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# A Prospective Test of the Temporal Sequencing of Risk Factor Emergence in the Dual Pathway Model of Eating Disorders

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## Abstract

**Objective:** Prospective studies have identified risk factors that predict future onset of eating disorders, but none has provided a test of a of the temporal sequencing of the emergence of risk factors hypothesized in a multivariate etiologic model of eating disorder development.

**Method:** Using data from an 8-year prospective study of 496 adolescent girls, we first conducted receiver operator characteristic plots to identify cut-points for each risk factor that optimally predicted future onset of threshold or subthreshold bulimia nervosa, binge eating disorder, and purging disorder. We then used growth curve models to estimate the age at which each participant crossed the disorder-predictive cut-point for each risk factor, or if they did not, during follow-up, permitting a test of whether the risk factors emerged in the sequence hypothesized in the Dual Pathway etiologic model.

**Results:** Overall, 47% of the 51 youth who showed onset of one of these eating disorders first showed emergence of disorder-predictive levels of perceived pressure to be thin and/or thin-ideal internalization, before showing onset of disorder-predictive levels of body dissatisfaction, before showing onset of disorder-predictive levels of dieting and/or negative affect, before showing onset of the eating disorder; another 29% had one of these steps out of order or did not cross one step in this model. Youth who did not show onset of an eating disorder were significantly less likely to cross the disorder-predictive cut-points for each risk factor or to conform to the sequence of risk factor emergence hypothesized in this model.

**Conclusions:** Results provide novel support for the temporal sequencing of risk factor emergence hypothesized in this multivariate etiologic model and suggest that prevention programs that reduce perceived pressure to be thin and thin-ideal internalization among early adolescent girls with these factors should reduce eating disorder onset, as well as downstream risk factors that are also aversive (e.g., body dissatisfaction and negative affect).

**General Scientific Summary:** Finding suggest that typically, eating disorder-predictive levels of perceived pressure to be thin and pursuit of the thin beauty ideal emerge before disorder-predictive levels of body dissatisfaction, which emerge before disorder-predictive levels of dieting and negative affect, which emerge before onset of threshold or subthreshold bulimia nervosa, binge eating disorder, or purging disorder. Results imply that prevention programs should reduce

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perceived pressure to be thin and pursuit of the thin beauty ideal among early adolescent girls, as this should both reduce eating disorder onset and emergence of aversive downstream risk factors.

**Keywords**

risk factors; dual pathway model; prospective; bulimia nervosa; binge eating disorder; purging disorder

Approximately13% of females will experience an eating disorder at some point in their lives, which are characterized by chronicity, emotional distress, functional impairment, and increased risk for future obesity, depression, suicide, substance abuse, and mortality (Allen, Byrne, Oddy, & Crosby, 2013; Arcelus, Mitchell, Wales, & Nielsen, 2011; Stice, Marti, & Rohde, 2013). It is critical to elucidate factors that predict future onset of eating disorders, as this should inform etiologic theories, which is vital for guiding the content of optimally effective prevention programs and identifying high-risk subpopulations to target with selective prevention programs. Although several prospective studies have identified risk factors that predict future onset of eating disorders, none have provided a rigorous test of a multivariate model that proposes how the risk factors work in concert to predict future eating disorder onset, including the hypotheses regarding the temporal sequencing of risk factor emergence.

The Dual Pathway model is an etiologic theory that hypothesizes how several well-studied psychosocial risk factors may operate together in a meditational fashion to predict future eating disorder onset (Stice, 1994). This theory hypothesizes that pressure for thinness and pursuit of the thin beauty ideal increases risk for subsequent body dissatisfaction, which in turn increases risk for subsequent dietary restriction and negative affect, which in turn increase risk for subsequent onset of eating disorders characterized by binge eating and compensatory weight control behaviors. A previous study provided evidence that pressure to be thin and pursuit of the thin beauty ideal predicted future increases in body dissatisfaction, that body dissatisfaction predicted future increases in dieting and negative affect, and that dieting and negative affect predicted future increases in bulimic symptoms in adolescent girls followed over 2-years (Stice, 2001). However, no prospective study has provided a rigorous test of the *temporal sequencing* of risk factor emergence hypothesized in this multivariate etiologic theory. The goal of the present report is to introduce a new analytic approach for testing hypotheses regarding the temporal sequencing of risk factor emergence in multivariate etiologic models in relation to emergence of clinically significant eating pathology. This analytic approach was specifically developed to test hypotheses regarding the temporal relations among risk factors in multivariate meditational models, and may therefore prove useful for testing hypotheses about the temporal sequencing of risk factor emergence for other public health problems.

One impediment to testing the temporal sequencing of risk factor emergence is that most risk factors are continuous variables, making it challenging to determine at what *age* participants exhibit a *level* of the risk factor that predicts future eating disorder onset. We introduce a three-step approach that might be useful for addressing this important research question. First, receiver operating characteristic (ROC) plots could be used to determine the

specific cut-point of a risk factor that optimally predicts future onset of eating disorders, balancing predictive sensitivity and specificity. This approach could be used for each risk factor in a multivariate etiologic model to generate reliable cut-points for each risk factor that optimally predict future eating disorder onset. Second, latent growth curve models could be used to generate individual-level slopes and intercepts that reflect individual differences in the development of each risk factor. These individual slopes and intercepts could then be used to estimate the specific age at which each individual participant initially crossed the cut-points for each risk factor that optimally predict future eating disorder onset, or whether the participant did not cross these cut-points during follow-up. Third, the ages at which participants crossed each cut-point could then be used to test hypotheses regarding the temporal sequencing of risk factor emergence posited in multivariate meditational etiologic models, such as the Dual Pathway model. This approach would permit a test of the hypothesis that disorder-predictive levels of perceived pressure to be thin and/or internalization of the thin beauty ideal temporally precede emergence of disorder-predictive levels of body dissatisfaction, which in turn temporally precedes emergence of disorder-predictive levels of dieting and/or negative affect, which in turn temporally precede emergence of eating disorders, as proposed *a priori* (Stice, 1994). We hypothesize that adolescent girls who subsequently develop eating disorders would show emergence of disorder-predictive levels of each of these risk factors in this etiologic model, and in the order specified above, whereas adolescent girls who remain eating disorder free will typically not exhibit disorder-predictive levels these risk factors.

Although methodologists often counsel against dichotomizing continuous variables, such as risk factors and eating pathology, the objective of this report is to introduce a novel statistical approach for examining the temporal sequencing of risk factor emergence as it relates to onset of clinically meaningful eating pathology, which seems to necessitate a focus on discrete events that happen at particular developmental points in time. For this reason, we think it might be necessary to dichotomize the risk factors and the pathological outcome, and to translate these discrete events into developmental ages. Further, several studies have established that participants with threshold or subthreshold eating disorders show significant elevations in psychosocial impairment, negative affect, and mental health service utilization (Allen et al., 2013; Stice et al., 2009; 2013). Thus, we elected to predict age of emergence of threshold or subthreshold eating disorders because this is one defensible way of ensuring that the dichotomous outcome captures clinically meaningful eating pathology.

Studies suggest that risk factors that predict future onset of bulimia nervosa, binge eating disorder, and purging disorder are qualitatively distinct from those that predict future onset of anorexia nervosa. Specifically, prospective studies that used diagnostic interviews to assess eating disorder onset have found that social pressure for thinness, pursuit of the thin beauty ideal, body dissatisfaction, dieting, negative affect, alcohol use, low interoceptive awareness, social support deficits, psychosocial impairment, early puberty, overeating, and elevated BMI 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 & Bohon, 2013; Stice, Gau, Rohde, & Shaw, 2017; Stice, Marti, & Durant, 2011). Similarly, social pressure for thinness, pursuit of the thin beauty ideal, body dissatisfaction, dieting, negative affect, psychosocial impairment, and overeating

predicted future onset of threshold or subthrehold binge eating disorder (Stice et al., 2011; Stice et al., 2017). Likewise, pursuit of the thin beauty ideal, body dissatisfaction, negative affect, dieting, psychosocial impairment, and overeating predicted future onset of threshold or subthreshold purging disorder (Stice et al., 2011a; Stice et al., 2017). In contrast, low BMI, low dieting, and psychosocial impairment predicted onset of threshold or subthreshold anorexia nervosa (Stice & Bohon, 2013; Stice et al., 2017). This pattern of findings implies that the risk factors that predict onset of bulimia nervosa, binge eating disorder, and purging disorder are different from those that predict onset of anorexia nervosa, consistent with the thesis that the dual pathway model predicts emergence of eating disorders characterized by binge eating and compensatory weight control behaviors, rather than anorexia nervosa (Stice, 1994). We therefore offer the more nuanced hypothesis that adolescent girls who subsequently develop threshold or subthreshold bulimia nervosa, binge eating disorder, or purging disorder would show emergence of disorder-predictive levels of each of these risk factors in the Dual Pathway model, and in the order specified in that model, whereas adolescent girls who subsequently develop threshold or subthreshold anorexia nervosa or who remain eating disorder free will typically not exhibit disorder-predictive levels these risk factors.

An improved understanding of the temporal sequencing of risk factor emergence is important for several reasons. First, it would provide a rigorous test of the temporal patterns articulated in multivariate etiologic models, which is important for advancing etiologic theory for these pernicious disorders. Second, this information should elucidate which risk factor emerges first, which would be a logical target for prevention programs because the interventions might interrupt the etiologic cascade of risk factors that theoretically give rise to eating disorder onset at the earliest possible time. Prevention programs that reduce the earliest emerging risk factor may also reduce downstream risk factors, such as body dissatisfaction and negative affect, which are subjectively distressing, irrespective of whether they eventuate in eating disorder onset. Third, information regarding which disorder predictive risk factor emerges first should also identify high-risk sub-populations to target with selective prevention programs. Targeting those at greatest risk, and as early as possible, may allow for more cost-effective and efficacious prevention efforts for mental health problems.

## Methods

### Participants and Procedures

Participants were 496 adolescent girls recruited from public and private middle schools in a large US city ($7^{th}$ or $8^{th}$ grade) who had a mean age of 13 at baseline ($SD = .71$). The sample included 2% Asian/Pacific Islanders, 7% African Americans, 68% Caucasians, 18% Hispanics, 1% Native Americans, and 4% who specified other/mixed racial heritage, which was representative of the schools from which we sampled. There were no ethnic differences in the likelihood of developing an eating disorder when comparing Caucasian ($N = 336$) to non-Caucasian ($N = 160$) participants, $\chi^2(1) = .24$, $p = .628$. Average parental education, a proxy for socioeconomic status, was 29% high school graduate or less, 23% some college,

33% college graduate, and 15% graduate degree, which was representative of the city from which we sampled.

The study was described as an investigation of adolescent mental and physical health. Participants were recruited through an informed consent letter sent to parents of eligible girls that described the study (a second mailing was sent to non-responders). This resulted in a participation rate of 56%, similar to other school-recruited samples that used comparable active consent procedures, structured interviews, and longitudinal follow-up (e.g., 61% for Lewinsohn et al., 2000). Female assessors with at least a bachelor's degree in psychology conducted semi-structured interviews that assessed eating disorder symptoms and recorded participant's weight and height at baseline and seven subsequent years for a total of eight annual assessments (T1 - T8). Assessors attended 24 hours of training wherein they received instruction in structured interview skills, reviewed diagnostic criteria for relevant DSM-IV disorders, observed simulated interviews, and role-played interviews. Assessors were required to demonstrate inter-rater agreement ($k > .80$) with supervisors on tape-recorded interviews prior to collecting data. Assessors also completed annual training workshops to minimize interviewer drift. At each of annual assessment, participants completed questionnaires measuring the risk factors. Participants received a gift certificate or cash for completing each assessment.

## Measures

**Pressure to be thin.—**Perceived sociocultural pressure to be thin from family, friends, dating partners, and the media over the past year was assessed with the 10-item Perceived Sociocultural Pressure Scale (Stice, Marti, & Durant, 2011). Response options ranged from 1 = *none* to 5 = *a lot*. Items were averaged for this scale and those below. This scale has shown internal consistency ($\alpha = .88$), 2-week test-retest reliability ($r = .93$), and predictive validity for future onset of bulimic symptoms (Stice et al., 2011; $\alpha = .85$ at T1).

**Thin-ideal internalization.—**The 6-item Ideal-Body Stereotype Scale–Revised assessed pursuit of the thin beauty ideal over the past year (Stice et al., 2011). Response options ranged from 1 = *strongly disagree* to 5 = *strongly agree*. This scale has shown internal consistency ($\alpha = .91$), 2-week test–retest reliability ($r = .80$), predictive validity for bulimic symptom onset, and sensitivity to intervention effects (Stice et al., 2011; $\alpha = .81$ at T1).

**Body dissatisfaction.—**The 9-item Body Dissatisfaction Scale (Stice et al., 2001) assessed dissatisfaction with various body parts over the past year. Response options ranged from 1 = *extremely satisfied* to 5 = *extremely dissatisfied*. This scale has shown internal consistency ($\alpha = .94$), 3-week test–retest reliability ($r = .90$), predictive validity for eating disorder onset, and sensitivity to intervention effects (Stice et al., 2008, 2011; $\alpha = .94$ at T1).

**Dieting.—**The 10-item Dutch Restrained Eating Scale (van Strien, Frijters, van Staveren, Defares, & Deurenberg, 1986) assesses the frequency of various dieting behaviors over the past year. Response options ranged from 1 = *never* to 5 = *always*. This scale has shown internal consistency ($\alpha = .95$), 2-week test–retest reliability ($r = .82$), convergent validity with self-reported (but not objectively measured) caloric intake, predictive validity for

bulimic symptoms, and sensitivity to intervention effects (Stice et al., 2008; van Strien et al., 1986; α = .91 at T1).

**Negative affect.—**Propensity to become affectively distressed over the past year was assessed with the 12-item Emotionality Scale (Buss & Plomin, 1984). Response options ranged from 1 = *never true of me* to 5 = *always true of me*. This scale showed internal consistency, predictive validity for onset of bulimic symptoms, and convergent validity with alternative measures of emotionality (Patrick, Curtin, & Tellegen, 2002; Stice et al., 2011; α = .80 at T1).

**Eating pathology.—**The semi-structured 30-question Eating Disorder Diagnostic Interview (EDDI; Stice et al., 2013) assessed eating disorder symptoms over the past 3 months at baseline and since previous interview at follow-ups on a month-by-month basis. DSM-IV criteria for eating disorders, as operationalized in Stice et al. (2009), were used. EDDI eating disorder diagnoses have shown sensitivity to intervention effects (Stice et al, 2008). Individuals with EDDI-assessed threshold or subthreshold DSM eating disorders have shown significantly greater psychosocial impairment, emotional distress, and mental health service utilization than individual without these disorders (Stice et al., 2009, 2013), providingh evidence for the validity of DSM eating disorder diagnoses. Diagnoses for threshold and subthreshold eating disorders showed 1-week test-retest reliability (κ = .96) and inter-rater agreement (κ = .86) for a subset of participants in this study (Stice et al., 2009).

## Statistical Methods

First, we plotted the ROC curves for the relation of each continuous risk factor, assessed at age 13, 14, 15, and 16, in predicting onset of threshold or subthreshold bulimia nervosa, binge eating disorder, or purging disorder within the next 4 years from each of these ages. The ROC curve illustrates the accuracy with which a risk factor is able to predict a dichotomous outcome in terms of both specificity (i.e., the ability to correctly predict those who will not develop an eating disorder, or true negatives) and sensitivity (i.e., the ability to correctly predict who will develop an eating disorder, or true positives). Each point on the ROC curve represents a sensitivity/specificity combination (or, more accurately, a combination of sensitivity and 1 – specificity) corresponding to a particular value of the predictor (Zweig & Campbell, 1993). ROC curves can be used to identify the cut-point for the predictor at which both specificity and sensitivity are maximized, which corresponds to the point on the curve closest to the upper left-hand corner of the graph. That is, one uses the cut-point that corresponds to the location of the peak of the ROC graph to determine the cut-point that maximizes both specificity and sensitivity when predicting a dichotomous outcome. We averaged the statistically significant cut-points generated from the plots that separately examined the predictive effects at those 4 ages for each risk factor to generate more reliable cut-points.

Second, because we only assessed the level of each risk factor at one time per year over follow-up, we could not determine the precise age at which each participant crossed the disorder-predictive cut-points for each risk factor. We therefore estimated latent growth

models, in which assessments were aligned with participant age, to generate individual slope and intercept values *for each participant* for each risk factor.

To analyze individual growth in risk factor scores, we fit a series of latent growth models for each risk factor using Mplus 7.1 (Muthén & Muthén, 2012), which provides unbiased estimates in the presence of missing data. In each case, we evaluated the relative contribution of both linear and quadratic terms to model fit using the adjusted Bayesian Information Criterion (BIC); the terms were only retained if they contributed to model fit (i.e., the adjusted BIC was smaller). Once the form of each curve was determined, we reported model fit indices, including the chi-square value ($\chi^2$), comparative fit index (CFI), Tucker-Lewis index (TLI), and root-mean squared error of approximation (RMSEA). CFI/TLI values greater than .95, RMSEA values less than 0.5, and a non-significant $\chi^2$ (or a ratio of $\chi^2/df < 3.0$) indicate good fit (Hu & Bentler, 1999). We then derived individual growth curves for each risk factor for each individual. From this, we could estimate the age at which each individual exceeded, or crossed, the cut-points identified in the first phase of the analysis, if they exceeded the cut-points at any time during follow-up. To ensure that the linear and quadratic growth terms were uncorrelated, we coded the growth parameters such that the intercept would reside in the middle of the curve (i.e., age 17).

Finally, we used these age estimates to test whether participants who subsequently developed threshold or subthreshold bulimia nervosa, binge eating disorder, or purging disorder exhibited the hypothesized sequencing of risk factor emergence or deviated from this sequence. These age estimates also allowed us to test whether participants who did not show onset of one these eating disorders were less likely to show disorder-predictive levels of each risk factor, and whether they typically deviated from the risk factor emergence sequencing hypothesized in the Dual Pathway model.

## Results

### Preliminary Analyses

The means, standard deviations (SD), and ranges for the risk factors at baseline are presented in Table 1. Participants showed onset of bulimia nervosa ($N = 7$, 1.4%), subthreshold bulimia nervosa ($N = 29$, 5.9%), binge eating disorder ($N = 3$, .6%), subthreshold binge eating disorder ($N = 12$, 2.5%), purging disorder ($N = 19$, 3.9%), and subthreshold purging disorder ($N = 13$, 2.7%).

### Cut-Point Analyses

The averaged disorder-predictive cut-points from the ROC models were 1.94 for pressure to be thin, 3.50 for thin-ideal internalization, 3.17 for body dissatisfaction, 2.50 for dieting, and 1.40 for negative affect. The individual cut-points at each age are shown in Table 1. Using individual growth curves for each risk factor, we next calculated the age at which each participant exceeded each of these cut-points, or whether they never reached the cut-points during the follow-up.

### Confirmation of Predictive Validity of Risk Factors

We first evaluated group differences (i.e., those who did not develop an eating disorder over follow-up versus those who did) in terms of their likelihood of exceeding the disorder-predictive cut-points for each risk factor. We used chi-square analysis; the associated contingency tables are presented in Table 2. Participants who did not subsequently develop an eating disorder were less likely to exceed each of the cut-points than those who subsequently developed an eating disorder (though the chi-square analysis for pressure to be thin is only trend-level at $p = .05$). Out of the 429 participants who did not develop an eating disorder, 278 (65%) crossed the cut-point for pressure to be thin; comparatively, 40 of the 51 participants (78%) who developed an eating disorder crossed this cut-point. For the thin-ideal internalization, 228 of 429 (53%) participants who did not develop an eating disorder crossed the cut-point, versus 35 of 51 (69%) of participants who developed an eating disorder. For body dissatisfaction, 178 of 429 (41%) participants who did not develop an eating disorder crossed the cut-point, versus 39 of 51 (77%) of participants who developed an eating disorder. For dieting, 201 of 429 (47%) participants who did not develop an eating disorder crossed the cut-point, versus 42 of 51 (82%) of participants who developed an eating disorder. For negative affect, 223 of 429 (52%) participants who did not develop an eating disorder crossed the cut-point, versus 47 of 51 (92%) of participants who developed an eating disorder.

Although only 5 participants showed onset of threshold or subthreshold anorexia nervosa, for descriptive purposes, we examined how many of these participants crossed the disorder-predictive cut-points. All five (100%) crossed cut-points for pressure to be thin and thin-ideal internalization, but only one (20%) crossed the cut-point for body dissatisfaction, and three out of the five (60%) crossed the cut-points for dieting and negative affect.

### Growth Curve Models for each Risk Factor

Next, we fit growth curves for each risk factor. For the pressure to be thin model, both the linear (6108.69 vs. 6289.10) and quadratic terms (6045.83 vs. 6108.69) contributed to model fit and were retained; the final model demonstrated good fit [CFI = 1.00, TLI = 1.00, RMSEA = .02, $\chi^2(27) = 31.91$, $p = .196$]. For thin-ideal internalization, both the linear (6153.54 vs. 6285.89) and quadratic terms (6107.45 vs. 6153.54) contributed to model fit and were retained; the final model demonstrated good fit [CFI = .98, TLI = .98, RMSEA = .05, $\chi^2(27) = 64.25$, $p < .001$]. For body dissatisfaction, both the linear (6974.08 vs. 7040.61) and quadratic terms (6950.69 vs. 6974.08) contributed to model fit and were retained; the final model demonstrated good fit [CFI = .97, TLI = .97, RMSEA = .05, $\chi^2(27) = 66.58$, $p < .001$]. For dieting, both the linear (7616.20 vs. 7800.76) and quadratic terms (7522.03 vs. 7616.20) contributed to model fit and were retained; the final model demonstrated good fit [CFI = .98, TLI = .98, RMSEA = .05, $\chi^2(27) = 66.37$, $p < .001$]. For negative affect, both the linear (2085.49 vs. 2262.28) and quadratic terms (2031.27 vs. 2085.49) contributed to model fit and were retained; the final model demonstrated good fit [CFI = .98, TLI = .98, RMSEA = .05, $\chi^2(27) = 49.53$, $p < .001$]. Data for the mean growth curves are presented in Table 3.

For descriptive purposes, we examined the percentage of participants who crossed the various disorder-predictive cut-points, and the mean ages at which this occurred, among those who crossed each cut-point. The percentages and mean ages were as follows: pressure to be thin ($N$ = 333 out of 496 or 67%, $M$ age = 14.77, $SD$ = 2.22); thin-ideal internalization ($N$ = 273 out of 496 or 55%, $M$ age = 14.82, $SD$ = 2.17); body dissatisfaction ($N$ = 228 out of 496 or 46%, $M$ age = 14.20, $SD$ = 2.19); dieting ($N$ = 254 out of 496 or 51%, $M$ age = 14.55, $SD$ = 2.32); and, negative affect ($N$ = 283 out of 496 or 57%, $M$ age = 14.49, $SD$ = 2.07).

## Test of Temporal Sequencing of Risk Factors in the Dual Pathway Model

We then examined the percent of participants who eventually showed onset on an eating disorder that showed the predicted temporal sequencing of risk factor emergence posited by the Dual Pathway Model. Overall, 24 of the 51 participants who developed an eating disorder (47%) followed the hypothesized pathway (see Table 4). Specifically, these 24 participants showed emergence of disorder-predictive levels of pressure to be thin and/or thin-ideal internalization before they showed emergence of disorder-predictive levels of body dissatisfaction, which occurred before they showed onset of disorder-predictive levels of dieting and/or negative affect, which occurred before they showed onset of the eating disorder. For these participants, the average lag time for the first step (pressure to be thin and/or thin-ideal internalization to body dissatisfaction) was 1.0 months ($SD$ = .41), the average lag time for the second step (body dissatisfaction to dieting and/or negative affect) was 8.0 months ($SD$ = 1.61), and the average lag time for the third step (dieting and/or negative affect to emergence of an eating disorder) was 26.8 months ($SD$ = 1.93).

Another 8 participants (16%) crossed each threshold in the hypothesized pathway, but had one step out of order. Of the remaining 19 participants, 7 (14%) crossed thresholds at two steps in the pathway; they tended to map onto the latter portion, although two participants had a step out of order (see Table 4). Eleven participants crossed one step in the pathway, and of these, again the majority followed the latter portion, with two participants having one step out of order (see Table 4). Finally, one participant did not cross enough thresholds for us to map any step.

For the 429 participants who did not develop an eating disorder over the 8-year follow-up, none by definition fulfill the final step in the hypothesized pathway (i.e., dieting and/or negative affect → eating disorder). However, there were 112 (26%) who surpassed enough of the thresholds for us to map the first two steps in the hypothesized pathway. Among these participants, 93 (22%) demonstrated the first two steps in the pathway (i.e., pressure to be thin and/or thin-ideal internalization → body dissatisfaction, and body dissatisfaction → dieting and/or negative affect); for 11 (3%) participants, pressure to be thin and/or thin-ideal internalization emerged after body dissatisfaction, and for 8 (2%) participants, body dissatisfaction emerged after dieting and/or negative affect. Of the 54 participants (13%) who surpassed enough thresholds for us to map one step in the hypothesized pathway, 30 (7%) demonstrated the first step in the pathway (pressure to be thin and/or thin-ideal internalization → body dissatisfaction), and 22 (5%) demonstrated the second step in the pathway (body dissatisfaction → dieting and/or negative affect); for one participant, thin

and/or thin-ideal internalization emerged after body dissatisfaction. The remaining 263 participants (61%) in the sample did not surpass enough thresholds for us to map any step in the hypothesized pathway.

It is possible that some participants who showed eating disorder onset did not conform to the hypothesized temporal sequencing of risk factor emergence because they were from ethnic minority groups that might have different appearance ideal than ethnic majority participants. However, among those participants who developed an eating disorder ($N = 51$), there were no ethnic differences in the likelihood of following the hypothesized temporal sequencing of risk factor emergence when comparing Caucasian ($n = 33$) to non-Caucasian ($n = 18$) participants; $\chi^2(1) = .75$, $p = .388$.

For descriptive purposes, we evaluated the pathways for the 5 participants who subsequently showed onset of threshold or subthreshold anorexia nervosa. Of these, two had a co-morbid non-anorexia nervosa diagnosis and were already considered above. Of the remaining three, none of them demonstrated the hypothesized sequence of risk factor emergence from the Dual Pathway Model. In general, pressure to be thin and/or thin-ideal internalization emerged later instead of earlier.

## Exploratory Analyses

The Dual Pathway model theorizes how a set of well-characterized risk factors work together to predict future eating disorder onset, focusing on culture pressures to conform to the thin beauty ideal and risk factors that theoretically occur in response to these pressures. Yet, the literature review revealed that several other risk factors have been found to predict future onset of any eating disorder, including low parental support, low peer support, and psychosocial impairment. As we measured these risk factors in the present study, it afforded an opportunity to conduct exploratory analyses that about when disorder-predictive levels of these more recently identified risk factors emerge in comparison to the risk factors in the Dual Pathway Model. Specifically, we tested the hypothesis that low social support from parents, low social support from peers, and psychosocial impairment increase risk for future onset of disorder-predictive levels of thin-ideal internalization, as adolescent girls may pursue the culturally sanctioned appearance ideal to gain social acceptance. Perceived social support was measured with Network of Relationships Inventory (Furman, 1996) items assessing companionship, guidance, intimacy, affection, admiration, and reliable alliance from parents and peers (6 items each). These scales have shown internal consistency ($M\alpha = .88$), 4-week test-retest reliability ($Mr = .69$), and predictive validity (Furman, 1996; Stice, Ragan, & Randall, 2004). Psychosocial impairment in the family, peer group, romantic, and school domains was measured with 17 items from the Social Adjustment Scale-Self Report for Youth (Weismann & Bothwell, 1976). This scale has shown internal consistency ($\alpha = .77$), 1-week test–retest reliability ($r = .83$) and sensitivity to intervention effects (Stice et al., 2008b).

The averaged cut-points from the ROC plots were 4.17 for parental support, 4.67 for peer support, and 2.55 for psychosocial impairment. With regard to the percentage of participants who crossed the various disorder-predictive thresholds, and the mean ages at which this occurred, among those who crossed each threshold, we found the following: parental support

(*N* = 252 out of 496 or 51% crossed the disorder-predictive cut-point and did so at a mean age = 13.00, *SD* = .50); peer support (*N* = 415 out of 496 or 84% crossed the disorder-predictive cut-point; *M* age = 13.00, *SD* = .20); and psychosocial impairment (*N* = 423 out of 496 or 85% crossed the disorder-predictive cut-point, *M* age = 13.02, *SD* = .31). We next tested whether participants who later showed onset of an eating disorder were more likely to cross these disorder-predictive cut-points versus participants who did not show eating disorder onset. Out of the 429 adolescent girls who did not develop an eating disorder, 213 (50%) crossed the cut-point for low parental support; comparatively, 31 of the 51 participants (61%) who developed an eating disorder crossed this cut-point; this difference was not significant, $\chi^2(1) = 2.26$, $p = .133$. For low peer support, 360 of 429 (84%) participants who did not develop an eating disorder crossed the cut-point, compared to 43 of 51 (84%) of participants who developed an eating disorder; this difference weas not significant, $\chi^2(1) = .01$, $p = .942$. For psychosocial impairment, 363 of 429 (85%) participants who did not develop an eating disorder crossed the cut-point, compared to 49 of 51 (96%) of participants who developed an eating disorder; this difference is not significant, $\chi^2(1) = 2.57$, $p = .109$.

We next estimated the growth curve models for these three risk factors. For parental support, both the linear (8097.44 vs. 8219.22) and quadratic terms (8016.93 vs. 8097.44) contributed to model fit and were retained; the final model showed good fit [CFI = .97, TLI = .97, RMSEA = .05, $\chi^2(27) = 65.38$, $p < .001$]. For peer support, both the linear (7741.71 vs. 7812.26) and quadratic terms (7739.24 vs. 7741.71) contributed to model fit and were retained; the final model showed good fit [CFI = .95, TLI = .95, RMSEA = .05, $\chi^2(27) = 64.43$, $p < .001$]. For psychosocial impairment, both the linear (3332.73 vs. 4085.42) and quadratic terms (3302.52 vs. 3332.73) contributed to model fit and were retained; the final model showed moderate fit [CFI = .90, TLI = .89, RMSEA = .10, $\chi^2(27) = 101.88$, $p < .001$].

Among the 51 participants who developed an eating disorder, 20 (39%) showed emergence of disorder-predictive levels of low parental support before showing emergence of disorder predictive levels of thin-ideal internalization, 29 (57%) showed emergence of disorder-predictive levels of low peer support before showing emergence of disorder predictive levels of thin-ideal internalization, and 33 (65%) showed emergence of disorder-predictive levels of psychosocial impairment before showing emergence of disorder-predictive levels of thin-ideal internalization. Out of the 429 participants who did not develop an eating disorder, 117 (27%) showed emergence of disorder-predictive levels of low parental support before showing emergence of disorder predictive levels of thin-ideal internalization, 195 (45%) showed emergence of disorder-predictive levels of low peer support before showing emergence of disorder predictive levels of thin-ideal internalization, and 197 (46%) showed emergence of disorder-predictive levels of psychosocial impairment before showing emergence of disorder-predictive levels of thin-ideal internalization.

## Discussion

To our knowledge, this is the first study to investigate the temporal sequencing of the emergence of risk factors for binge eating and compensatory weight control behavior eating

disorders hypothesized on an *a priori* basis (Stice, 1994). Specifically, the Dual Pathway theory postulates that pressure to be thin and internalization of the thin beauty ideal results in subsequent emergence of body dissatisfaction, which results in subsequent emergence of dieting and negative affect, which increases risk for subsequent emergence of eating disorders characterized by binge eating and/or compensatory weight control behaviors. We used data from a community sample of 496 adolescent girls who completed annual diagnostic interviews over an 8-year follow-up, which afforded a unique opportunity to examine the temporal sequencing of risk factor emergence during adolescence.

The first step in the novel analytic technique for testing hypotheses about the temporal sequencing of risk factor emergence is to determine the levels of each risk factor that optimally predict future eating disorder onset. We plotted ROC curves that generated cut-points that optimally predicted future eating disorder onset, balancing sensitivity and specificity, using data collected at age 13, 14, 15, and 16 to predict eating disorder onset over the subsequent 4 years in each of the 4 ROC plots. We then averaged the cut-points identified in each of the ROC plots for each of the 5 risk factors and used results from the latent growth curve models to determine the age at which *each participant* crossed each disorder-predictive cut-point. The average cut-point for pressure to be thin was 1.9, which corresponded to the response option between *none* and *some*; 67% of the sample crossed this threshold at an average age of 14.8. The average cut-point for thin-ideal internalization was 3.5, which corresponded to the response option of *agree*; 55% of the sample crossed this threshold at an average age of 14.8. The average cut-point for body dissatisfaction was 3.2, which corresponded to the response option between *neutral* and *moderately dissatisfied*; 46% of the sample crossed this threshold at an average age of 14.2. The average cut-point for dieting was 2.5, which corresponded to a response option of *sometime;* 51% of the sample crossed this threshold at an average age of 14.6. The average cut-point for negative affect was 1.4, which corresponded to a response option of *agree*; 57% of the sample crossed this threshold at an average age of 14.5. The fact that on average participants crossed these disorder-predictive cut-points between the ages of 14.2 and 14.8, suggests that it might be optimal to implement eating disorder prevention programs around the age of 13 or 14 to reduce these particular intervention targets.

We next tested whether participants who showed onset of an eating disorder during follow-up were significantly more likely to cross the various disorder-predictive cut-points for each examined risk factor than participants who did not develop an eating disorder. In total, 51 participants who were free of an eating disorder at baseline, when the modal age was 13, showed onset of threshold or subthreshold bulimia nervosa, binge eating disorder, or purging disorder over 8-year follow-up. Analyses confirmed that participants who showed eating disorder onset were significantly more likely to cross each of the disorder-predictive thresholds than participants who did not develop an eating disorder. These results thus provide evidence of the predictive validity of the identified cut-points for these risk factors.

The final step in this novel analytic approach was to determine the percentage of participants who subsequently showed eating disorder onset who exhibited the temporal sequencing of risk factor emergence hypothesized in the Dual Pathway model. Overall, 47% of participants showed emergence of disorder-predictive levels of pressure to be thin and/or thin-ideal

internalization before they showed emergence of disorder-predictive levels of body dissatisfaction, which occurred before they showed onset of disorder-predictive levels of dieting and/or negative affect, which occurred before they showed onset of an eating disorder. Although only 47% of participants who showed onset of an eating disorder exhibited the temporal sequencing of risk factor emergence hypothesized in this model, the null hypothesis would be that only 4% of participants would have been expected to show this particular temporal sequencing ($4 \times 3 \times 2 = 24$; $1/24 = .04\%$). Thus, the fact that 47% of the participants who showed onset of an eating disorder was greater than the 4% expected based on chance provides reasonable support for the hypothesized temporal sequencing of risk factor emergence. Another 16% of participants crossed each cut-point in the hypothesized pathway, but had one step out of order. Further, 14% of participants crossed cut-points at two steps in the pathway, although 2 participants had a step out of order. Thus, 77% of participants conformed to the predicted temporal sequencing of risk factor emergence, or had 1 step out of order or missed only 1 step in the hypothesized etiologic sequence, which provides reasonable support for the temporal sequencing of risk factor emergence hypothesized in the Dual Pathway Model.

Nonetheless, the fact that among those who showed eating disorder onset, 16% had one step out of order implies that the investigated risk factors may show bi-directional relations (e.g., that elevated body dissatisfaction might lead some adolescent girls to subsequently perceive greater pressure for thinness, or that elevated negative affect might contribute to subsequent increases in body dissatisfaction). Further, the fact that another 14% did not cross all cut-points seems to imply that some risk factors may directly increase risk for onset of eating disorders, rather than operating through the hypothesized mediators (e.g., that elevated body dissatisfaction may contribute directly to emergence of compensatory weight control behaviors without first prompting dieting and negative affect). It would be very useful for future research to investigate the most common deviations from the etiologic process hypothesized in this etiologic model using larger data sets, as it is possible that there are qualitatively distinct pathways to onset of these eating disorders that are reliable. It is important to acknowledge that a deviation from the hypothesized temporal sequencing of risk factor does not necessarily mean that the risk factor was not endorsed for those participants, as the risk factor scores might have been just below the disorder-predictive cut-points that we generated.

The data on the average lag between steps for those who conformed to the Dual Pathway Model was also intriguing. Emergence of disorder-predictive levels of body dissatisfaction occurred very rapidly after emergence of disorder-predictive levels of pressure for thinness and thin-ideal internalization, averaging only 1 month. This implies that pressure for thinness and pursuit of the thin beauty ideal may very rapidly result in body dissatisfaction. In contrast, emergence of disorder predictive levels of dieting or negative affect did not occur for an average of 8 months after emergence of disorder-predictive levels of body dissatisfaction, suggesting adolescent girls typically experience prolonged body dissatisfaction before turning to dieting to conform to the thin beauty ideal or experiencing the emergence of negative affect. Strikingly, eating disorders emerged a full 27 months on average after disorder-predictive levels of dieting or negative affect emerged, implying that

the risk from these latter two risk factors accumulate over a very extended period before eating disorder emergence.

We also conducted exploratory analyses that investigated the hypothesis that disorder-predictive levels of low parental support, low peer support, and psychosocial impairment emerge before disorder-predictive levels of thin-ideal internalization, as the former three risk factors have been found to predict future onset of any eating disorder after the Dual Pathway Model was proposed. According to the ROC curves, the average cut-point for low parental support was 4.17, which corresponded to the response option of *agree*; 51% of the sample crossed this cut-point at an average age of 13.0. The average cut-point for low peer support was 4.67, which corresponded to the response option of *strongly agree*; 84% of the sample crossed this cut-point at an average age of 13.0. The average cut-point for psychosocial impairment was 2.55, which corresponded to the response option of *sometimes*; 85% of the sample crossed this cut-point at an average age of 13.0. However, participants who showed eating disorder onset during follow-up were not significantly more likely to cross the disorder-predictive cut-points for these three risk factors that those who did not show eating disorder onset, suggesting these risk factors show less robust relations to future eating disorder onset than the other examined risk factors. Nonetheless, results provided support for our hypothesis that disorder-predictive levels of low parental support, low peer support, and psychosocial impairment temporally precede emergence of disorder-predictive levels of thin-ideal internalization, based on the theory that adolescent girls might pursue the culturally sanctioned thin beauty ideal to gain social acceptance. Of the 51 participants who subsequently developed an eating disorder, 39% showed emergence of disorder-predictive levels of low parental support before showing emergence of disorder predictive levels of thin-ideal internalization, 57% showed onset of disorder-predictive levels of low peer support before showing onset of disorder-predictive levels of thin-ideal internalization, and 65% showed emergence of disorder-predictive levels of psychosocial impairment before showing emergence of disorder-predictive levels of thin-ideal internalization. Thus, these exploratory analyses suggest that these three variables that tap into the broader construct of social support and functioning often emerge before pursuit of the thin beauty ideal emerges.

More generally, the present results suggest that this new analytic approach for testing hypotheses about the temporal sequencing of risk factor emergence represents a potentially useful method for evaluating multivariate etiologic models for psychological disorders or other public health problems. Indeed, this analytic technique might be viewed as an extension of procedures for testing meditational hypotheses (e.g., Baron & Kenny, 1986; Kraemer, Stice, Kazdin, & Kupfer, 2001; Stice, Presnell, Gau, & Shaw, 2007). Although it is necessary to measure risk factors and the public health problem numerous times over a long developmental span, this analytic approach appears to represent a rigorous method of testing complex multivariate meditational etiologic models.

It is important to consider study limitations. First, although participants completed annual assessments from age 13 to 20, it would have been ideal if participants had provided data at younger and older ages to produce a more complete characterization of the emergence of the risk factors and eating disorders. Second, the examined risk factors involved variables implicated in sociocultural etiologic models of eating disorders. It would be useful if future

studies included any biological risk factors that are eventually found to predict future eating disorder onset. Third, although the novel analytic technique is useful in documenting temporal precedence, it does not establish causality, which requires experimental manipulation of the risk factors in a design that controls for potential alternative explanations for reductions in the incidence of eating disorders in the sample (e.g., expectancies or demand characteristics). Fourth, we did not adjust alpha to correct for chance findings that might have emerged because of multiple testing. However, the fact that we only conducted eight inferential tests (whether the disorder-predictive levels of each risk factor were more likely to occur in those who did versus did not develop an eating disorder) suggests that not even one effect would have been expected based on chance.

Results suggest that prevention programs should seek to reduce pursuit of the thin beauty ideal and social pressure for thinness among adolescent girls around the ages of 13 to 14, as this should not only reduce risk for future eating disorders, but also reduce the risk for emergence of body dissatisfaction, unhealthy dieting behaviors, and negative affect. Although eating disorder prevention programs that reduce pursuit of the thin beauty ideal have been developed, it would be useful if prevention programs also targeted pressure for thinness (e.g., by also intervening with parents, siblings, and peers). In addition, the exploratory analyses suggested that it might also be useful to evaluate prevention programs that seek to improve social support and psychosocial functioning, as these risk factors have not been directly targeted in eating disorder prevention programs.

It will be important for future studies to investigate how the other risk factors that have been found to predict future eating disorder onset work in concert with the risk factors examined herein, as this should continue to advance our understanding of the etiologic processes that give rise to these pernicious psychological disorders. Another useful direction for future research would be to develop an analytic approach that can be used to test hypotheses about the temporal sequencing of risk factor emergence using continuous variables to represent risk factors and psychopathology. For instance, with enough assessments it should be possible to fit higher order polynomials and then calculate derivatives that identify the age at which each participant shows an initial increase in each risk factor and eating disorder symptom dimension.

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**Table 1.**

Descriptive data and cut-points for risk factors

| | *Baseline data* | *Cut-points* | | | | |
|---|---|---|---|---|---|---|
| **Risk Factor** | ***M (SD), range*** | ***Age 13*** | ***Age 14*** | ***Age 15*** | ***Age 16*** | ***Overall*** |
| Pressure to be thin | 1.82 (.81), 1.00–5.00 | 2.00 | 1.88 | | | 1.94 |
| Thin-ideal internalization | 3.25 (.72), 1.00–5.00 | 3.50 | | | | 3.50 |
| Body dissatisfaction | 2.80 (1.02), 1.00–5.00 | 3.33 | 3.00 | 3.22 | 3.11 | 3.17 |
| Dieting | 2.22 (.92), 1.00–5.00 | 2.56 | | | 2.44 | 2.50 |
| Negative affect | 1.34 (.37), 1.00–3.15 | 1.38 | 1.38 | 1.46 | 1.38 | 1.40 |

**Table 2.**

Contingency tables evaluating rates at which ED vs. non-ED crossed each threshold

| | ***Pressure to be thin*** | | ***Thin-ideal internalization*** | | ***Body dissatisfaction*** | | ***Dieting*** | | ***Negative Affect*** | |
|---|---|---|---|---|---|---|---|---|---|---|
| | ***No*** | ***Yes*** | ***No*** | ***Yes*** | ***No*** | ***Yes*** | ***No*** | ***Yes*** | ***No*** | ***Yes*** |
| No ED | | | | | | | | | | |
| Observed count | 151 | 278 | 201 | 228 | 251 | 178 | 228 | 201 | 206 | 223 |
| Expected count | 144.8 | 284.2 | 193.9 | 235.1 | 235.1 | 193.9 | 211.8 | 217.2 | 187.7 | 241.3 |
| Non-anorexia ED | | | | | | | | | | |
| Observed count | 11 | 40 | 16 | 35 | 12 | 39 | 9 | 42 | 4 | 47 |
| Expected count | 17.2 | 33.8 | 23.1 | 27.9 | 27.9 | 23.1 | 25.2 | 25.8 | 22.3 | 28.7 |
| Chi-square test | $\chi^2(1) = 3.79$, $p = .052$ | | $\chi^2(1) = 4.41$, $p = .036$ | | $\chi^2(1) = 22.51$, $p < .001$ | | $\chi^2(1) = 22.98$, $p < .001$ | | $\chi^2(1) = 29.90$, $p < .001$ | |

**Table 3.**

Descriptive data for mean growth curve for each risk factor

| | ***Pressure to be thin*** | | ***Thin-ideal internalization*** | | ***Body dissatisfaction*** | | ***Dieting*** | | ***Negative affect*** | |
|---|---|---|---|---|---|---|---|---|---|---|
| | ***M*** | ***Var*** | ***M*** | ***Var*** | ***M*** | ***Var*** | ***M*** | ***Var*** | ***M*** | ***Var*** |
| Intercept | 1.961 | .428 | 3.335 | .357 | 2.871 | .519 | 2.084 | .476 | 1.442 | .106 |
| Linear slope | .052 | .013 | .025 | .006 | .009 | .015 | .010 | .017 | .016 | .001 |
| Quadratic slope | .002 | .002 | −.001 | .001 | −.003 | .002 | .007 | .002 | −.003 | < .001 |

*Note*. *M* = mean. *Var* = variance. Growth curve parameters are coded such that the intercept is at age 17

**Table 4.**

Patterns for those who developed an eating disorder (N = 51)

| | ***PTBT/TI → BD*** | ***BD → Diet/Neg*** | ***Diet/Neg → ED*** |
|---|---|---|---|
| Exceeded cut point at three steps | | | |
| All three steps in the pathway (*n* = 24) | Yes | Yes | Yes |
| Middle/Late pathway (*n* = 4) | No | Yes | Yes |
| Early/Late pathway (*n* = 2) | Yes | No | Yes |
| Early/Middle pathway (*n* = 1) | Yes | Yes | No |
| Middle pathway only (*n* = 1) | No | Yes | No |
| Exceeded cut point at two steps | | | |
| Middle/Late pathway (*n* = 5) | | Yes | Yes |
| Late pathway only (*n* = 2) | | No | Yes |
| Exceeded cut point at one step | | | |
| Late pathway only (*n* = 9) | | | Yes |
| No pathway (*n* = 2) | | | No |
| No cut points crossed (*n* = 1) | | | |

*Note*. PTBT: Pressure to be thin. TI: Thin ideal. BD: Body Dissatisfaction. Diet: Dieting. Neg: Negative affect. ED: Eating disorder.