An Examination of Participants Who Develop an Eating Disorder Despite Completing an Eating Disorder Prevention Program: Implications for Improving the Yield of Prevention Efforts

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Abstract Numerous trials provide support for the *Body Project*, an eating disorder prevention program wherein young women with body image concerns critique the thin ideal. Despite medium to large effects, some participants subsequently develop an eating disorder, suggesting that intervention or recruitment procedures could be improved. This study investigated baseline and acute intervention predictors of DSM-5 eating disorder development during a 3-year follow-up among Body Project participants. Combined data from two trials compare participants who experienced eating disorder onset during follow-up ($n=20$) to those who did not ($n=216$). Participants who did versus did not develop an eating disorder started the intervention with higher eating disorder symptoms ($\eta^2=0.08$), negative affect ($\eta^2=0.06$), thin-ideal internalization ($\eta^2=0.02$), and body dissatisfaction ($\eta^2=0.02$); the same baseline predictors of eating disorder onset emerged in controls. Attenuated pre–post reductions in eating disorder symptoms ($\eta^2=0.01$) predicted eating disorder onset but not after controlling for baseline levels. Given that Body Project and control participants who later developed an eating disorder started with initial elevations in risk factors and eating disorder symptoms, it might be useful to develop a more intensive variant of this program for those exhibiting greater risk at baseline and to deliver the prevention program earlier to prevent initial escalation of risk. The fact that nonresponders also showed greater negative affect and eating disorder symptoms suggests that it might be useful to add activities to improve affect and increase dissonance about disordered eating.

Keywords Prevention · Eating disorder · Cognitive dissonance · Prediction · Response

Over 10 % of adolescent girls and young women in the US meet criteria for *Diagnostic and Statistical Manual of Mental Disorders* (4th ed. [DSM-IV]; American Psychiatric Association 1994) anorexia nervosa (AN), bulimia nervosa (BN), or eating disorder not otherwise specified (EDNOS; Hudson et al. 2007; Stice et al. 2009a; Wade et al. 2006). The incidence of DSM-5 eating disorder criteria by age 20 exceeded 13 % (Stice et al. 2013). Peak onset age of eating disorder onset is 19–20 for AN, 16–20 for BN, and 18–20 for binge eating disorder (BED), purging disorder (PD), and feeding and eating conditions not elsewhere classified (FEC-NEC) (Stice et al. 2013). More than half of individuals seeking treatment for eating pathology receive a DSM-IV EDNOS diagnosis and often resemble individuals with AN and BN regarding disordered eating severity and persistence (Fairburn and Bohn 2005). Individuals with EDNOS/FEC-NEC versus AN and BN also show similar functional impairment, morbidity, psychiatric comorbidity, and risk for future physical and mental health problems (Eddy et al. 2008; Fairburn and Bohn 2005; Keel et al. 2011; Stice et al. 2013).

Elevated thin-ideal internalization, body dissatisfaction, dietary restraint, and negative affect have been found to predict onset of eating disorders or symptoms (Fairburn et al. 2005; Killen et al. 1996; Patton et al. 1999; Stice et al. 2011; Striegel-Moore et al. 2007). Individuals reporting weight concerns and elevated negative affectivity have shown a higher future onset of threshold or subthreshold BN (Killen et al. 1996), and self-reported social pressure to be thin and body dissatisfaction predicted onset of threshold or subthreshold BN or BED (McKnight 2003).

The pernicious nature of eating disorders suggests that it is vital to develop and disseminate efficacious eating disorder prevention programs that reduce eating disorder risk factors,

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thereby decreasing eating disorder symptoms and future eating disorder onset. Although several prevention programs have reduced eating disorder symptoms through at least a 6-month follow-up in a single trial (e.g., Jones et al. 2008; McVey et al. 2007; Neumark-Sztainer et al. 1995; Stewart et al. 2001), considerably more empirical support has emerged for the *Body Project*, which is a selective dissonance-based eating disorder prevention program in which young women with body image concerns voluntarily critique the thin ideal in verbal, written, and behavioral exercises (Stice et al. 2000). Grounded in the theory that humans seek to maintain consistency between their words, thoughts, and actions, criticizing the thin ideal publically in this group-based intervention is thought to produce a motivational drive for participants to reduce their subscription to the unrealistic beauty ideal (Stice et al. 2000). This decreased pursuit of the thin ideal theoretically reduces body dissatisfaction, dietary restraint, negative affect, eating disorder symptoms, and risk for eating disorder onset.

Efficacy trials show that the Body Project produces greater reductions in eating disorder risk factors (e.g., thin-ideal internalization, body dissatisfaction, reported dieting, negative affect), eating disorder symptoms, functional impairment, and eating disorder onset over a 3-year follow-up relative to assessment-only control conditions and three alternative interventions (e.g., Stice et al. 2008b; Stice et al. 2000; Stice et al. 2012a; Stice et al. 2006). Independent efficacy trials have found that, relative to assessment-only control conditions and alternative interventions, dissonance-based eating disorder prevention programs produce significantly larger reductions in eating disorder risk factors and symptoms (Becker et al. 2005; Matusek et al. 2004; Mitchell et al. 2007; Halliwell and Diedrichs 2014). The Body Project is the only eating disorder prevention program to produce positive intervention effects that have been both independently replicated and significantly outperformed credible alternative interventions, making it the only eating disorder prevention program to meet the American Psychological Association's (1995) designation as an efficacious intervention.

Reductions in thin-ideal internalization appear to mediate the effects of the Body Project on change in the other outcomes (Seidel et al. 2009; Stice et al. 2007a). Participants assigned to high- versus low-dissonance versions of this program showed significantly greater reductions in eating disorder symptoms, supporting the theory that dissonance induction contributes to intervention effects (Green et al. 2005; McMillan et al. 2011). Classification tree analysis determined that Body Project participation reduced the risk conveyed by the most potent eating disorder risk factor in that trial—denial of the costs of pursuing the thin ideal (Stice et al. 2012b): Participants who denied the costs of pursuing the thin ideal who completed the Body Project showed an eating disorder incidence of 0 % over the 3-year follow-up versus 18 % for those who completed two alternative interventions and 50 % for assessment-only controls. In addition, participation in the Body Project eliminated the negative effect of

exposure to supermodels on body dissatisfaction in young adolescent girls observed in controls, which was assessed in an independent study, reducing demand characteristics (Halliwell and Diedrichs 2014).

Despite the Body Project's medium to large effect sizes, it is important to investigate ways to improve the effects of this prevention program. One novel and potentially informative approach is to examine factors that distinguish participants who complete the Body Project but still go on to develop a DSM-5 eating disorder from those who complete this intervention and remain free of an eating disorder during follow-up. Such research could inform the design of more effective variants of this prevention program and improve matching of participants to the appropriate level of intervention. For example, if participants who developed an eating disorder began with elevated risk, it might suggest that prevention should be delivered at a younger age, when risk factors are less elevated, or that more intensive variants of this program are needed. Alternatively, if eating disorder onset is driven by weaker reductions in risk factors when receiving the program, it may suggest that a qualitatively different prevention program is needed that produces larger reductions in certain risk factors or that some Body Project recipients might require booster sessions for enhanced change. Although research has examined moderators of the effects of the Body Project (Müller and Stice 2013; Stice et al. 2008a), this is the first study to examine participants who received the Body Project, but later developed the psychiatric conditions the program was designed to prevent.

The first aim of the present study was to test the hypothesis that participants who develop an eating disorder despite completing the Body Project begin with elevated levels of eating disorder risk factors (thin-ideal internalization, body dissatisfaction, reported dieting, and negative affect) and eating disorder symptoms. Post hoc analyses included the entire sample and tested whether condition (Body Project vs. educational brochure control condition) moderated the effects of variables on eating disorder onset. The second aim tested the hypothesis that Body Project participants who develop eating disorders show smaller reductions in eating disorder risk factors and symptoms during the intervention. Thus, we examined two distinct research questions: one, that individuals who initially present with greater risk retain that elevated risk status following the intervention, and two, that individuals who experience less improvement from the prevention program are at increased risk for the negative outcome.

Methods

Participants and Procedure

To achieve an adequate sample of participants who showed eating disorder onset despite completing the prevention

program, we merged data from two large randomized controlled trials that used very similar research designs and methodologies. Study participants were young women and adolescent girls with body image concerns recruited for selective prevention programs. A total of 552 women participated in two randomized controlled trials (Body Project I and Body Project II). Analyses primarily focused on participants assigned to the Body Project condition ($n=268$, M age = 16.33, $SD=1.41$, M BMI [kg/m^2] = 24.16, $SD=5.19$), but some secondary analyses included participants assigned to the educational brochure control condition ($n=284$, M age = 16.20, $SD=1.42$, M BMI [kg/m^2] = 24.25, $SD=5.65$). Approximately 75 % of participants were in high school and 25 % were in college. The sample was 70 % Caucasians, 14 % Latinos, 6 % Asian/Pacific Islanders, 4 % African Americans, 1 % American Indians/Alaska Natives, and 5 % who specified other/mixed racial heritage, which was representative of the sampling frame (Stice et al. 2006, 2009b).

Of the 552 study participants, 32 Body Project participants and 17 control participants met criteria for a DSM-5 eating disorder diagnosis at pretest (we had excluded only individuals with full threshold AN and BN originally); these participants were excluded from analyses to ensure a prospective test of hypotheses. Of the remaining 503 study participants, 20 Body Project participants (study 1, $n=12$; study 2, $n=8$, 8.5 % total) and 23 control participants (study 1, $n=12$; study 2, $n=11$, 8.6 % total) were diagnosed with a DSM-5 eating disorder during the 3-year study follow-up. Participants completed assessments at pretest, posttest, and 6, 12, 24, and 36 months following the prevention program. The two trials from which the current sample is derived were described in detail previously (Stice et al. 2006, 2009b).

Measures

Thin-Ideal Internalization The Ideal-Body Stereotype Scale—Revised assessed thin-ideal internalization (Stice et al. 2006). Items used a response format ranging from 1 = *strongly disagree* to 5 = *strongly agree*. Items were averaged for this scale and those described below. This scale has shown internal consistency ($\alpha=0.91$), 2-week test–retest reliability ($r=0.80$), predictive validity for bulimic symptom onset, and sensitivity to detecting intervention effects (Stice et al. 2008b). Internal consistency for the present study was $\alpha=0.82$.

Body Dissatisfaction Items from the Satisfaction and Dissatisfaction with Body Parts Scale (Berscheid et al. 1973) assessed dissatisfaction with nine body parts using a response scale ranging from 1 = *extremely satisfied* to 6 = *extremely dissatisfied*. This scale has shown internal consistency ($\alpha=0.94$), 3-week test–retest reliability ($r=0.90$), predictive validity for bulimic symptom onset, and sensitivity to detecting

intervention effects (Stice et al. 2008b). Internal consistency for the present study was $\alpha=0.87$.

Dieting The Dutch Restrained Eating Scale (DRES; van Strien et al. 1986) assessed the frequency of dieting behaviors using a response scale ranging from 1 = *never* to 5 = *always*. The DRES has shown internal consistency ($\alpha=0.95$), 2-week test–retest reliability ($r=0.82$), convergent validity with self-reported caloric intake (but not objectively measured caloric intake), predictive validity for bulimic symptom onset, and sensitivity to detecting intervention effects (Stice et al. 2006, 2010; van Strien et al. 1986). Internal consistency for the present study was $\alpha=0.91$.

Negative Affect Negative affect was measured with the negative affect subscale from the Positive Affect and Negative Affect Scale—Revised (PANAS-X; Watson and Clark 1992) for Body Project I and the Center for Epidemiologic Studies-Depression Scale (CES-D; Radloff 1977) for Body Project II. The Sadness, Guilt, and Fear/Anxiety subscales from the PANAS assessed the extent to which participants had felt negative emotional states using a response format ranging from 1 = *very slightly or not at all* to 5 = *extremely*; it has shown internal consistency ($\alpha=0.95$), 3-week test–retest reliability ($r=0.78$), convergent validity, and predictive validity for bulimic symptom onset (Stice et al. 2006). Internal consistency for the present study was $\alpha=0.95$. The CES-D asked participants assess depressive symptoms such as “I have been feeling pretty down and unhappy this week” on a 4-point scale (0 = *never* to 3 = *most of the time*). The CES-D has shown internal consistency ($\alpha=0.8$ to 0.9) and test–retest stability ($r=0.50$ to 0.60) (Robert et al. 1991). Internal consistency for the present study was $\alpha=0.94$. To allow analysis of the combined data from the two trials, we used z-transformed versions of PANAS and CES-D variables, which seemed justifiable given the correlation ($r=0.75$) between these two negative affect measures (Watson and Clark 1992).

Eating Disorder Symptoms The Eating Disorder Diagnostic Interview (EDDI) assessed eating disorder symptoms over the past 12 months or since the last interview. Participants reported on eating disorder symptoms on a month-by-month basis, over the entire 3-year follow-up period. Items assessing symptoms in the past month were summed to form a symptom composite at each assessment. This composite has shown internal consistency ($\alpha=0.92$), inter-rater agreement ($r=0.93$), and 1-week test–retest reliability ($r=0.95$), sensitivity to detecting effects from eating disorder prevention and treatment interventions, and predictive validity for future onset of depression in past studies of adolescent girls and young women (Burton and Stice 2006; Stice et al. 2009b). Internal consistency for the present study was $\alpha=0.81$.

DSM-5 Eating Disorders The EDDI was also used to assess diagnostic criteria for DSM-5 eating disorders during the 3-year follow-up that followed the posttest assessment. Responses were used to determine whether participants met criteria for AN, BN, BED, and FEC-NEC, which included atypical AN, subthreshold BN, subthreshold BED, and purging disorder at any time during the 3-year follow-up (operationalized in Stice et al. 2013). EDDI DSM-5 eating disorder diagnoses showed 1-week test–retest reliability ($r=0.79$) and inter-rater agreement ($r=0.75$; Stice et al. 2013). EDDI eating disorder diagnoses have also shown sensitivity in detecting intervention effects and functional impairment, as well as predictive validity for future depression onset (Burton and Stice 2006; Seeley et al. 2009; Stice et al. 2008a, 2013).

Analytical Procedures

Preliminary analyses compared participants from the two Body Project trials on demographics and on pretest and posttest levels of the outcomes. We then characterized the incidence of eating disorder onset over the 3-year follow-up across intervention and control conditions, using logistic regression and Cox proportional hazard survival models to test whether eating disorder onset or time to onset differed for Body Project participants relative to controls.

Next, we compared Body Project participants who showed onset of a DSM-5 eating disorder during the 3-year follow-up to those who did not on pretest eating disorder risk factors (thin-ideal internalization, body dissatisfaction, dietary restraint, and negative affect) and eating disorder symptoms. We then tested whether condition moderated these relations using data from the full sample to examine whether the predictive effects of pretest risk factors and symptoms differed significantly for Body Project participants versus controls. In the event of a significant condition X baseline variable interaction, follow-up univariate ANOVAs compared participants who showed eating disorder onset to those who did not in the two conditions on pretest risk factors and symptoms.

Finally, we used binary logistic regression analyses to test whether pre-to-post change scores for risk factors and eating disorder symptoms predicted onset of DSM-5 eating disorders over the subsequent 3-year follow-up among Body Project participants. Predictor variables were computed to reflect magnitude of pre–post change on thin-ideal internalization, body dissatisfaction, dietary restraint, negative affect, and eating disorder symptoms (posttest scores–pretest scores). If the unadjusted pre–post change score for a variable significantly predicted eating disorder onset, we then tested whether controlling for pretest levels of that variable altered the predictive relations, to determine whether elevated pretest levels of the predictor(s) drove any significant effects. Because the small number of participants who showed eating disorder onset limited our power and because the analyses tested the

predictive effects only five variables, study result had a higher likelihood of type II error than a type I error. As a result, alpha was set to 0.05 across analyses.

Results

Preliminary Analyses

There were no significant differences between Body Project I and Body Project II participants on demographic variables or on pretest and posttest outcome variables. The proportion of participants who developed a DSM-5 eating disorder did not vary significantly for Body Project I versus II, $\chi^2(1, N=495)=3.57, p=0.08$. Demographic factors (i.e., age, ethnicity, parental education) and pretest BMI were tested to determine whether eating disorder onset was associated with these variables. Because none of these variables predicted eating disorder onset, we did not examine these factors further or control for them in subsequent analyses.

Among the 236 Body Project participants, 20 met criteria for a DSM-5 eating disorder onset during the 3-year follow-up. Among these 20 Body Project participants, the incidence of DSM-5 diagnoses was 1 for AN, 1 for BN, 9 for BED, 2 for atypical AN, 2 for subthreshold BN, 2 for subthreshold BED, and 3 for purging disorder. Among the 267 control participants, 23 met criteria for a DSM-5 eating disorder onset during the 3-year follow-up. The incidence of DSM-5 diagnoses for these 23 control participants was 5 for BN, 8 for BED, 1 for atypical AN, 7 for subthreshold BN, 2 for subthreshold BED, and 8 for purging disorder. Seven control participants had comorbid eating disorder diagnoses during follow-up.

Collapsing across trials, 8.5 % of Body Project participants showed onset of a DSM-5 eating disorder during the 3-year follow-up, compared to 8.6 % of control participants, which was not statistically different $\chi^2(1, N=495)=0.00, p=0.98$. This nonsignificant difference is in contrast to our test for onset of DSM-IV eating disorders that indicated that 4.8 % of Body Project participants showed onset of an eating disorder over the 3-year follow-up, relative to 9.4 % of control participants, which was a significant difference ($\chi^2(1, 546)=4.29, p=0.038$). We focused on DSM-5 eating disorders in the present paper because the higher incidence increased the sensitivity of our analyses and because the diagnostic criteria for DSM-5 eating disorder were recently published. The hazard model revealed that for the small percentage of Body Project participants who developed a DSM-5 eating disorder, mean time to disorder onset was 22.25 months ($SD=10.16$), compared to 17.43 months ($SD=11.19$) for control participants, though this difference was not statistically significant ($F(1, 41)=1.50, p=0.15$).

Pretest Predictors of Eating Disorder Onset

A MANOVA tested whether Body Project participants who did versus did not show onset of a DSM-5 eating disorder differed on pretest eating disorder risk factors and symptoms. Assumptions of the analysis were met for dependence and linearity. As the assumption of multivariate normality was violated for eating disorder symptoms, a log 10 transformation normalized this variable and the test of homogeneity of variance was then met according to Box's M. Using Wilk's test of multivariate significance, eating disorder diagnosis onset was significantly related to the weighted multivariate combination of pretest eating disorder risk factors and symptoms, $\Lambda=0.93$, $F(5, 225)=3.60$, $p<0.05$, $\eta^2=0.08$. The root of the multivariate solution accounted for 8 % of the variance explained by the model. The standardized discriminate function coefficients (SDFCs) used to weight the multivariate composite revealed that negative affect (SDFC=-0.69) contributed most in forming the function that discriminated the eating disorder onset group from the eating disorder-free group.

Follow-up univariate ANOVAs for each of the five measures comprising the multivariate composite compared Body Project participants who did versus did not develop a DSM-5 eating disorder during follow-up on pretest variables. Assumptions of the analysis were met with the exception of homogeneity of variances, which was not met between groups for dietary restraint and negative affect ($p<0.05$, Levene's test). This assumption was satisfied for the remained of outcomes. Weighted ANOVA results (Welch 1951) were used to account for difference in variance. Body Project participants who subsequently developed a DSM-5 eating disorder versus those who did not showed significant pretest elevations on thin-ideal internalization, body dissatisfaction, negative affect, and eating disorder symptoms, but did not differ on pretest dieting (Table 1).

To examine the impact of baseline measures on a sample that included at-risk participants who did not receive a prevention intervention, a MANOVA using the full $N=503$ sample tested whether participants who did versus did not show onset of a DSM-5 eating disorder differed on pretest eating disorder risk factors and symptoms. Eating disorder diagnosis onset was significantly related to the weighted multivariate combination of pretest eating disorder risk factors and symptoms, $\Lambda=0.91$, $F(5, 468)=9.0$, $p<0.05$, $\eta^2=0.10$. The root of the multivariate solution accounted for 10 % of the variance explained by the model, and the SDFC revealed that negative affect (SDFC=-0.62) contributed most in forming the function that discriminated the eating disorder onset group from the eating disorder-free group. Most relevant to the present study, MANOVA analyses found that intervention condition did not significantly moderate the effects of any pretest eating disorder risk factors and symptoms on predicting eating disorder diagnosis during study follow-up, suggesting that the

Table 1 One-way analysis of variance summary table for the effect of pretest risk factors and symptoms on eating disorder onset

Pretest variables		Disorder free		DSM-5 ED		F	η^2	p
		M	SD	M	SD			
Thin-ideal internalization	Body Project	3.49	0.58	3.80	0.42	5.36	0.02	0.021*
	Control	3.57	0.46	3.76	0.47	3.42	0.02	0.066
Body dissatisfaction	Body Project	3.38	0.71	3.76	0.87	5.20	0.02	0.023*
	Control	3.19	0.75	3.67	0.70	9.38	0.04	0.002*
Dietary restraint	Body Project	2.48	0.83	2.85	1.12	2.50	0.02	0.067
	Control	2.38	0.85	2.87	0.82	6.90	0.03	0.009*
Negative affect	Body Project	-0.13	0.87	0.65	1.26	13.35	0.06	<0.001*
	Control	-0.13	0.96	0.84	1.14	19.47	0.08	<0.001*
Eating disorder symptoms	Body Project	10.33	10.76	23.65	24.51	10.43	0.08	0.001*
	Control	8.84	8.42	17.48	11.18	17.47	0.07	<0.001*

Pre-transformed descriptive values are presented here

* $p<0.05$

pattern of predictors for participants who received the Body Project intervention did not significantly differ from predictors for participants who did not receive the prevention program. Although the condition $\times$ predictor interactions were not significant, univariate ANOVAs comparing control participants who did versus did not develop a DSM-5 eating disorder during follow-up on pretest variables were computed for comparison purposes (Table 1).

Pre-Post Change Predictors of Eating Disorder Onset

Hierarchical binary logistic regression analysis tested whether the magnitude of pre-to-post change in eating disorder risk factors and symptoms predicted eating disorder onset during the subsequent 3-year follow-up (Table 2). The multivariate model was not significant and indicated that pre-post eating disorder risk factor and symptom change scores did not predict eating disorder onset. The statistical significance of individual regression coefficients was tested using the Wald chi-square statistic. Within the multivariate model, results indicated that eating disorder symptom change score was the only significant pre-post change predictor of eating disorder diagnosis onset (Wald $\chi^2(1)=4.74$, $p=0.03$, odds ratio=3.67); mean change in eating disorder symptoms was -0.05 (SD=0.31) for Body Project participants who developed a future eating disorder compared to a mean of -0.18 (SD=0.31) for Body Project participants who did not develop a disorder during follow-up, suggesting an attenuated intervention response from those who would develop a future eating disorder. In a second regression model that controlled for pretest levels of eating disorder symptoms, the change score failed to produce significant results (Wald $\chi^2(1)=0.28$, $p=0.60$, odds ratio=1.19), suggesting that pretest levels of this predictor may have driven the significant effect of eating disorder symptom change observed in the first regression model.

Discussion

The first aim of the study was to test the predictive value of baseline factors on eating disorder onset and whether the predictive effects differed for Body Project versus control participants. Extending prior research which has examined the mediators and moderators of the effects of the Body Project, the present study adds to this program of research in a novel fashion by focusing particularly on individuals who received the prevention program but still went on to develop an eating disorder.

Results provided support for the hypothesis that, across intervention conditions, participants who develop an eating disorder during follow-up entered the study with higher levels of risk factors and eating disorder symptoms. Body Project

Table 2 Binary logistic regression of change score predictors of DSM-5 eating disorder onset among Body Project participants

Predictor variable	β	S.E.	Wald	p	Odds ratio
Step 1					
Δ Thin-ideal internalization	0.23	0.40	0.34	0.56	1.26
Δ Body dissatisfaction	-0.06	0.28	0.05	0.82	0.94
Δ Dietary restraint	0.38	0.31	1.50	0.22	1.47
Δ Negative affect	-0.23	0.23	0.93	0.33	0.80
Δ Eating disorder symptoms	1.30	0.60	4.74	0.03*	3.67

$n=236$

* $p<0.05$

participants who develop an eating disorder during follow-up begin the intervention with higher levels of four of the five examined predictor variables, including thin-ideal internalization, body dissatisfaction, and negative affect, as well as elevated eating disorder symptoms; results for the fifth examined predictor, dietary restraint, had a similar pattern but did not reach statistical significance. Though none of the condition $\times$ predictor interactions were statistically significant, this pattern was in slight contrast to control participants who develop an eating disorder during follow-up and enter the study with higher levels of body dissatisfaction, dietary restraint, negative affect, and elevated eating disorder symptoms. For control participants who did versus did not develop an eating disorder, levels of thin-ideal internalization were trending toward significance. Thus, the present results suggest that the same risk factors that have predicted eating disorder onset in community samples of young women also predict eating disorder onset in this high-risk sample, whether participants receive the Body Project intervention or not. Elevations in these risk factors have been found to increase risk for eating disorder onset in prospective etiologic studies with community samples (e.g., Fairburn et al. 2005; Killen et al. 1996; Patton et al. 1999; Stice et al. 2000, 2013; Striegel-Moore et al. 2007), implying that the same risk processes appear to be operating among individuals who enroll in a selective eating disorder prevention program.

There was limited support for the second aim of the study, testing the hypothesis that Body Project participants who showed smaller reductions in risk factors and eating disorder symptoms from pre-to-post would be more likely to show subsequent onset of an eating disorder. The present results did indicate that Body Project participants who showed smaller pre-post reductions in eating disorder symptoms were significantly more likely to show eating disorder onset; however, this effect became nonsignificant after controlling for baseline levels of eating disorder symptoms. Indeed, in the present data, baseline levels of eating disorder showed a strong negative relation with pre-post change in symptoms ($r = -0.82$). Analyses for the second aim functionally provide further evidence that individuals who entered the trials with the greatest eating disorder symptoms were at greatest risk for subsequent eating disorder onset, despite completing the Body Project eating disorder prevention program.

The evidence that participants who developed an eating disorder despite completing the Body Project started with initial elevations in eating disorder risk factors and symptoms appears to have three implications regarding improving the yield of prevention efforts. First, findings suggest a need for more intensive variants of this prevention program for women who have already shown emergence of significant eating disorder symptoms. The marked elevation in eating disorder symptoms at pretest among Body Project participants who later developed an eating disorder suggests that this factor

could be used to identify participants who may benefit from an intensive variant of this intervention. For instance, these individuals might benefit from a more intensive, indicated variant of the Body Project that directly addresses the presence of eating disorder symptoms, such as exercises directly designed to create feelings of cognitive dissonance regarding engaging in any disordered eating (e.g., participants could be asked to discuss the negative effects of eating disordered behaviors in sessions and in home exercises). Recruitment for the original Body Project is based on elevated levels of body dissatisfaction, and many participants who enroll reported no eating disorder symptoms; thus, an explicit focus on current eating disorder symptoms for these latter participants would be inappropriate.

In this context, it is important to note that prior research has found that the Body Project produces stronger reductions in eating disorder symptoms for participants with elevated eating disorder symptoms at pretest (Stice et al. 2008b; Müller and Stice 2013). The present results taken in conjunction with the earlier findings suggest that there is a natural negative relation between baseline levels of a variable and subsequent change because the people with the highest initial levels of a positively skewed variable have the greatest opportunity to show subsequent reductions over time (Measelle et al. 2006). However, results also suggest that a subset of these participants are simultaneously at elevated risk of developing an eating disorder, which also makes sense because they enter the program already symptomatic. The Cox proportional hazard model indicated that Body Project participants were most likely to show onset of an eating disorder 10–30 months after baseline, rather than immediately after baseline, suggesting that the elevated eating disorder symptoms at pretest were not simply a prodromal phase of a later eating disorder.

The second potential implication based on the present findings is to develop an effective version of the Body Project for younger adolescent girls who presumably would have lower levels of the risk factors and symptoms. Eating disorder risk factors may develop in early adolescence (e.g., Pearson et al. 2012), but eating disorder symptoms tend to develop during or after the high school period (e.g., Stice et al. 1998). Complicating this recommendation is the finding from meta-analytic reviews of eating disorder prevention programs (e.g., Stice et al. 2007a) that the effects for currently available interventions are in general significantly larger for older (i.e., 15 years of age or older) compared to younger adolescents. Thus, new programs need to be developed and evaluated for younger populations. Hypothetically, the risk for development of eating disorders despite completing a prevention program would be lower if the intervention was delivered when the risk factors and symptoms were at lower levels.

Given the finding that individuals who begin the Body Project with elevated negative affect are at greater risk for onset of eating disorders, it would be useful to refine this

prevention program so that it produces larger reductions in negative affect. It might be valuable to add exercises from cognitive behavioral therapy for depression to this prevention program, such as cognitive restructuring or behavioral activation, to more directly reduce this risk factor. It might also be useful to have participants discuss the costs of negative affect and the benefits of doing pleasant activities and other things that reduce negative affect, such as exercise, to increase motivation for positive changes that should reduce affective disturbances. However, it will be important for future trials to compare versions of the Body Project with and without content focused on reducing negative affect, as introducing a new intervention component to this prevention program could dilute the effects of the Body Project on previously supported outcomes. It might also be most efficient to have the additional content focused on negative affect reduction delivered solely to the subset of participants who enter the intervention with elevated negative affect scores.

It was noteworthy that the present results revealed that the Body Project significantly reduced risk for onset of DSM-IV eating disorders, but a similar effect did not emerge for DSM-5 eating disorders. This difference across nosologic systems may imply that thin-ideal internalization, which is the primary target of the Body Project and has been shown to mediate the effects of the Body Project on other outcomes (e.g., Stice et al. 2007b), plays a more pronounced etiologic role in onset of DSM-IV eating disorders, relative to the newly proposed DSM-5 eating disorders, which are somewhat different.

Study limitations should be considered when interpreting these findings. First, the small number of Body Project participants who developed an eating disorder did not provide an opportunity to examine the study outcome as a function of specific eating disorder diagnoses. Second, it is possible that participants who did not meet criteria for DSM-5 eating disorder diagnoses during the 3-year follow-up period may develop an eating disorder in the more distant future, which would have the effect of reducing sensitivity of the analyses. Third, negative affect was assessed by two different measures in the two studies. Despite the use of two measures, these instruments are highly correlated, and no differential effects emerged from the separate measures. Finally, whereas this study examines individual risk factors, data were not available to examine specific intervention variables, such as dosage or treatment engagement, or the impact of known contextual factors, such as peer or familial environment.

In conclusion, the present results suggest that the small subset of participants who complete the Body Project but still experience onset of a DSM-5 eating disorder during follow-up entered the program with initially elevated levels of three of the four examined risk factors and with elevated eating disorder symptoms. We found little evidence to support the hypothesis that participants who showed smaller pre-post reductions in risk factors and symptoms were at elevated risk for

future eating disorder onset. We contend that analyses such as these have the potential to improve the overall public health yield of eating disorder prevention efforts by informing the future design and adaptation of programs targeting individuals less likely to respond to current evidence-based interventions. Additionally, these results have significant heuristic value beyond the field of eating disorder prevention, and applying this approach to programs aimed to reduce other physical and mental health problems will improve the overall public health yield of diverse prevention efforts.

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Conflict of Interest The authors declare that they have no conflict of interest.

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