized that effects would be larger for trials with longer follow-up periods, as this should increase the incidence of eating disorder onset and thus power to detect effects. Based on the pattern of findings revealed in the narrative review, we hypothesized that prevention effects would be larger for trials that delivered the prevention program in-person versus via the internet. We also hypothesized that prevention effects would be larger for trials that recruited participants with elevations on a single risk factor versus with elevations in multiple risk factors, as the greater heterogeneity in risk potentially introduced more noise to the data, which could reduce power. In addition, we hypothesized that prevention effects would be larger for dissonance-based and lifestyle modification-based prevention programs than for cognitive-behavioral prevention programs (referred to as intervention theory). Finally, we hypothesized that prevention effects would be smaller for trials with a lower risk of bias. (See Table 4.)

2.4.4. Meta-analysis models

All effect size analyses were modeled using multilevel random-effects models that were implemented using the R metafor package (Viechtbauer, 2010). Models were fit using Restricted Maximum Likelihood (REML) which corrects downward bias in the maximum likelihood estimates of the covariance matrix of the random effects when the number of studies is small as is the case in the present meta-analysis (Schwarzer, Carpenter, & Rücker, 2015). The Knapp and Hartung (2003) method was used, which produces more accurate standard errors and thus more accurate inferential statistics and confidence intervals (van Aert & Jackson, 2019) in addition to reducing the false positive rate (IntHout, Ioannidis, & Borm, 2014).

The multilevel random-effect model is a variance-known multilevel model that accounts for non-independence in effect sizes from a common source (Hox, 2010), which is the case in the present meta-analysis wherein independent treatment groups are compared with a common control group in some of the trials. The effect size variance is based on the effect size variance described above and variation in other model parameters is estimated in a multilevel model framework. The covariance of dependent effect sizes is unknown in meta-analysis but is captured in overestimating study-level covariation (Van den Noortgate, López-López, Marin-Martinez, & Sánchez-Meca, 2013). We initially fit an unconditional model that provides an overall effect size. We used the unconditional model to examine effect size heterogeneity using the Q statistic (Cochran, 1954) that evaluates the null hypotheses that effect size variance is zero. In the event of significant heterogeneity, we next examined moderators that could potentially explain variability in bivariate regression models. Intervention theory contained three levels (cognitive behavioral, dissonance, and healthy lifestyle modification); we dummy-coded for dissonance and healthy lifestyle modification, using cognitive behavioral programs as the reference group. Continuous moderators were standardized (i.e., z score) prior to fitting models so that continuous moderators were on the same scale. Continuous moderators were evaluated in linear and quadratic models and, in the event of a significant quadratic effect, the quadratic model would be preferred; there were no significant quadratic effects and the continuous moderators were thus fit in linear models. Significant moderator effects were probed by estimating marginal odds ratios (i.e., odds ratios predicted by the model). Categorical moderators were estimated at each level of the moderator; continuous moderators were estimated at 1 *SD* above and below the mean. In addition, we included all moderators in a multivariate model.

We assessed publication bias using Egger's regression test for funnel plot asymmetry with effect sizes pooled at the study level, which is preferable to visual inspection of funnel plots (Simmonds, 2015). We used the sample size as the independent variable given limitations using standard errors for the natural log of the odds ratio due to its association with the natural log of the odds ratio (Peters, Sutton, Jones, Abrams, & Rushton, 2006). The number needed to treat (NNT) was calculated using the NNT function in the dmeatar package (Harrer, Adam, et al., 2019; Harrer, Cuijpers, et al., 2019) which implements methods described in Kraemer and Kupfer (2006). NNT reflects the number of participants who must complete an intervention to prevent the onset of one eating disorder (Kraemer & Kupfer, 2006).

2.5. Results of the literature search

Fifteen trials were identified that tested for prevention of future onset of eating disorders. These 15 trials evaluated the prevention effects with 5080 participants from the United States and Europe. Thirteen trials included only females; two trials included both males and females. Studies focused primarily on adolescent and young adult populations with mean ages ranging from 14.5 to 22.3. Six of the 14 trials did not report racial/ethnic demographic data for participants, which limited the ability to understand potential differences in response to interventions. Of the 9 trials that did report these demographics, the racial distribution was: 65.2% white, 13.1% Hispanic/Latino, 10.4% Asian/Pacific Islander, 5.4% Black or African-American, 2.6% other or more than one race, 1.6% American Indian/Alaskan Native, and 0.2% Native Hawaiian/Pacific Islander. This composition is a reasonable representation of the college and high school populations from which the trials sampled. No trial has tested whether an eating disorder prevention program produces similar reductions in eating disorder onset for different ethnic groups.

In the 15 identified trials, 19 contrasts tested whether an eating disorder prevention program reduced future onset of eating disorders relative to a comparison condition. We categorized prevention programs as universal, selective, or indicated using the Institute of Medicine's levels of classification (Mrazek & Haggerty, 1994). An intervention was considered universal if it targeted the entire population, selective if it targeted a subgroup of the population at elevated risk (variables considered risk factors are summarized in Table 1), and indicated if it targeted participants with symptoms of an eating disorder below the diagnostic threshold. All prevention programs were considered selective based on the reported inclusion criteria, as none targeted the entire population or required that all participants reported eating disorder symptoms. Three reports described their prevention programs as indicated, however upon examination of the intervention theory and

Table 2
Study characteristics.

Study	Sample size	Sample (age, % female, % White)	Study conditions (1. intervention, 2. control)	Length of follow-up (months)	Outcome measures	Intervention duration (hours)	Intervention format	Intervention theory	Risk of bias
Favaro et al. (2005)	138	M = 17.0 (SD = 1.1), 100%, N/A	1. Psychoeducational intervention 2. Assessment only control	12	SCID	12	Classroom, in-person	Psychoeducation	High
Jacobi et al., 2012	126	M = 22.3 (SD = 2.9), 100%, N/A	1. Student bodies+ 2. Waitlist control	6	EDE	5	Internet	Cognitive Behavioral	High
Stice et al. (2011)	306	M = 15.7 (SD = 1.1), 100%, 82%	1. Body project 2. Educational brochure	36	EDDI	4	group based; in-person	Dissonance Based	Low
Stice, Rohde, et al. (2015)	408	M = 21.6 (SD = 5.6), 100%, 58%	1. Body project 2. Educational brochure	36	EDDI	4	Group based; In-person	Dissonance Based	Low
Stice, Rohde, Shaw, and Gau (2020)	680	M = 22.2 (SD = 7.1), 100%, 60%	1. Clinician-led *Body Project* 2. Educational video	48	EDDI	4	Group based; in-person	Dissonance Based	Some Concerns
Stice, Rohde, Shaw, and Gau (2020)	680	M = 22.2 (SD = 7.1), 100%, 60%	1. Peer-led *Body Project* 2. Educational video	48	EDDI	4	Group based; in-person	Dissonance Based	Some Concerns
Stice, Rohde, Shaw, and Gau (2020)	680	M = 22.2 (SD = 7.1), 100%, 60%	1. Ebodyproject 2. Educational video	48	EDDI	4	Self-guided, Internet	Dissonance Based	Some Concerns
Stice, Rohde, Shaw, and Gau (2020)	680	M = 22.2 (SD = 7.1), 100%, 60%	1. Clinician-led *Body Project* 2. Peer-led *Body Project*	48	EDDI	4	Group based; in-person	Dissonance Based	Some Concerns
Stice, Rohde, Shaw, and Gau (2020)	680	M = 22.2 (SD = 7.1), 100%, 60%	1. Clinician-led *Body Project* 2. Ebodyproject	48	EDDI	4	Group based; in-person	Dissonance Based	Some Concerns
Stice, Rohde, Shaw, and Gau (2020)	680	M = 22.2 (SD = 7.1), 100%, 60%	1. Peer-led *Body Project* 2. Ebodyproject	48	EDDI	4	Group based; in-person/ Internet	Dissonance Based	Some Concerns
Jacobi et al. (2018)	66	M = 13.8 (SD = 1.1), 100%, N/A	1. Parents Act Now 2. Wait list control	12	EDE	999	Family based; Internet	Family Based Treatment	High
Martinsen et al. (2014)	577	M = 16.5 (SD = 0.3), 33.7%, N/A	1. Intervention 2. Assessment only control	9	EDE	6	Classroom, in-person	Self Esteem/Self Efficacy Enhancement	High
Stice et al. (2008)	481	M = 17.0 (SD = 1.4), 100%, 58%	1. Body project 2. Assessment only control	36	EDDI	3	Group based; In-person	Dissonance Based	Low
Stice et al. (2008)	481	M = 17.0 (SD = 1.4), 100%, 58%	1. Healthy weight 2. Assessment only control	36	EDDI	3	Group based; In-person	Lifestyle Modification	Low
Stice et al. (2008)	481	M = 17.0 (SD = 1.4), 100%, 58%	1. Body project 2. Expressive writing	36	EDDI	3	Group based; In-person	Dissonance Based	Low
Stice et al. (2008)	481	M = 17.0 (SD = 1.4), 100%, 58%	1. Healthy weight 2. Expressive writing	36	EDDI	3	Group based; In-person	Lifestyle Modification	Low
Stice et al. (2008)	481	M = 17.0 (SD = 1.4), 100%, 58%	1. Body project 2. Healthy weight	36	EDDI	3	Group based; In-person	Dissonance Based	Low
Stice et al. (2008)	481	M = 17.0 (SD = 1.4), 100%, 58%	1. Expressive writing 2. Assessment only control	36	EDDI	3	Group based; In-person	Psychoeducation	Low
Taylor et al. (2006)	480	M = 20.8 (SD = 2.6), 100%, 60%	1. Student bodies 2. Waitlist control	36	EDE	5	Internet	Cognitive Behavioral	High
Taylor et al. (2016)	206	M = 20 (SD = 1.8), 100%, 51%	1. Image and mood 2. Waitlist control	24	EDE	5	Internet	Cognitive Behavioral	High
Ghaderi et al. (2020)	443	M = 17.3 (SD = 1.4), 100%, N/A	1. Virtual body project 2. Expressive writing	24	EDDI	4	Group based; Virtual (via teleconferencing)	Dissonance Based	Low
Tanofsky-Kraff et al. (2014)	113	M = 14.5 (SD = 1.7), 100%, 56.6%	1. Interpersonal psychotherapy 2. Health education program	12	EDE	20	Group based; In-person	Interpersonal Psychotherapy	High
	398			24	EDDI	4			Low

(*continued on next page*)

Table 2 (*continued*)

Study	Sample size	Sample (age, % female, % White)	Study conditions (1. intervention, 2. control)	Length of follow-up (months)	Outcome measures	Intervention duration (hours)	Intervention format	Intervention theory	Risk of bias
Stice, Rohde, et al. (2013)		M = 18.4 (SD = 0.6), 100%, 83%	1. Healthy weight 2. Educational brochure				Group based; In-person	Lifestyle Modification	
Stice et al. (2018)	364	M = 19.1 (SD = 1.2), 72%, 68%	1. Project health 2. Healthy weight	24	EDDI	6	Group based; In-person	Lifestyle Modification	Low
Stice et al. (2018)	364	M = 19.1 (SD = 1.2), 72%, 68%	1. Healthy weight 2. Educational video	24	EDDI	6	Group based; In-person	Lifestyle Modification	Low
Stice et al. (2018)	364	M = 19.1 (SD = 1.2), 72%, 68%	1. Project health 2. Educational video	24	EDDI	6	Group based; In-person	Lifestyle Modification	Low
Lowe et al. (2012)	294	M = 17.0 (SD = 1.1), 100%, N/A	1. Intervention 2. Assessment only control	24	EDE	6	Group based; In-person	Behavioral Therapy	High

Abbreviations: SCID, Structured Clinical Interview for DSM-IV Axis I Disorders; EDE, Eating Disorder Examination; EDDI, Eating Disorder Diagnostic Interview.

Table 4
Operationalization and descriptive statistics for moderators.

Moderator	Coding description and criteria	Descriptive statistics
Length of follow-up	Continuous variable reflecting length of follow-up in months.	*M* = 27.32; *SD* = 13.38; *Range* = (6, 48)
Length of intervention	Continuous variable reflecting the length of the intervention in hours	*M* = 5.72, *SD* = 4.10; *Range* = (3,20)
Homework	Dichotomous variable reflecting whether intervention assigned homework. 1 = *Yes*; 0 = *No*	*Yes* = 16; *No* = 1
Implementation modality	Dichotomous variable reflecting whether intervention was delivered over the internet or in-person. 1 = Internet; 0 = *No*	*Internet* = 6; In-person = 13
Single risk sample	Dichotomous variable reflecting whether intervention recruitment was based on a single risk factor (v. multiple risk factors). 1 = *Yes*; 0 = *No*	*Yes* = 13; *No* = 6
Intervention theory	Categorical variable reflecting whether intervention was a dissonance-based, cognitive behavioral, or a healthy lifestyle modification	Dissonance = 7; Cognitive behavioral = 4; Healthy lifestyle modification = 4
Low risk of bias	Dichotomous variable reflecting whether intervention was low or moderate risk (v. high risk) 1 = *Yes*; 0 = *No*	*Yes* = 11; *No* = 8

inclusion criteria it was determined that these programs did not fit the parameters of indicated prevention. With regard to the intervention theory of the prevention programs evaluated in these 19 contrasts, 1 was psychoeducational, 1 was based on interpersonal psychotherapy, 1 was based on family treatment, 1 focused on promoting self-esteem and self-efficacy, 1 focused on weight gain prevention using behavioral therapy techniques from obesity treatments and principles from nutrition science, 3 promoted lifestyle refinements that bring caloric intake into balance with caloric expenditure, 3 were based on cognitive behavioral therapy, and 8 were dissonance-based prevention programs. These programs were delivered as in-person group interventions (n = 13), virtually delivered group interventions (n = 1), and internet-delivered interventions (n = 5). The interventions varied from 3 h to 20 h.

2.6. Narrative review of the identified trials

We provide a narrative review of the trials that we located that tested whether a prevention program reduced future onset of eating disorders, in which we describe each prevention program, the research design, the sample, and the analytic approach. We also attempted to gauge the acceptability of each prevention program. However, the papers did not provide parallel information about completion rates of the prevention programs, which made it impossible to apply a single definition of acceptability. We could only report the data provided in each trial about completion of the prevention program and comment on whether this suggested "good", "reasonable", "moderate", or "low" acceptability. For studies that reported the percent of participants who completed all of the intervention sessions, the most consistently reported completion statistic, we characterized acceptability to be "good" if over 80% of the sample completed all sessions, "reasonable" if between 60% to 79% completed all sessions, "moderate" if between 50% to 59% completed all sessions, and "low" if less than 50% completed all sessions.

2.7. Psychoeducational prevention program

One trial tested whether a psychoeducational eating disorder prevention program reduced future onset of eating disorders during a 1-year follow-up compared to an assessment-only control condition (Favaro, Zanetti, Huon, & Santonastaso, 2005). This prevention program focused on discouraging use of unhealthy weight control practices (e.g., strict dieting) and the cultivation of skills to respond to the media's overemphasis on thinness, consistent with evidence that dietary restriction and pursuit of the thin beauty ideal predict future onset of eating disorders. It also provided information about eating disorders and risk factors that predict onset of eating disorders. This effectiveness trial recruited 141 female adolescents from a vocational training school, though only 129 provided complete data (mean age 17.0 SD 1.1). This trial did not take an intent-to-treat analytic approach wherein data from all participants randomly assigned to each condition are included in analyses of intervention effects (Little & Yau, 1996). Statisticians have shown that using multiple imputation to impute missing data, allowing an intent-to-treat analytic approach, produces more accurate and efficient parameter estimates than alternative analytic approaches such as analyzing only participants who provide complete data, making multiple imputation the recommended procedure for handling missing data (Graham, 2009; Schafer & Graham, 2002). Investigators have imputed the data used to make eating disorder diagnoses (e.g., Allen et al., 2014). Thus, the test of the intervention effects is probably overly liberal compared to trials that used intent-to-treat analyses because the participants who did not provide complete data may have not completed the prevention program. We considered this a selective prevention program because it focused solely on females, and female sex has been found to increase risk for onset of eating disorders (Allen et al., 2014). The prevention program was delivered to groups of participants in 6 2-h

sessions. Participants completed diagnostic interviews that assessed any threshold or subthreshold eating disorder with assessors who were blinded to the condition of participants. In total, 5.3% of participants who received the prevention program developed an eating disorder during the 1-year follow-up versus 11.0% in the control condition. Although this represented a 52% reduction in future onset of eating disorders, this difference was not significant (t = 1.17, n.s.), likely because of limited power.

2.8. Cognitive-behavioral prevention programs

Three trials tested whether *Student Bodies,* a cognitive-behavioral eating disorder prevention program reduced future onset of eating disorders. The first was an efficacy trial that tested whether *Student Bodies* reduced future onset of eating disorders during a 1- to 3-year follow-up compared to an assessment-only control condition (Taylor et al., 2006). *Student Bodies* is delivered in 8 30-min Internet-delivered modules that focus on reducing weight concerns using cognitive behavioral therapy and included moderated asynchronous on-line discussions between group participants and a facilitator. Participants were young women (mean age = 20.8; SD = 2.6) who reported weight concerns, fear of weight gain, and overvaluation of weight and shape with a BMI between 18 and 32 at baseline, making this a selective prevention trial. Eating disorders were assessed with diagnostic interviews by assessors who were blinded to the condition to which participants were assigned. In total, 480 participants were randomized to condition, but only data from 421 were included in the analyses because the rest of the participants only provided baseline data (i.e., analyses were not conducted on an intent-to-treat basis). Because the authors did not include all participants randomized to condition, results likely provide an overly liberal test of intervention effects. Excluding participants who were randomized to *Student Bodies* who did not start or complete the prevention program from the analyses of intervention effects violates the principles of a randomized controlled trial in which all participants randomized to condition are retained in analyses (Shadish, Cook, & Campbell, 2002). Although the authors did not provide the sample size for the survival model and Dr. Taylor was unable to provide the sample size when contacted, calculations suggest that only 261 participants were included. The authors state that they excluded individuals with a threshold or subthreshold eating disorder at baseline and that 43 participants developed an eating disorder over follow-up. Fig. 2 from Taylor et al. (2006) indicates that 19% of *Student Bodies* participants showed onset of an eating disorder versus 14% of controls. Proportionally this means that 57.5% of the cases must have occurred in the *Student Bodies* condition and 42.4% in the control condition (19% + 14% = 33%; 19%/33% = 57.5%; 14%/33% = 42.4%). If 43 participants showed onset of an eating disorder over follow-up, 25 of the 43 cases must have been in the *Student Bodies* condition and 18 in the control condition (43 × 57.5% = 25 and 43 × 42.4% = 18). If 25 cases represent 19% of the participants in the *Student Bodies* condition, the cell size for that condition must have been 132 (25/19% = 1.32 × 100 = 132). If 18 cases represent 14% of the participants in the control condition, the cell size for that condition must have been 129 (18/14% = 1.28 × 100 = 129). These calculations indicate that the sample used in the survival model must have been 132 + 129 = 261. Given that Taylor et al. (2006) stated that 206 participants from the *Student Bodies* condition and 215 participants from the control condition were included in analyses (206 + 215 = 421), this means that data from only 261 of the 421 participants who were supposed to be included in the analyses were included and that 160 participants (38%) must have been excluded from the survival model for reasons not stated. Attrition from the prevention program (16%) was greater than attrition from the assessment-only control condition (9%). Overall 11% of participants assigned to *Student Bodies* never logged into the program, suggesting low acceptability. There were no significant differences in onset of any eating disorder over the 1- to 3-year follow-up. The incidence of eating disorder onset over follow-up was 19% in the intervention condition versus 14% in the control condition.

A second efficacy trial tested whether *Student Bodies* reduced future onset of eating disorders over a 6-month follow-up compared to an assessment-only control condition (Jacobi, Völker, Trockel, & Taylor, 2012). Participants were 126 young women (mean age = 22.3, SD = 2.9) who reported weight concerns, dieting, binge eating, or purging. Given that weight concerns and dieting are not symptoms of eating disorders, we classified this as a selective prevention program. Although 126 participants were randomized to condition, data from only 103 participants were included in analyses. Again, because the authors did not include all of participants randomized to condition, results likely provide an overly liberal test of intervention effects. Eating disorders were assessed by diagnostic interviews and assessors were blinded to the condition to which participants had been randomized. Participants assigned to *Student Bodies* opened 67% of the pages and completed 38% of the required postings (we note the completion rates reported in the articles, which differed across studies, but sometimes was not reported). Onset of eating disorders over follow-up was 7% in the control condition compared to 0% in the intervention condition. Although the authors did not report a test of whether the incidence of eating disorder onset was significantly lower in the intervention versus control condition, we conducted a Fisher's exact test (because of the low incidence) that revealed that the reduction in future eating disorder onset was not statistically significant (p = .109).

A third efficacy trial tested whether *Student Bodies* produced a reduction in future onset of any eating disorders over 2-year follow-up compared to an assessment-only control condition (Taylor et al., 2016). The intervention targeted individuals with weight concerns plus being teased about weight, having current or historical depression, or reporting compensatory weight control behaviors, leading us to classify this as a selective prevention program. This version of *Student Bodies* was adapted for use with this more selective population, resulting in a longer 10-week intervention. In this trial, *Student Bodies* also included monthly email messages to participants about the content of the intervention and reminders to use the skills from the intervention over a 9-month follow-up (making this nearly a 12-month intervention). In total 206 participants were recruited at universities (mean age = 20.0, SD = 1.8) and randomized to condition. However, only 185 participants were included in the analyses, meaning analyses were not intent-to-treat and that the test of intervention effects was likely overly liberal. Eating disorders were assessed via diagnostic interviews by assessors who were blinded to condition. Of the participants assigned to *Student Bodies* 5% never logged onto the program and another 10% dropped out after logging into the program; of the remaining participants only 56% completed at least half of the intervention sessions. These data also suggest low acceptability of this version of *Student Bodies*. By 2-year follow-up 24% of intervention participants showed onset of an eating disorder compared to 31% of the assessment-only control group; this difference was not statistically significant (Hazard Ratio [*HR*] = 0.73, p = .28).

2.9. Dissonance-based eating disorder prevention programs

Five trials evaluated whether a dissonance-based eating disorder prevention program, referred to as the *Body Project,* reduced onset of eating disorders. One efficacy trial tested whether *Body Project* produced a reduction in future onset of eating disorders over 3-year follow-up relative to a credible alternative prevention program (*Healthy Weight*), a credible expressive writing alternative intervention, and an assessment-only control condition (Stice, Marti, Spoor, Presnell, & Shaw, 2008). Participants in the group-delivered *Body Project* prevention program engage in verbal, written, and behavioral exercises in which they collectively explore the negative effects of pursuing the thin ideal, which putatively prompts participants to reduce their subscription to this ideal because people are motivated to align their attitudes with their publicly displayed behavior. Theoretically this intervention reduces pursuit of the thin ideal, which decreases body dissatisfaction,

dieting, and negative affect, which should reduce eating disorder symptoms and future eating disorder onset. Participants in the group-delivered *Healthy Weight* prevention program are encouraged to make gradual lifestyle refinements to bring energy intake into balance with energy expenditure, based on the reasoning that this would reduce body dissatisfaction, a key risk factor for eating disorders. Both prevention programs were described as body acceptance interventions and implemented in three 1-h sessions by a clinician and an undergraduate peer educator. In the expressive writing condition participants write about any emotionally important topic of their choosing in 3 individuals 45-min sessions. Participants were 481 adolescent girls (mean age 17.0, SD 1.4) recruited from high schools (51%) and colleges who reported body image concerns. Participants were randomized to condition. Assessors blinded to condition conducted diagnostic interviews over 3-year follow-up. Analyses included data from the full sample (i.e., were intent-to-treat), which means that those who were randomized to one of the prevention programs but did not attend any sessions were included in analyses, making this a conservative test of intervention effects. With regard to attendance, 91% of *Body Project* participants attended all 3 sessions, 91% of *Healthy Weight* participants attended all 3 sessions, and 98% of expressive writing controls completed all 3 writing sessions, suggesting good acceptability. At 3-year follow-up, onset of any eating disorder was 7% for *Body Project* participants, 6% for *Healthy Weight* participants, 9% for expressive writing controls, and 16% for assessment-only controls. Eating disorder onset was significantly lower for *Body Project* participants and *Healthy Weight* participants relative to assessment only controls ($HR = 2.75$, $p = .022$ and $HR = 2.50$, $p = .018$, respectively), but not relative to expressive writing controls. As such, *Body Project* produced a 60% reduction in future eating disorder onset and *Healthy Weight* produced a 61% reduction in future eating disorder onset.

A second trial tested whether the *Body Project* produced a reduction in future eating disorder onset over 3-year follow-up compared to an educational brochure control condition (Stice, Rohde, Shaw, & Gau, 2011). In this effectiveness trial high school clinicians recruited participants and delivered *Body Project* groups. To make it easier for clinicians to cover the material, the intervention was delivered in 4 1-h sessions in contrast to 3 1-h sessions as in previous trials, though the content was similar. In total, 306 female high school students (mean age 15.7, SD = 1.1) who reported body image concerns were recruited and randomized. Participants completed diagnostic interviews at all assessments with assessors blinded to condition. Intent-to-treat analyses were used. Of the 306 participants, 78% attended all 4 sessions of the intervention, implying reasonable acceptability. By the end of the 3-year follow-up 4.0% of *Body Project* participants showed onset of an eating disorders compared to 5.5% in the educational brochure control condition, which was not a significant difference ($p = .28$).

A third trial tested whether the *Body Project* produced a reduction in future eating disorder onset over 3-year follow-up compared to an educational brochure control condition (Stice, Rohde, Butryn, Shaw, & Marti, 2015). In this effectiveness trial college clinicians recruited participants and delivered *Body Project* groups. This trial evaluated a new enhanced-dissonance version of the *Body Project* that underscored the voluntary commitment, increased accountability for counter-attitudinal exercises, and increased the level of effort required because these factors increase dissonance induction. In total, 408 female college students (mean age 21.6, SD = 5.6) who reported body image concerns were recruited and randomized to condition. Assessors were blinded to the condition of participants. Among participants in the *Body Project* condition, 84% attended or made up all 4 sessions, suggesting good acceptability. Analyses were intent-to-treat. By the end of the 3-year follow-up 12.9% of *Body Project* participants showed onset of an eating disorders compared to 12.9% in the educational brochure control condition, which was not a significant difference ($p = .16$).

A fourth effectiveness trial tested whether implementation of the *Body Project* could be task shifted from implementation by clinicians to peer educators or to Internet delivery, without loss of effectiveness in reducing future eating disorder onset over 4-year follow-up compared to an educational video control condition (Stice, Rohde, Shaw, & Gau, 2020). Young women ($N = 680$; mean age 22.2) with body image concerns were randomly assigned to clinician-led *Body Project* groups, peer-led *Body Project* groups, the internet-delivered *eBody Project*, or an educational video control condition. Clinicians and peers delivered the standard 4 1-h session enhanced dissonance version of the *Body Project*. The *eBody Project* contains activities designed to mirror the activities in the *Body Project* and is implemented without a moderator to reduce cost. Assessors were blinded to the condition of participants. Analyses were intent-to-treat. In clinician-led *Body Project* groups 47% of participants completed all sessions, in peer-led *Body Project* groups 45% of participants completed all sessions, and in the *eBody Project* condition 57% of participants completed all modules, suggesting lower acceptability of the *Body Project* than in past trials. It was noteworthy that in this trial the completion rate was 11% higher for the *eBody Project* than for the in-person *Body Project*, replicating the evidence that the completion rate for the *eBody Project* was 10% higher than for the in-person *Body Project* from an earlier trial (Stice, Rohde, Durant, & Shaw, 2012), as this provides evidence that digital eating disorder prevention programs can have higher acceptability than in-person prevention programs. By 4-year follow-up, eating disorder onset was 8.1% in the peer-led *Body Project* condition, 19.3% in the clinician-led *Body Project* condition, 15.5% in the *eBody Project* condition, and 17.6% in educational video control participants. Eating disorder onset was significantly lower in the peer-led *Body Project* condition than in the clinician-led *Body Project* condition ($HR = 2.53$, $p = .009$) and the educational video control condition ($HR = 2.33$, $p = .020$), and marginally lower than in the *eBody Project* condition ($HR = 2.04$, $p = .056$). Thus, peer-led *Body Project* groups produced a 54% reduction in eating disorder onset compared to educational video controls, a 58% reduction in eating disorder onset compared to clinician-led *Body Project* groups, and a 48% reduction in eating disorder onset relative to the *eBody Project*.

A fifth trial tested whether *Body Project* groups delivered virtually over the internet by peer educators produced a reduction in future eating disorder onset over 2-year follow-up relative to an expressive writing comparison condition (Ghaderi et al., 2020). In this effectiveness trial 443 female adolescents and young women (mean age = 17.2, SD = 1.5) with body image concerns were randomized to virtual *Body Project* groups, an expressive writing comparison condition, or a 6-month waitlist control condition (participants in the latter condition were not included in the onset analyses because they did not provide data for the full 2-year follow-up). The standard *Body Project* was delivered virtually, with only minimal modifications to improve cultural fit and virtual implementation. Assessors were blinded to the condition of participants and analyses were intent-to-treat. In total, 72% of participants completed at least half of the *Body Project* sessions. By the end of the 2-year follow-up eating disorder onset was 2.0% in the virtual *Body Project* condition and 8.8% in the expressive writing comparison condition, a significant difference ($HR = 3.85$ $p = .037$). This translated into a 77% reduction in future eating disorder onset.

2.10. Lifestyle modification eating disorder prevention programs

Three trials evaluated an eating disorder prevention program that encourages participants to make lasting lifestyle modifications that bring caloric intake into balance with caloric expenditure, which should reduce body dissatisfaction, a key risk factor for eating disorders. The first efficacy trial, which was summarized previously because it also evaluated the effects of the *Body Project* (Stice et al., 2008) found that *Healthy Weight* significantly reduced future onset of eating disorders by 61% relative to assessment-only controls. A second efficacy trial tested whether *Healthy Weight* reduced future onset of eating disorders over a 2-year follow-up compared to an educational brochure control condition (Stice, Rohde, Shaw, & Marti, 2013). Participants were 398 young

women and men (mean age = 18.4, SD = 0.6) who reported weight concerns and were randomly assigned to condition. Assessors who conducted diagnostic interviews were blinded to condition. Analyses were intent-to-treat. In total, 67% of *Healthy Weight* participants attended all four sessions, suggesting only moderate acceptability. By the end of the 2-year follow-up 3.4% of *Healthy Weight* participants showed onset of any eating disorder, compared to 8.5% in the educational brochure control condition, a significant difference ($p = .039$). This translated into a 60% reduction in future eating disorder onset. These findings are noteworthy because *Healthy Weight* was the first prevention program to significantly reduce future eating disorder onset in two trials and the only one to produce this effect in a sample containing both female and male adolescents.

A third trial tested whether a new dissonance-based version of *Healthy Weight* (referred to as *Project Health*) reduced future onset of eating disorders relative to the original *Healthy Weight* prevention program and an educational video control condition (Stice, Rohde, Shaw, & Gau, 2018). In the group-based *Project Health* participants make the small lasting lifestyle improvements to bring caloric intake into balance with caloric expenditure, as in the original *Healthy Weight* prevention program, and also discuss the negative effects of obesity, overeating, and a sedentary lifestyle and the benefits of a healthy body weight, eating a healthy diet, and regular exercise. These latter activities theoretically create dissonance about engaging in behaviors that contribute to unhealthy weight gain. In this effectiveness trial young women and men with weight concerns ($N = 364$) were randomized to the three conditions and completed blinded diagnostic interviews assessing eating disorders over a 2-year follow-up. Analyses were intent-to-treat. In the *Project Health* condition 36% of participants attended all sessions and in the *Healthy Weight* condition 48% of participants attended all sessions, suggesting low acceptability. By 2-year follow-up 3% of participants in *Project Health*, 3% of participants in *Healthy Weight*, and 8% of participants in the educational control condition showed onset of an eating disorder. The 63% reduction in future eating disorder onset in the two eating disorder prevention programs relative to the control condition was only marginal ($HR = 2.77$ $p = .064$).

2.11. Behavioral therapy-based eating disorder & weight gain prevention program

One unpublished efficacy trial tested whether a weight gain prevention program reduced future onset of eating disorders over 2-year follow-up among female college students (Lowe, Stice, Butryn, & Sarwer, 2012). This prevention program used behavioral therapy techniques from obesity treatments to reduce caloric intake and increase exercise, principles from nutrition science that focused on reducing the energy density of dietary intake, and persuasion principles from social psychology to increase motivation for change. Participants in this program were female college students (M age = 18.24, $SD = 0.44$) who were at high-risk for weight gain and eating disorders by virtue of body dissatisfaction, elevated dieting, and elevated weight suppression, making this a selective prevention program. Among participants assigned to this prevention program, 26% attended all six sessions and 78.7% attended at least half of the sessions, suggesting low acceptability. In total, 294 participants were randomized to the intervention or an assessment-only control condition. Analyses for this trial were intent-to-treat. By 2-year follow-up, 18.1% of participants in the prevention program showed onset of an eating disorder, compared to 13.2% in the control condition, which was not a significant difference ($OR = 0.69$, 95% CI [0.74–2.83], $p = .278$).

2.12. Self-esteem/self-efficacy enhancement eating disorder prevention program

One effectiveness trial tested whether an intensive 1-year classroom delivered prevention program reduced future onset of eating disorders among female and male adolescents who were attending high schools for athletes (Martinsen et al., 2014). This intervention primarily focused on improving self-esteem and self-efficacy and was delivered to students in 4 90-min sessions and in social media advertisements by respected athletes. These intervention elements were supplemented with educational seminars for coaches and school staff on puberty and disordered eating. Overall, 55.5% of participants attended all intervention sessions, suggesting moderate acceptability. In total, 577 participants were randomized to condition, though it appears that only 439 participants were included in the analyses (i.e., analyses were not intent-to-treat). Randomized participants were selectively assessed at baseline, and only participants that provided data at follow-up were included in the analyses. Diagnostic interviews were used to assess eating disorders, though assessors were not blinded to condition. By the end of the 1-year follow-up, 0% of intervention participants showed onset of an eating disorder compared to 4.2% of controls. Although the authors did not test whether the incidence of eating disorder onset was lower in the intervention versus control condition for the full sample, we conducted a Fisher's exact test that revealed that this effect was statistically significant ($p = .001$). This effect translates into a 100% reduction in future eating disorder onset.

2.13. Interpersonal therapy-based eating disorder prevention program

One efficacy trial tested whether an interpersonal psychotherapy intervention reduced future eating disorder onset compared to a health education intervention over 1-year follow-up (Tanofsky-Kraff et al., 2014). Both interventions were group-based and lasted 20-h, which is a positive design feature because only three other trials have compared two prevention programs that were equal in credibility and contact time. Participants were 116 adolescent girls recruited from the community who were overweight and reported loss of control eating, making this a trial of selective prevention programs. However, the participant flowchart noted that only 98 were included in the analyses, indicating that analyses were not intent-to-treat, despite the claims in the article. Data indicated 65% of interpersonal psychotherapy participants and 85% of health education participants attended at least 80% of the sessions, suggesting moderate acceptability. By 12-month follow-up, 8.2% of participants in the health education intervention developed an eating disorder compared to 2.0% participant in interpersonal psychotherapy (a 76% reduction in eating disorder onset), though this difference was not statistically significant ($OR = 4.27$, $p = .20$), likely due to limited power caused by the small sample.

2.14. Family-therapy-based eating disorder prevention program

One efficacy trial tested whether a parent-based internet-delivered intervention, *Parents Act Now,* reduced onset of anorexia nervosa over 12-month follow-up relative to an assessment-only control condition (Jacobi et al., 2018). This intervention included activities from the first phase of family-therapy for anorexia nervosa developed by Lock and Le Grange (2001), which primarily aims to have parents play the key role in re-feeding their underweight children. Female adolescents who reported a low body weight, as well as weight concerns, drive for thinness, perfectionism, amenorrhea, excessive exercise, or family history of an eating disorder were recruited along with their parents. It is critical to note that only a low BMI has been shown to predict future onset of anorexia nervosa (Stice et al., 2017); none of the other factors (e.g., body dissatisfaction, perfectionism, pursuit of the thin ideal, excessive exercise, amenorrhea or parental history of an eating disorder) have been shown to predict future onset of anorexia nervosa in a prospective risk factor study. Female adolescents (mean age = 13.8, SD = 1.0) and their parents ($N = 66$) were recruited, but due to extreme attrition (59% by 12-month follow-up), this trial was terminated before the full sample was recruited. Unfortunately, 50% of participants assigned to the intervention dropped out of treatment, parents opened only 28% of the

program pages, and 29% of the parents never logged into the program, signaling very low acceptability of this intervention. Assessors who conducted diagnostic interviews were not blinded to intervention condition of participants. However, results revealed that no participants in either condition, including the assessment-only control condition, showed onset of anorexia nervosa. The fact that none of the participants in the control condition showed onset of anorexia nervosa is consistent with the impression that this population was not at high-risk for this eating disorder, likely because they did not target youth with established risk factors for anorexia nervosa. We requested data on whether participants showed future onset of any other threshold or subthreshold eating disorder, but the first author did not think that they collected this data.

In sum, the 15 trials that we located provided 19 tests of whether a prevention program produced a significant reduction in future eating disorder onset over 1- to 4-year follow-up periods relative to either a minimal intervention control condition or another prevention program. Seven of these tests revealed significant reductions in eating disorder onset over follow-up compared to some type of comparison condition. The dissonance-based *Body Project* significantly reduced future onset of eating disorders when implemented or co-implemented by peer educators in three trials, but not when implemented solely by adult clinicians in three trials. This pattern of findings suggests that the intervention is more credible if implemented by someone similar to the participants in age and sex, implying that future implementation projects would be more effective in preventing eating disorder onset if peer educators deliver or co-deliver the *Body Project.* Further, the lifestyle modification-based *Healthy Weight* prevention program significantly reduced future onset of eating disorders in two trials, and produced a marginal reduction in eating disorder onset in a third trial. The new dissonance-based *Project Health* also produced a marginal reduction in future eating disorder onset. The fact that these lifestyle modification interventions produced significant or marginal reductions in future eating disorder onset is encouraging, though acceptability of these interventions was only moderate. These two lifestyle modification prevention programs also significantly reduced excess weight gain, suggesting that these prevention programs are unique in preventing both eating disorder onset and obesity onset. The prevention program targeting self-esteem and self-efficacy among high school athletes also significantly reduced onset of future eating disorders, suggesting that it would be useful to attempt to replicate those findings. In contrast, the cognitive-behavioral *Student Bodies* eating disorder prevention program did not significantly reduce onset of future eating disorders in the three trials included in this review. Results imply that cognitive behavioral eating disorder prevention programs are not effective in reducing future onset of eating disorders, potentially because of the limited acceptability of this intervention. However, it is also possible that the Internet is not the ideal medium for eating disorder prevention programs. Data likewise indicated that psychoeducational, behavioral therapy-based, interpersonal-therapy-based, and family-therapy-based prevention programs did not reduce future onset of eating disorders.

3. Meta-analytic results

3.1. Research quality assessment

Of the 15 studies included in this meta-analysis, 6 studies (40%) were considered low risk of bias, 1 study (6.7%) had some concerns for bias, and 8 studies (53.3%) were considered high risk of bias. Missing outcome data, statistical analysis methods, and measurement of outcome introduced the most bias in the included studies. Nine studies (60%) were high risk or had some concerns for bias regarding missing data and its impact on results. Six studies (40%) did not use intent-to-treat analyses when testing for intervention effects, likely contributing to an overly liberal estimation of intervention effects. Other sources of bias included non-blinded assessors and problems with randomization procedures; 26.7% of studies did not use blinded assessors, and 60% of studies either did not report or did not perform allocation sequence concealment in their randomization procedures. Risk of bias findings are presented in Fig. 3.

3.2. Average effect size and effect size heterogeneity

The literature search identified 15 published or unpublished reports from which we extracted data for 19 contrasts for prevention programs. Table 1 provides an overview of the effect size from each prevention program. The average effect size was significantly different from zero (OR = 1.64, 95% CI = [1.09, 2.46], 95% prediction interval = [0.50, 5.32], t = 2.54, p = .020). The heterogeneity in effect sizes was significant (Q [18] = 35.98, p = .007). Egger's regression test for funnel plot asymmetry was not significant (t[13] = 0.04, p = .972), providing no evidence for a relation between effect size and sample size and thus no evidence of publication bias.

3.3. Moderator analysis

To maximize what can be learned from this literature, we conducted exploratory analyses of moderators. We restricted our focus to moderators that were available for at least 14 of the 19 contrasts and we excluded any moderators for which fewer than two studies provided effect sizes for each level of the moderator (e.g., only one prevention program involved no homework). Correlations between the examined potential moderators are shown in Table 5. Results for the bivariate moderator analyses are displayed in Table 6. Intervention effects were significantly larger for trials that recruited participants with elevations on only a single risk factor versus recruiting participants with elevations in multiple risk factors (t = 2.14, p = .047); marginal odds were 2.00 (95% CI = [1.31, 3.06]) and 0.95 (95% CI = [0.52, 1.73]) for single risk factor and multiple risk factor trials, respectively. In the intervention theory model, healthy lifestyle modification programs exhibited significantly larger eating disorder prevention effects than cognitive behavioral programs (t = 2.32, p = .039); marginal odds were 1.15 (95% CI = [0.64, 2.06]), 1.47 (95% CI = [>1.00, 2.15]), and 2.93 (95% CI = [1.52, 5.67]) for cognitive behavioral, dissonance, and healthy lifestyle modification programs, respectively. The average eating disorder prevention effect was not significantly larger for dissonance-based prevention programs than for cognitive behavioral prevention programs. None of the other examined moderators correlated with intervention effect sizes, including length of follow-up, length of the prevention program, implementation modality, and risk of bias. None of the moderators showed significant unique effects when entered simultaneously in a multivariate model.

Table 5
Correlation matrix of moderators.

1. Length of follow-up in months						
2. Length of intervention in hours	−0.51					
3. Implementation modality	−0.12	−0.24				
4. Single risk sample	0.43	−0.32	−0.51			
5. Dissonance intervention	0.66	−0.39	−0.09	0.47		
6. Healthy lifestyle modification intervention	−0.19	0.00	−0.43	0.30	−0.56	
7. Low bias risk	0.68	−0.48	−0.34	0.80	0.56	0.36

Note: p values are not provided as moderator values from the same study are not independent; thus, the correlation matrix is intended for descriptive purposes only.

Table 6
Moderator models for effect sizes from prevention programs versus control conditions.

Moderator	Parameter	Estimate	SE	*t*	*p*	σ between	σ within	I^2 between [95% CI]	I^2 within [95% CI]
Length of follow-up in months	Intercept	0.58	0.21	2.79	0.013	0.52	0.18	51.79 [0, 54.57]	5.95 [0, 34.35]
	Length of follow-up	−0.40	0.26	−1.55	0.139				
Length of intervention in hours	Intercept	0.53	0.21	2.60	0.019	0.50	0.23	50.44 [0, 54.95]	10.18 [0, 35.88]
	Length of intervention	0.18	0.28	0.64	0.534				
Implementation modality	Intercept	0.56	0.22	2.49	0.023	0.44	0.31	37.03 [0, 50.08]	19.04 [0, 39.35]
	Internet delivery	−0.18	0.35	−0.52	0.611				
Single risk factor sample	Intercept	−0.05	0.28	−0.17	0.865	0.34	0.21	29.89 [0, 49.85]	11.31 [0, 35.36]
	Single risk factor	0.74	0.35	2.14	0.047				
Intervention theory	Intercept	0.14	0.27	0.51	0.619	<0.01	0.24	<0.01 [0, 43.24]	21.94 [0, 39.61]
	Dissonance	0.24	0.32	0.76	0.461				
	Healthy lifestyle modification	0.94	0.40	2.32	0.039				
Risk of bias	Intercept	0.15	0.28	0.55	0.591	0.38	0.23	34.64 [0, 52.74]	12.02 [0, 34.99]
	Low bias risk	0.50	0.36	1.40	0.179				

Note: Confidence intervals computed using the Q-profile method (Viechtbauer, 2007).

4. Discussion

The goal of this report was to characterize the results of randomized controlled trials that tested whether an eating disorder prevention program significantly reduced future onset of eating disorders. Our literature search identified 15 trials that provided 19 tests of whether a prevention program reduced future onset of eating disorders relative to a minimal intervention control condition or a credible alternative intervention. Results revealed that three prevention programs significantly reduced the future onset of eating disorders over follow-up in one or more trials (Fig. 2). Risk for bias ratings for the trials ranged from low to high, with 8 trials being rated high risk for bias, one being rated as some concerns, and 6 being rated as low risk for bias. It was reassuring that five of the six trials that detected significant eating disorder prevention effects were rated as low risk of bias, as this suggests that it is unlikely that the conclusions are misleading because the significant prevention effects were driven by trials with a low risk of bias.

The dissonance-based *Body Project* showed the most repeated effects of reduction of eating disorder onset, in that this prevention program (1) significantly reduced future onset of eating disorders in 3 separate trials conducted in 2 countries (Ghaderi et al., 2020; Stice et al., 2008; Stice, Rohde, Shaw, & Gau, 2020), (2) was the only prevention program shown to produce a significantly greater reduction in future onset of eating disorders relative to a credible alternative intervention, and did so in two trials, and (3) produced prevention effects that persisted through 4-year follow-up. Yet these results were nuanced in that these prevention effects only emerged when this intervention was delivered or co-delivered by peer educators who were similar to participants in terms

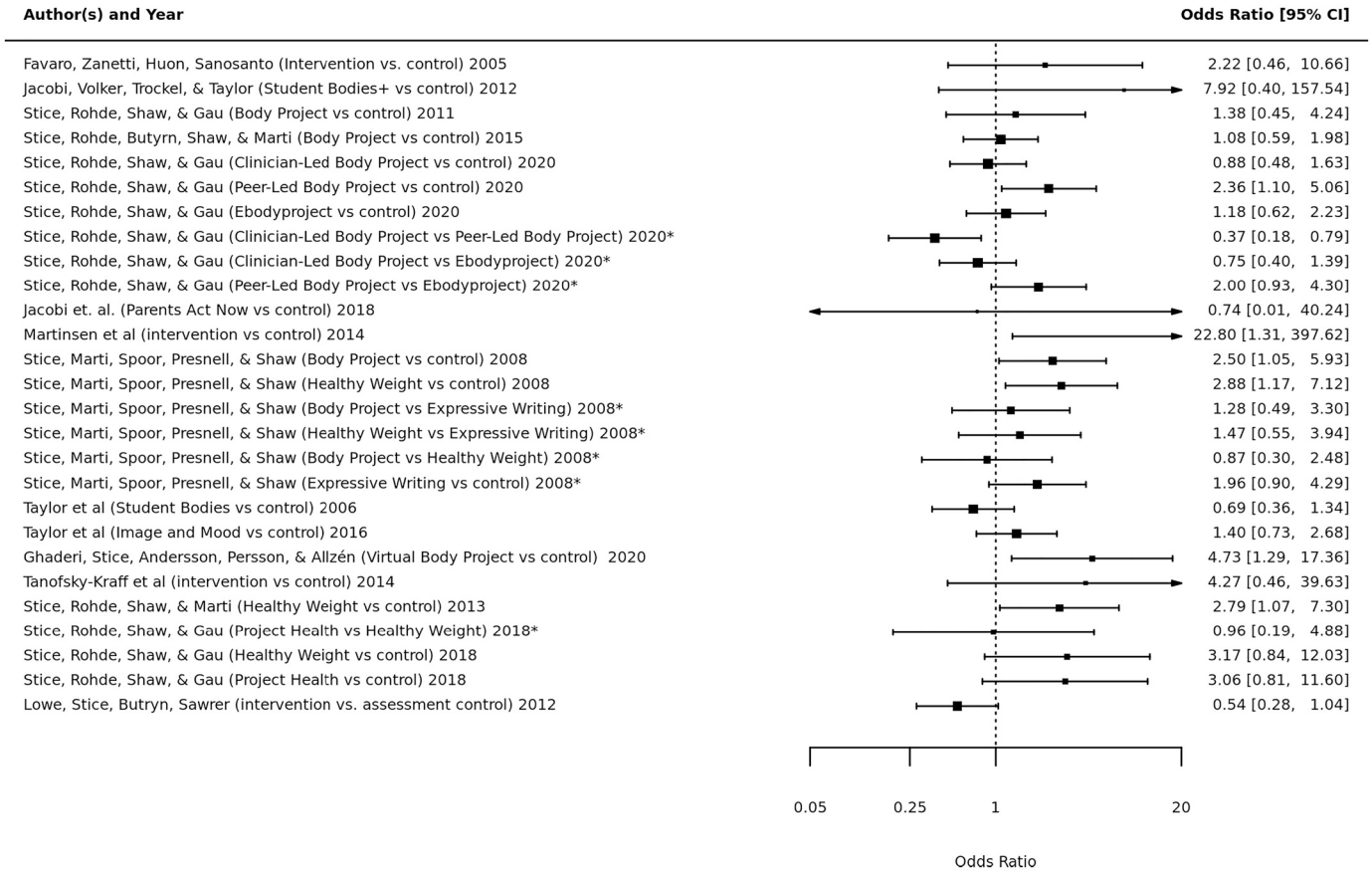


Fig. 2. Forest plot of odds ratios with 95% confidence intervals. Point sizes are inverse-variance weighted to indicate the contribution of each individual study. Asterisks indicate effect size contrast not included in the meta-analysis models.

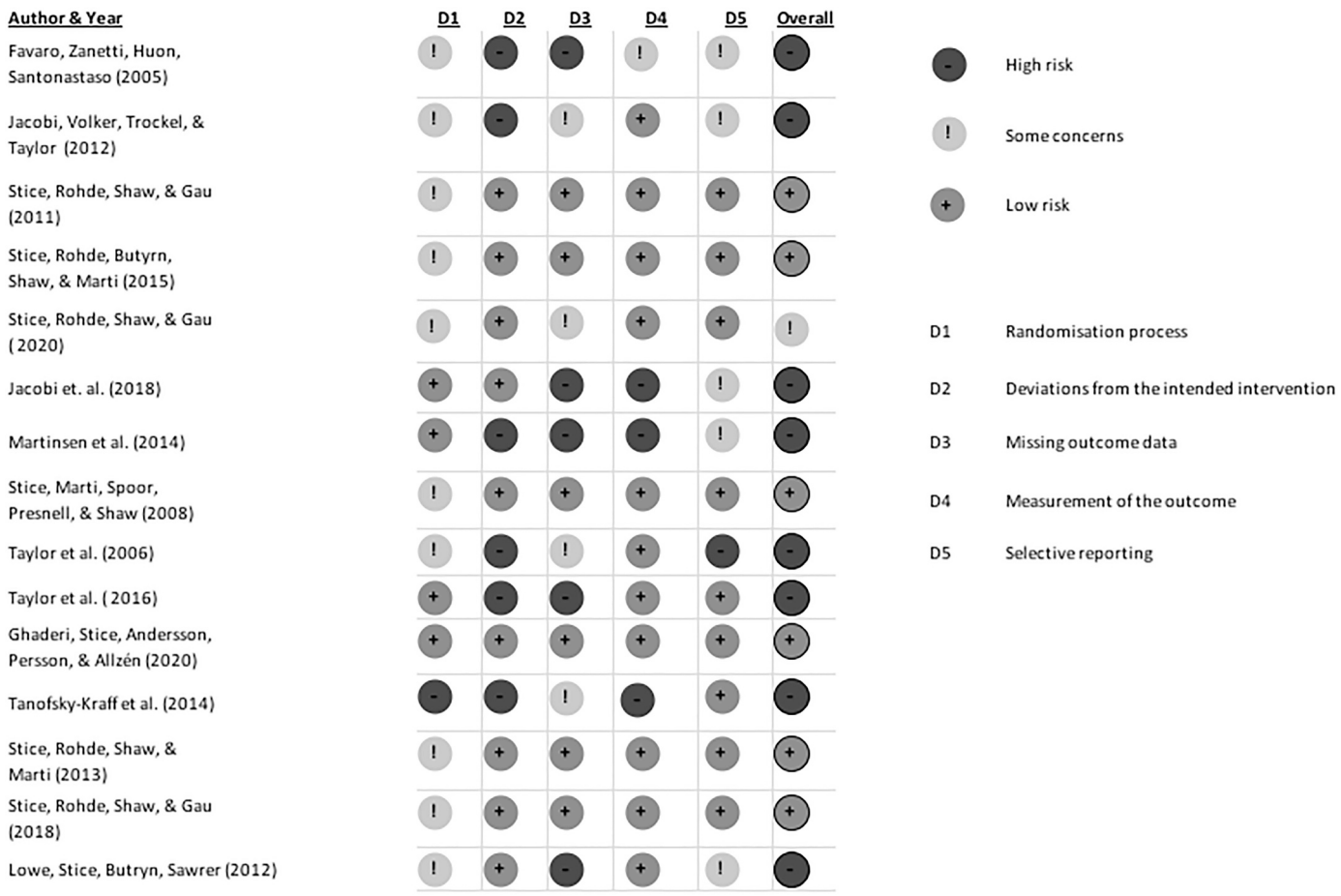


Fig. 3. Risk of bias summary.

of sex and age. Three trials found that the *Body Project* significantly reduced future onset of eating disorders when delivered or co-delivered by peer educators and 3 trials found that the *Body Project* did not significantly reduce future onset of eating disorders when delivered by clinicians. Indeed, the fact that in one trial, peer-led *Body Project* groups produced a significantly greater reduction (*OR* = 0.37 [95% CI = [0.18–0.79]) in future eating disorder onset relative to clinician-led *Body Project* groups powerfully illustrates this point. We thus conducted post hoc analyses that tested whether reductions in future onset of eating disorders were significantly larger for *Body Project* groups implemented or co-implemented by peer educators versus clinicians. Intervention effects were significantly larger when the *Body Project* was implemented by peer educators versus clinicians ($t = 2.88$, $p = .045$); marginal odds were 2.70 (95% CI = [1.29, 5.67]) and 1.02 (95% CI = [0.57, 1.81])) for peer-led and clinician-led groups, respectively. The fact that the 2.70 odds ratio was not within the 95% confidence interval for the effects of cognitive behavioral prevention programs (0.64, 2.06) also provides evidence that peer-implemented *Body Project* groups produce significantly stronger prevention effects than cognitive behavioral programs. In addition, peer-delivered, but not clinician-delivered *Body Project* groups produced a marginally greater reduction in future onset of eating disorders than the internet-delivered *eBody* Project. It was also noteworthy that virtually implemented *Body Project* groups produced a significant reduction in eating disorder onset relative to a credible alternative intervention, because virtual implementation could markedly expend the reach of this prevention program and allow easy implementation to most communities because internet access is now widespread. The NNT effect sizes indicated that it would be necessary to implement this prevention program to 8 to 11 participants to prevent onset of one case of an eating disorder, the largest effect sizes for any of the prevention programs evaluated in the identified trials. Theoretically the preventive effects of the *Body Project* emerged because this intervention reduces pursuit of the thin beauty ideal, which has been shown to increase risk for future eating disorder onset (Dakanalis et al., 2017; Rohde et al., 2015; Stice et al., 2017). Consistent with the intervention theory, reductions in thin-ideal internalization mediate the effect of this prevention program on reduction in eating disorder symptoms (Seidel, Presnell, & Rosenfield, 2009; Stice et al., 2007), this prevention program reduces brain reward region response to thin models (Stice, Yokum, & Waters, 2015), as well as attentional-bias for thin models (Tobin, 2020) and implicitly-assessed valuation of the thin ideal (Kant, Wong-Chung, Evans, Stanton, & Boothroyd, 2019).

The healthy lifestyle modification prevention program *Healthy Weight* produced significant reductions in future onset of eating disorders in two trials (Stice et al., 2008; Stice, Rohde, et al., 2013), though in the third trial, reductions in future eating disorder onset were only marginal (Stice et al., 2018), potentially because that trial included males, which lowered the incidence of eating disorder onset over follow-up and thus sensitivity to detect preventive effects. The NNT effect sizes indicated that it would be necessary to implement this prevention program to between 10 and 20 participants to prevention one case of an eating disorder. Thus, the effect size for this prevention program was smaller than for the *Body Project.* These findings might be considered unexpected because many clinicians and researchers believe that dietary restriction increases risk for eating disorders (e.g., Fairburn, 1997; Heatherton & Polivy, 1992; Neumark-Sztainer et al., 2006). However, this prevention program encourages lasting gradual reductions in caloric intake and increases in physical activity to bring caloric intake into balance with caloric expenditure, rather than transient dietary restriction. The evidence that a prevention program that reduces future weight

gain and onset of overweight and obesity significantly reduces onset of eating disorders is also consistent with evidence that overeating increases risk for future onset of eating disorders (Stice et al., 2017). The fact that healthy lifestyle modification prevention programs reduce risk for both future onset of eating disorders and overweight/obesity should be considered a positive feature given that overweight and obesity is the second leading cause of premature mortality in Western countries (Flegal, Carroll, Kit, & Ogden, 2012), markedly eclipsing the mortality produced by eating disorders (by a factor of 30). Nonetheless, this prevention program was somewhat less acceptable to participants than the dissonance-based *Body Project*, based on the somewhat lower attendance and completion data for the former prevention program.

The other prevention program to significantly reduce future eating disorder onset focused on increasing self-efficacy and self-esteem (Martinsen et al., 2014). The NNT for this prevention program, indicates that 24 participants would need to receive this intervention in order to prevent the emergence of one case of an eating disorder, which is a smaller effect size than observed for the dissonance-based and lifestyle modification-based prevention programs. The theoretical underpinnings of this prevention program are based on the idea that individuals must have the required personal skills, and degree of self-efficacy to change behaviors (Bandura, 1986). However, low self-esteem and low self-efficacy have not been shown to predict future onset of eating disorders to our knowledge. Indeed, prior prevention programs that focused on increasing self-esteem have not produced significant reductions in eating disorder symptoms (O'Dea, 1995; Wade, Davidson, & O'Dea, 2003). Though the intervention evaluated by Martinsen et al. (2014) produced significant results, based on the NNT it does not appear to reduce eating disorder onset at the same magnitude as the above described prevention programs. Moreover, that trial has a high risk for bias, due to missing outcome data and the use of non-blinded assessors. These aspects of bias have the potential to favor the intervention in the results, so it is important to consider the results within this context. Based on the data provided, the program did reduce eating disorder onset more effectively than some other prevention programs included in this analysis, however it would be useful to evaluate this program in another trial with stronger methodological and statistical rigor in future trials.

The remaining eating disorder prevention programs were cognitive behavioral therapy, interpersonal therapy, behavioral therapy, family therapy, and a psychoeducational intervention. None of these prevention programs resulted in significant reductions of eating disorder onset. Somewhat surprisingly given the evidence-base for cognitive behavioral treatments for a variety of psychiatric disorders, the one prevention program based on cognitive behavioral therapy, *Student Bodies*, did not significantly reduce future onset of eating disorders in three trials (Jacobi et al., 2012; Taylor et al., 2006; Taylor et al., 2016). Although these findings may suggest that cognitive behavioral principles are not effective in reducing risk factors for eating disorders, it is also possible that delivery of eating disorder prevention programs over the internet is less effective, potentially because of the relatively low acceptability of this prevention program based on completion rates.

The one trial that tested an interpersonal psychotherapy prevention program (Tanofsky-Kraff et al., 2014) did not find a significant reduction in eating disorder onset compared to the health education comparison condition. In that trial, the interpersonal therapy intervention was tested against a health education comparison condition, making this a more conservative test than provided by many of the other trials. Nonetheless, the fact that there was a 76% greater reduction in future eating disorder onset in the interpersonal therapy condition suggests that it might be useful to evaluate interpersonal therapy prevention programs in a larger trial that has greater sensitivity to detecting significant prevention effects.

The family therapy-based prevention program, *Parents Act Now,* also did not produce a reduction in eating disorder onset, though that trial appears to have only assessed anorexia nervosa (Jacobi et al., 2018). This intervention focused primarily on attaining a healthy weight via involvement of the family in feeding. However, this trial did find that the family therapy prevention program produced significantly greater increases in BMI than observed in the control condition. This study's lack of significant reduction in anorexia nervosa onset may have occurred because of the low participant engagement, which forced the authors to close recruitment before the planned sample was enrolled. Thus, although this prevention program attempted to target a unique population that is at high risk for anorexia nervosa, this population seemed to have limited motivation to complete the intervention and the low acceptability of this prevention program would likely preclude broad implementation of this intervention.

Results likewise revealed that the prevention program that used behavioral therapy principles from weight loss treatments and concepts from nutrition science did not significantly prevent eating disorders (Lowe et al., 2012). The low acceptability of this prevention program and the fact that this trial recruited participants based on multiple risk factors may have played a key role in the lack of preventive effects

Finally, the psychoeducational program did not produce a statistically significant reduction in onset of eating disorders (Favaro et al., 2005). Although the incidence of eating disorders reported in the intervention condition were lower than that of the control condition, the difference was not significant. These findings are in line with the conclusions drawn from eating disorder prevention meta-analyses (Stice et al., 2007; Yager & O'Dea, 2008) and other prevention areas that psychoeducational interventions are not effective in producing behavioral change (e.g., Larimer & Cronce, 2002).

4.1. *Effects of the meta-analytic review*

The average prevention effect size was statistically significant (OR = 1.64, 95% CI = [1.09, 2.46], $t = 2.54$, $p = .020$) and there was heterogeneity in effect sizes (Q [18] = 35.96, $p = .007$). These results converge with those from an earlier meta-analytic review with a narrower focus on trials conducted at universities (Harrer, Adam, et al., 2019; Harrer, Cuijpers, et al., 2019).

Moderator analyses provided evidence that eating disorder prevention effects were significantly larger for trials that recruited participants with elevations on a single risk factor versus those that recruited participants based on elevations in multiple risk factors. Theoretically this is because the latter trials recruited more heterogeneous samples which may have attenuated the ability to detect effects relative to trials involving samples that were more homogeneous in terms of risk for eating disorders. This finding implies that it might be best if future eating disorder prevention trials recruit participants with elevations on only a single risk factor. Moderator analyses also revealed that healthy lifestyle modification eating disorder prevention programs produced significantly larger eating disorder prevention effects than the cognitive behavioral eating disorder prevention programs. Further, the post hoc analyses provided evidence that the dissonance-based prevention program produced larger eating disorder prevention effects when implemented by peer educators versus clinicians, and data also suggested the peer-implemented dissonance-based prevention programs produce larger effects than cognitive behavioral prevention programs.

Moderator analyses did not provide support for the other factors that were hypothesized to correlate with larger eating disorder prevention effects, which included length of follow-up, length of the prevention program, internet versus in-person implementation, and risk of bias of the trials. However, the small number of trials included in this meta-analytic review limited the ability to detect the effects of moderators in the univariate models. Further, single versus multiple risk samples and lifestyle modification prevention programs versus cognitive behavioral prevention programs did not produce a unique effect when entered alongside the other examined moderators, though the collinearity among moderators likely contributed to the null unique effect of the two significant moderators.

4.2. Limitations

It is important to consider the limitations of this review when interpreting the findings and our conclusions. First, we only identified 15 trials that could be included in our meta-analysis, which limits the generalizability of our findings, as most eating disorder prevention programs that have been developed were not evaluated in trials designed to detect the prevention of new onset of eating disorders. It is thus possible that other prevention programs might show efficacy in reducing future onset of eating disorders if they were evaluated in rigorously-designed and adequately-powered trials. Second, the relatively small number of trials that tested for prevention effects also limited the sensitivity to detecting moderators that correlate with larger prevention effects. Third, some studies did not confirm that alternative interventions were perceived as equally credible as the eating disorder prevention program evaluated in the trials (Ghaderi et al., 2020; Tanofsky-Kraff et al., 2014), raising the possibility that the superior effects for the eating disorder prevention program produced in the former trial were driven by greater perceived credibility of that intervention compared to the comparison intervention. Fourth, none of the trials that we located were powered to test whether the eating disorder prevention effects differed for various ethnic groups, genders, or individuals with different sexual orientations or identities. An important priority for future studies will be to test whether these prevention programs are similarly effective for various sub-populations that experience eating disorders, though this would likely require over-sampling of participants from each ethnic minority group in order to have sufficient sensitivity. Finally, many of the trials did not report sufficient information for coding of important moderators, which prevented an extensive evaluation of moderators in the meta-analysis.

4.3. Future directions

It will be critical for more researchers to conduct trials that are designed to detect whether prevention programs reduce future eating disorder onset (i.e., enroll large samples, conduct blinded diagnostic interviews over at least a 2-year follow-up). Our literature review revealed that only a small portion of prevention programs that have been developed have been evaluated in trials designed to detect true prophylactic effects. Results suggest that it might be optimal to work with peers to deliver the prevention programs and to target individuals who are at risk for eating disorders because of a single risk factor. It will also be important to conduct large enough trials that permit analyses of whether the prevention programs are equally effective in reducing future eating disorder onset for various sub-groups, such as different ethnic groups, different genders, and people with different sexual identifications and orientations. Dissonance-based eating disorder prevention programs have been found to produce similar reductions in risk factors and eating disorder symptoms for White, Black, Asians, and Hispanic participants, as well as for heterosexual and non-heterosexual adolescent girls and young women (Rodriguez, Marchand, Ng, & Stice, 2008; Shaw, Rohde, Desjardins, & Stice, 2020; Stice, Marti, & Cheng, 2014). However, no trials have tested whether other eating disorder prevention effects are similar across various ethnic groups. Such research should allow identification of prevention programs that are not effective for all sub-populations and may inform the design of prevention programs that are more effective for certain sub-populations. It will also be important to conduct research on how to effectively implement prevention programs that have been shown to produce reliable reductions in future eating disorder onset on a broad scale. The evidence that prevention programs can produce a large reduction in future eating disorder onset when implemented virtually via videoconferencing is encouraging because this delivery modality could vastly expand the reach and accessibility of prevention programs. In addition, it will be important to determine how to improve the acceptability of the prevention programs, as they often had low attendance and completion rates. Improving acceptability of eating disorder prevention programs has the potential to increase the prevention effects of these interventions.

5. Conclusions

The fact that lifestyle modification and dissonance-based prevention programs significantly reduced future onset of eating disorders in multiple trials, producing a 54% to 77% reduction in future eating disorder onset implies that broadly implementing these prevention programs could reduce the population prevalence of eating disorders. The NNT effect sizes were between 8 and 11 for the dissonance-based prevention program, and between 10 and 20 for the lifestyle modification prevention program, suggesting that the former prevention program is more efficacious. The fact that the dissonance-based eating disorder prevention program produced a large reduction in eating disorder onset when implemented virtually is an encouraging new finding, as this implementation method should facilitate broad implementation, including the possibility of implementing this prevention program internationally. It is exciting that the field has produced several eating disorder prevention programs that have shown efficacy and effectiveness in reducing future onset of eating disorders, as this may permit a reduction in the population prevalence of these pernicious psychiatric disorders.

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Declaration of Competing Interest

The authors have no conflicts of interest to disclose.

References

van Aert, R., & Jackson, D. (2019). A new justification of the Hartung-Knapp method for random-effects meta-analysis based on weighted least squares regression. *Research Synthesis Methods, 10*, 515–527. https://doi.org/10.1002/jrsm.1356.

Allen, K., Byrne, S., Oddy, H., & Crosby, R. (2013). Eating disorders in adolescents: Prevalence, stability, and psychosocial correlates in a population-based sample of male and female adolescents. *Journal of Abnormal Psychology, 122*, 720–732.

Allen, K., Byrne, S., Oddy, W., Schmid, U., & Crosby, R. (2014). Risk factors for binge eating and pruging eating disorders: Differences based on age of onset. *International Journal of Eating Disorders, 47*, 802–812.

Arcelus, J., Mitchell, A., Wales, J., & Nielsen, S. (2011). Mortality rates in patients with anorexia nervosa and other eating disorders: A meta-analysis of 36 studies. *Archives of General Psychiatry, 68*, 724–731. https://doi.org/10.1001/archgenpsychiatry.2011.74.

Bandura, A. (1986). *Social foundations of thought and action* (pp. 23–28). Englewood Cliffs, NJ: Prentice-Hall.

Begg, S., Vos, T., Baker, B., Stevenson, C., Stanley, L., & Lopez, A. (2007). *The burden of disease and injury in Australia 2003*. Canberra: Australian Institute of Health and Welfaire.

Chen, H., Cohen, P., & Chen, S. (2010). How big is a big odds ratio? Interpreting the magnitudes of odds ratios in epidemiological studies. *Communications in Statistics, 39*, 860–864.

Claydon, E., & Zullig, K. (2019). Eating disorders and academic performance among college students. *Journal of American College Health*. https://doi.org/10.1080/07448481.2018.1549556.

Cochran, W. (1954). The combination of estimates from different experiments. *Biometrics, 10*, 101–129. https://doi.org/10.2307/3001666.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. Hillsdale, NJ: Erlbaum.

Crow, S., & Smiley, N. (2010). Costs and cost-effectiveness in eating disorders. In W. S. Agras (Ed.), *The Oxford handbook of eating disorders* (pp. 480–485). Oxford: Oxford University Press.

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

Fairburn, C. (1997). Eating disorders. In D. Clark, & C. Fairburn (Eds.), *Science and practice of cognitive behaviour therapy* (pp. 209–241). Oxford: Oxford University Press.
Fairburn, C., Bailey-Straebler, S., Basden, S., Doll, H., Jones, R., Murphy, R., et al. (2015). A transdiagnostic comparison of enhanced cognitive behaviour therapy (CBT-E) and interpersonal psychotherapy in the treatment of eating disorders. *Behaviour Research and Therapy, 70*, 64–71.
Favaro, A., Zanetti, T., Huon, G., & Santonastaso, P. (2005). Engaging teachers in an eating disorder prevention intervention. *International Journal of Eating Disorders, 38*, 73–77.
Flegal, K., Carroll, M., Kit, B., & Ogden, C. (2012). Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999–2010. *Journal of the American Medical Association [Internet], 307*, 491.
Ghaderi, A., & Scott, B. (2001). Prevalence, incidence and prospective risk factors for eating disorders. *Acta Psychiatrica Scandinavica, 104*, 122–130.
Ghaderi, A., Stice, E., Andersson, G., Enö Persson, J., & Allzén, E. (2020). A randomised controlled trial of the effectiveness of virtually delivered body project (vBP) groups to prevent eating disorders. *Journal of Consulting and Clinical Psychology, 88*, 643–656.
Graham, J. (2009). Missing data analysis: Making it work in the real world. *Annual Review of Psychology, 60*, 549–576.
Harrer, M., Adam, S., Mag, E., Baumeister, H., Cuijpers, P., Bruffaerts, R., et al. (2019). Prevention of eating disorders at universities: A systematic review and meta-analysis. *International Journal of Eating Disorders, 53*, 813–833.
Harrer, M., Cuijpers, P., Furukawa, T., & Ebert, D. D. (2019). dmetar: Companion R package for the guide 'Doing Meta-Analysis in R'. Retrieved from http://dmetar.protectlab.org.
Heatherton, T., & Polivy, J. (1992). Chronic dieting and eating disorders: A spiral model. In J. Crowther, D. Tennenbaum, S. Hobfold, & M. Parris (Eds.), *The etiology of bulimia nervosa: The individual and familial context* (pp. 133–155). Washington, DC: Hemisphere.
Higgans, J., Altman, D., Gotzsche, P., Moher, D., Oxman, A., Savovic, J., et al. (2011). The Cochrane Collaboration's tool for assessing risk of bias in randomized trials. *The British Medical Journal, 343*, d5928.
Hox, J. (2010). *Multilevel analysis: Techniques and applications* (2nd ed.). New York, NY: Routledge.
Hudson, J., Hiripi, E., Pope, H., & Kessler, R. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry, 61*, 348–358.
IntHout, J., Ioannidis, J., & Borm, G. (2014). The Hartung-Knapp-Sidik-Jonkman method for random effects meta-analysis is straightforward and considerably outperforms the standard DerSimonian-Laird method. *BMC Medical Research Methodology, 14*. https://doi.org/10.1186/1471-2288-14-25.
Jacobi, C., Fittig, E., Bryson, S. W., Wilfley, D., Kraemer, H., & Taylor, C. (2011). Who is really at risk? Identifying risk factors for subthreshold and full syndrome eating disorders in a high-risk sample. *Psychological Medicine, 41*, 1939–1949.
Jacobi, C., Hutter, K., Volker, U., Mobius, K., Richter, R., Trockel, M., et al. (2018). Efficacy of a parent-based, indicated prevention for anorexia nervosa: Randomized controlled trial. *Journal of Medical Internet Research, 20*, e296.
Jacobi, C., Völker, U., Trockel, M. T., & Taylor, C. B. (2012). Effects of an Internet-based intervention for subthreshold eating disorders: A randomized controlled trial. *Behaviour Research and Therapy, 50*(2), 93–99. https://doi.org/10.1016/j.brat.2011.09.013.
Kant, R., Wong-Chung, A., Evans, E., Stanton, E., & Boothroyd, L. (2019). The impact of a dissonance-based eating disorder intervention on implicit attitudes to thinness in women of diverse sexual orientations. *Frontiers in Psychology, 10*, 2611.
Killen, J., Taylor, C., Hayward, C., Haydel, K., Wilson, D., Hammer, L., et al. (1996). Weight concerns influence the development of eating disorders: A 4-year prospective study. *Journal of Consulting and Clinical Psychology, 64*, 936–940.
Knapp, G., & Hartung, J. (2003). Improved tests for a random effects meta-regression with a single covariate. *Statistics in Medicine, 17*, 2693–2710.
Kraemer, H., & Kupfer, D. (2006). Size of treatment effects and their importance to clinical research and practice. *Biological Psychiatry, 59*, 990–996.
Larimer, M., & Cronce, J. (2002). Identification, prevention and treatment: A review of individual-focused strategies to reduce problematic alcohol consumption by college students. *Journal of Studies on Alcohol, Supplement*(s14), 148–163.
Lipsey, M., & Wilson, D. (2001). *Practical meta-analysis*. Thousand Oaks, CA: Sage.
Little, R., & Yau, L. (1996). Intent-to-treat analysis of longitudinal studies with drop-outs. *Biometrics, 52*, 1324–1333.
Lock, J., & Le Grange, D. (2001). *Treatment manual for anorexia nervosa: A family-based approach*. New York: Guilford.
Lowe, M., Stice, E., Butryn, M., & Sarwer, D. (2012). *Experimental test of a weight gain prevention program among female college freshmen* (Unpublished study).
Martinsen, M., Bahr, R., Borresen, R., Holme, I., Pensgaard, A., & Sundgot-Borden, J. (2014). Preventing eating disorders among young elite athletes: A randomized controlled trial. *Medicine and Science in Sports & Exercise, 46*, 435–447. https://doi.org/10.1249/MSS.0b013e3182a702fc.
McIntosh, V., Jordan, J., Carter, J., Luty, S., McKenzie, J., Bulik, C., et al. (2016). Psychotherapy for transdiagnostic binge eating: A randomized controlled trial of cognitive-behavioural therapy, appetite-focused cognitive-behavioural therapy, and scheme therapy. *Psychiatric Research, 240*, 412–420.
McKnight, I. (2003). Risk factors for the onset of eating disorders in adolescent girls: Results of the McKnight longitudinal risk factor study. *American Journal of Psychiatry, 160*, 248–254.
Mrazek, P., & Haggerty, R. (1994). *Reducing risks for mental disorders: Frontiers for preventive intervention research*. Washington, D.C.: National Academy Press.
Neumark-Sztainer, D., Wall, M., Guo, J., Story, M., Haines, J., & Eisenberg, M. (2006). Obesity, disordered eating, and eating disorders in a longitudinal study of adolescents: How do dieters fare 5 years later? *Journal of the American Dietetic Association, 106*, 559–568.
O'Dea, J. (1995). *Everybody's different: A self-esteem program for young adolescents*. Sudney University Press.
Patton, G., Johnson-Sabine, E., Wood, K., Mann, A., & Wakeling, A. (1990). Abnormal eating attitudes in London school girls- a prospective epidemiological study: Outcome at twelve month follow-up. *Psychological Medicine, 20*, 383–394.
Patton, G., Selzer, R., Coffey, C., Carlin, J., & Wolfe, R. (1999). Onset of adolescent eating disorders: Population based cohort study over 3 years. *BMJ., 318*, 765–768.
Peters, J., Sutton, A., Jones, D., Abrams, K., & Rushton, L. (2006). Comparison of two methods to detect publication bias in meta-analysis. *JAMA, 295*, 676–680. https://doi.org/10.1001/jama.295.6.676.
Rodriguez, R., Marchand, E., Ng, J., & Stice, E. (2008). Effects of a cognitive-dissonance-based eating disorder prevention program are similar for Asian American, Hispanic, and White participants. *International Journal of Eating Disorders, 41*, 618–625.
Rohde, P., Stice, E., & Marti, N. (2015). Development and predictive effects of eating disorder risk factors during adolescence: Implications for prevention efforts. *International Journal of Eating Disorders, 48*, 187–198.
Santonastaso, P., Friederici, S., & Favaro, A. (1999). Full and partial syndromes in eating disorders: A 1-year prospective study of risk factors among female students. *Psychopathology, 32*, 50–56.
Schafer, J., & Graham, J. (2002). Missing data: Our view of the state of the art. *Psychological Methods, 7*, 147–177.
Schwarzer, G., Carpenter, J., & Rücker, G. (2015). *Meta-analysis with R*. New York: Springer.
Seidel, A., Presnell, K., & Rosenfield, D. (2009). Mediators in the dissonance eating disorder prevention program. *Behaviour Research and Therapy, 47*, 645–653.
Shadish, W., Cook, T., & Campbell, D. (2002). *Experimental and quasi-experimental designs for generalized causal inference*. Boston, MA: Houghton Mifflin.
Shaw, H., Rohde, P., Desjardins, C., & Stice, E. (2020). Sexual orientation correlates with baseline characteristics but show no moderating effects of dissonance-based eating disorder prevention programs for women. *Body Image, 32*, 94–102.
Simmonds, M. (2015). Quantifying the risk of error when interpreting funnel plots. *Systematic Reviews*, 4. https://doi.org/10.1186/s13643-015-0004-8.
Stice, E. (2016). Interactive and meditational etiologic models of eating disorder onset: Evidence from prospective studies. *Annual Review of Clinical Psychology, 12*, 359–381.
Stice, E., Davis, K., Miller, N., & Marti, C. N. (2008). Fasting increases risk for onset of binge eating and bulimic pathology: A 5-year prospective study. *Journal of Abnormal Psychology, 117*, 941–946.
Stice, E., Desjardins, C., Rohde, P., & Shaw, H. (in press). Sequencing of symptom emergence in anorexia nervosa, bulimia nervosa, binge eating disorder, and purging disorder and relations of prodromal symptoms to future onset of these disorders. Journal of Abnormal Psychology.
Stice, E., Gau, J., Rohde, P., & Shaw, H. (2017). Risk factors that predict future onset of each DSM-5 eating disorder: Predictive specificity in high-risk adolescent females. *Journal of Abnormal Psychology, 126*, 38–51.
Stice, E., Marti, C., & Cheng, Z. (2014). Effectiveness of a dissonance-based eating disorder prevention program for ethnic groups in two randomized controlled trials. *Behaviour Research and Therapy, 55*, 54–64.
Stice, E., Marti, N., & Durant, S. (2011). Risk factors for onset of eating disorders: Evidence of multiple risk pathways from an 8-year prospective study. *Behaviour Research and Therapy, 49*, 622–627.
Stice, E., Marti, C., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *Journal of Abnormal Psychology, 122*, 445–457.
Stice, E., Marti, C., Spoor, S., Presnell, K., & Shaw, H. (2008). Dissonance and healthy weight eating disorder prevention programs: Long-term effects from a randomized efficacy trial. *Journal of Consulting and Clinical Psychology, 76*, 329–340.
Stice, E., Rohde, P., Butryn, M., Shaw, H., & Marti, C. (2015). Effectiveness trial of a selective dissonance-based eating disorder prevention program with female college students: Effects at 2-and 3-year follow-up. *Behaviour Research and Therapy, 71*, 20–26. https://doi.org/10.1016/j.brat.2015.05.012.
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*, 907–916.
Stice, E., Rohde, P., Shaw, H., & Desjardins, C. (2020). Weight suppression increases odds for future onset of anorexia nervosa, bulimia nervosa, and purging disorder, but not binge eating disorder. *American Journal of Clinical Nutrition, 112*, 941–947.
Stice, E., Rohde, P., Shaw, H., & Gau, J. (2011). An effectiveness trial of a selected dissonance-based eating disorder prevention program for female high school students: Long-term effects. *Journal of Consulting and Clinical Psychology, 79*, 500–508.
Stice, E., Rohde, P., Shaw, H., & Gau, J. (2018). An experimental therapeutics test of whether adding dissonance-induction activities improves the effectiveness of a selected obesity and eating disorder prevention program. *International Journal of Obesity, 42*, 462–468.
Stice, E., Rohde, P., Shaw, H., & Gau, J. (2020). Clinician-led, peer-led, and internet-delivered dissonance-based eating disorder prevention programs: Effectiveness of these delivery modalities through 4-yr follow-up. *Journal of Consulting and Clinical Psychology, 88*, 481–494.
Stice, E., Rohde, P., Shaw, H., & Marti, C. (2013). Efficacy trial of a selective prevention program targeting both eating disorders and obesity among female college students:

1- and 2-year follow-up effects. *Journal of Consulting and Clinical Psychology, 81*, 183–189.
Stice, E., Shaw, H., & Marti, C. (2007). A meta-analytic review of eating disorder prevention programs: Encouraging findings. *Annual Review of Clinical Psychology, 3*, 233–257.
Stice, E., Yokum, S., & Waters, A. (2015). Dissonance-based eating disorder prevention program reduces reward region response to thin models: How actions shape valuation. *PLoS One, 10*(12), Article e0144530.
Swanson, S., Crow, S., le Grange, D., Swendsen, J., & Merikangas, K. (2011). Prevalence and correlates of eating disorders in adolescents: Results from the National Comorbidity Survey Replication Adolescent Supplement. *Archives of General Psychiatry, 68*, 714–723.
Tanofsky-Kraff, M., Shomaker, L., Wilfley, D., Young, J., Sbrocco, T., Stephens, M., et al. (2014). Targeted prevention of excess weight gain and eating disorders in high-risk adolescent girls: A randomized controlled trial. *American Journal of Clinical Nutrition, 100*, 1010–1018.
Taylor, C., Bryson, S., Luce, K., Cunning, D., Celio, A., Abascal, L., … Wilfley, D. (2006). Prevention of eating disorders in at-risk college-age women. *Archives of General Psychiatry, 63*, 881–888.
Taylor, C., Trockel, M., Cunning, D., Bailey, J., Aspen, V., Jacobi, C., … Wilfley, D. (2016). Reducing eating disorder onset in a very high risk sample with significant comorbid depression: A randomized controlled trial. *Journal of Consulting & Clinical Psychology, 84*(5), 402–414.
Tobin, L. (2020). *Dissonance-based eating disorder preventive intervention and attentional biases in body-dissatisfied university women: A cluster randomized controlled trial (unpublished doctoral thesis)*. Calgary, Alberta, Canada: University of Calgary.
Van den Noortgate, W., López-López, J., Marin-Martinez, F., & Sánchez-Meca, J. (2013). Three-level meta-analysis of dependent effect sizes. *Behavior Research Methods, 45*, 576–594. https://doi.org/10.3758/s13428-012-0261-6.
Viechtbauer, W. (2007). Confidence intervals for the amount of heterogeneity in meta-analysis. *Statistics in Medicine, 26*, 37–52. https://doi.org/10.1002/sim.2514.
Viechtbauer, W. (2010). Conducting meta-analyses in R with the metafor package. *Journal of Statistical Software, 36*, 1–48. Retrieved from http://www.jstatsoft.org/v36/i03/.
Wade, T., Davidson, S., & O'Dea, J. (2003). A preliminary controlled evaluation of a school-based media literacy program and self-esteem program for reducing eating disorder risk factors. *International Journal of Eating Disorders, 33*, 371–383.
Watson, H., Joyce, T., French, E., Willan, V., Kane, R., Tanner-Smith, E., … Egan, S. (2016). Prevention of eating disorders: A systematic review of randomized, controlled trials. *International Journal of Eating Disorders*. https://doi.org/10.1002/eat.22577. Advance on-line publication.
Wonderlich, S., Peterson, C., Crosby, R., Smith, T., Klein, M., Mitchell, J., et al. (2014). A randomized controlled comparison of integrative cognitive-affective therapy (ICAT) and enhanced cognitive-behavioral therapy (CBT-E) for bulimia nervosa. *Psychological Medicine, 44*, 543–553.
Yager, Z., & O'Dea, J. (2008). Prevention programs for body image and eating disorders on university campuses: A review of large controlled interventions. *Health Promotion International, 23*, 173–189.

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