Interactive and Mediational Etiologic Models of Eating Disorder Onset: Evidence from Prospective Studies

Eric Stice

Oregon Research Institute, Eugene, Oregon 94703; email: estice@ori.org

Annu. Rev. Clin. Psychol. 2016. 12:359–81

First published online as a Review in Advance on November 19, 2015

The *Annual Review of Clinical Psychology* is online at clinpsy.annualreviews.org

This article's doi: 10.1146/annurev-clinpsy-021815-093317

Keywords

risk factors, prospective, pathways, interactive, mediation, eating disorders

Abstract

It is vital to elucidate how risk factors work together to predict eating disorder onset because it should improve the yield of prevention efforts. Risk factors that have predicted eating disorder onset in multiple studies include low body mass index (BMI) for anorexia nervosa; thin-ideal internalization, perceived pressure to be thin, body dissatisfaction, dieting, and negative affect for bulimia nervosa; and body dissatisfaction and dieting for purging disorder. No such risk factors have been identified for binge eating disorder. Classification tree analyses have identified several amplifying interactions, mitigating interactions, and alternative pathway interactions between risk factors, such as evidence that elevated BMI amplifies the risk between appearance overvaluation and the future onset of recurrent binge eating. However, there have been no tests of mediational risk factor models in the prediction of eating disorder onset. Gaps in the literature are identified and procedures for providing rigorous tests of interactive and mediational etiologic models are outlined.

Contents

INTRODUCTION

Eating disorders affect 13% of females and 3% of males (Allen et al. 2013, Stice et al. 2013) and are marked by chronicity, relapse, distress, and functional impairment, and individuals with eating disorders are at increased risk for future obesity, depression, suicide attempts, anxiety disorders, substance abuse, and mortality (Arcelus et al. 2011, Crow et al. 2009, Stice et al. 2013, Swanson et al. 2011). It is thus vital to (*a*) identify risk factors that predict the future onset of eating disorders and (*b*) advance knowledge of multivariate models that specify how these risk factors work in concert to predict the emergence of these syndromes, because this should inform the content of effective prevention programs and identify high-risk populations to target with selective prevention programs.

In this article, results from prospective studies that investigated risk factors that predict future eating disorder onset are first reviewed. Although most did not evaluate multivariate etiologic models, findings from these studies provide the foundation for more complex interactive and mediational multivariate etiologic models. Second, results from prospective studies that used classification tree analyses to investigate interactions between risk factors that may signal differential pathways to disorder onset are summarized. Third, interactive and mediational multivariate etiologic models for the development of eating disorders that were evaluated with prospective data are reviewed. Fourth, gaps in the literature and methodological challenges in providing rigorous evaluations of interactive and mediational multivariate models are discussed. Finally, a procedure for advancing knowledge regarding how multiple risk factors work together to predict eating disorder onset is outlined.

FOUNDATIONAL RISK FACTOR STUDIES

This review primarily focuses on prospective studies that used data collected at baseline to predict future onset of specific eating disorders (e.g., anorexia nervosa, bulimia nervosa, binge eating

disorder) or any eating disorder among initially nonaffected participants. Such studies provide definitive evidence that the risk factors predate emergence of the eating pathology and that they predict emergence of clinically significant eating disorders.

Prospective studies that predicted future change in eating disorder symptoms are not summarized because it is not clear that these studies predicted initial emergence of clinically significant eating pathology. Prospective studies often find that the mean symptom levels decrease over follow-up, which implies that baseline risk factors are predicting symptom persistence versus normative symptom reductions instead of the emergence of new eating pathology. For instance, one study found that binge eating decreased over a one-year period in preadolescence (Pearson et al. 2012), which suggests that the prospective relations of negative urgency (which refers to a tendency to act rashly and engage in potentially harmful behaviors when distressed), and the expectation that eating reduces negative affect, to future change in binge eating are not capturing the emergence of binge eating, as the authors conclude, but rather the maintenance of binge eating. Further, most studies that predicted symptom change did not use diagnostic interviews to assess symptoms, which is a concern because symptoms assessed by questionnaire show poor agreement with gold-standard diagnostic interviews (Black & Wilson 1996, Decaluwe & Braet 2004).

It can be argued that it is useful to identify factors that predict the initial emergence of eating disorder symptoms (e.g., Stice & Agras 1998) because such analyses document temporal precedence. However, these studies have not established that symptom onset is associated with clinically meaningful functional impairment (as has been done for participants who meet criteria for an eating disorder). Moreover, most of the studies that modeled symptom onset used questionnaires rather than diagnostic interviews. Thus, findings from such studies are not summarized.

Risk Factors for Anorexia Nervosa Onset

Only two truly prospective studies were identified that used risk factors assessed at baseline to predict future onset of anorexia nervosa or subthreshold anorexia nervosa among individuals confirmed to be free of an eating disorder at baseline. (All of the studies reviewed in this article included only females unless otherwise noted.) The univariate effects are described because most studies did not report results from multivariate analyses, and the multivariate models reported varied regarding which covariates were included. Low body mass index (BMI) and low dieting predicted onset of threshold or subthreshold anorexia nervosa over a five-year follow-up, but early puberty, perceived pressure for thinness, thin-ideal internalization, body dissatisfaction, negative affect, and social support deficits did not (E. Stice, K. Presnell, and S. Bearman, unpublished manuscript). Low BMI and impaired psychosocial functioning predicted onset of threshold or subthreshold anorexia nervosa over a three-year follow-up in a high-risk sample of young women with body dissatisfaction, but parental education, thin-ideal internalization/positive expectancies regarding thinness, body dissatisfaction, dieting, healthy eating, exercise, denial of costs of pursuing the thin ideal, loss of control when eating, fasting, excessive exercise, negative affect, substance use, and mental health treatment did not (Stice et al. 2015).

Three studies assessed risk factors during a developmental period that predates the typical emergence of anorexia nervosa, which also provides relatively compelling evidence of temporal precedence, but the studies did not confirm that all participants were free of an eating disorder when the baseline data were collected. Vaginal instrumental delivery (e.g., vacuum extraction, forceps), cephalhematoma (hemorrhage of blood between the skull and the periosteum of a newborn), premature birth, low birth weight, and cobirth correlated with anorexia nervosa onset over 10- to 21-year follow-up, but maternal age at childbirth, number of overall maternal births, pregnancy complications, pregnancy hypertension, diabetes, pregnancy bleeding, preterm

membrane rupture, cesarean delivery, and neonatal jaundice did not (Cnattingius et al. 1999). Childhood eating conflicts, struggles around meals, and unpleasant meals correlated with subthreshold anorexia nervosa onset over 8- to 17-year follow-up, but childhood pica, digestive problems, not eating, disinterest in food, picky eating, eating too little, and eating too slowly did not (Kotler et al. 2001). Perfectionism and low BMI correlated with lifetime diagnoses of threshold or subthreshold anorexia nervosa, controlling for baseline eating disorder symptoms, but negative affect, impulsivity, and family functioning did not (Tyrka et al. 2002).

It is encouraging that several studies identified factors that predicted anorexia nervosa onset, because the low incidence of this disorder makes the identification of such factors challenging. However, little consensus has emerged because each study focused on fairly distinct predictors. Variation in the risk factors identified may also have resulted from the fact that the studies used different sampling frames. In risk factor studies, a given variable, by itself or in combination with other variables, may show differential predictive validity in representative samples versus samples of individuals at high risk for eating disorder onset, as was the case with these risk factor studies. Variation in sampling frames likely explains the mixed support that has emerged for risk factors for other eating disorders that are reviewed below. Nonetheless, it was noteworthy that low BMI predicted future onset of threshold or subthreshold anorexia nervosa in three out of three studies (see sidebar Risk Factors that Predicted Onset of Eating Disorders in Multiple Studies). This finding seems to converge with the predictive effects for low birth weight, low dieting, eating conflict, meal struggles, and unpleasant meals. Contrary to accepted theorizing, these findings imply that youth who are ambivalent to food or who undereat for other reasons (high executive control), and therefore have lower BMIs and less need to diet, are at risk for anorexia nervosa. Perfectionism emerged as a predictor in one study, providing some support for the thesis that this personality feature increases risk for anorexia nervosa. Impaired psychosocial functioning predicted anorexia nervosa onset, implying that individuals who have difficulty getting along well with their family, friends, and peers are at risk for this disorder. Negative affect did not show significant predictive effects in two studies. There was no support for thin-ideal internalization, body dissatisfaction, and other factors relating to cultural pressure for thinness, which have been theorized to increase risk for anorexia nervosa. Likewise, there was no evidence that prodromal eating-disordered behaviors (e.g., fasting) increase risk for anorexia nervosa onset.

Risk Factors for Bulimia Nervosa Onset

Seven truly prospective studies that used risk factors assessed at baseline to predict future onset of bulimia nervosa or subthreshold bulimia nervosa among individuals confirmed to be free of an eating disorder at baseline were identified. Adolescent dieters in comparison with nondieters showed greater onset of threshold or subthreshold bulimia nervosa onset over a one-year follow-up (Patton et al. 1990). Weight concerns, drive for thinness, body dissatisfaction, ineffectiveness (which refers to feelings of general inadequacy and ineffectiveness, insecurity, and worthlessness), negative affectivity, dieting, alcohol use, and lower interoceptive awareness predicted onset of threshold or subthreshold bulimia nervosa over a four-year follow-up, but perfectionism, maturity fears, interpersonal distrust, and BMI did not (Killen et al. 1996). Dieting and psychiatric problems predicted bulimia nervosa onset over a three-year follow-up, but peer dieting, daily exercise, and parental separation did not (Patton et al. 1999). Elevated BMI, social pressure for thinness, thin-ideal internalization, dieting, negative affect, social support deficits, and early puberty predicted onset of threshold or subthreshold bulimia nervosa over a five-year follow-up, but body dissatisfaction did not (E. Stice, K. Presnell, and S. Bearman, unpublished manuscript). Dieting and fasting predicted onset of threshold or subthreshold bulimia nervosa over a five-year

RISK FACTORS THAT PREDICTED ONSET OF EATING DISORDERS IN MULTIPLE STUDIES

Anorexia nervosa

Low body mass index

Bulimia nervosa

Thin-ideal internalization
Perceived pressure to be thin
Body dissatisfaction
Dieting
Negative affect

Binge eating disorder

None

Purging disorder

Dieting

follow-up (Stice et al. 2008a). Social pressure to be thin and body dissatisfaction predicted onset of threshold or subthreshold bulimia nervosa over a seven-year follow-up, but thin-ideal internalization, dieting, negative affect, and depressive symptoms did not (Stice et al. 2011). Thin-ideal internalization, positive expectancies from thinness, body dissatisfaction, dieting, denial of the costs of the pursuit of the thin ideal, fasting, impaired social functioning, mental health utilization, and negative affect predicted bulimia nervosa onset over a three-year follow-up in a high-risk sample of young women with body dissatisfaction, but parental education, healthy eating, exercise, excessive exercise, loss of control when eating, BMI, and substance use did not (Stice et al. 2015).

Two studies assessed risk factors during a developmental period that predates the typical emergence of bulimia nervosa, but they did not confirm that all participants were free of an eating disorder when the baseline data were collected. Eating too little during childhood correlated with bulimia nervosa onset over 8- to 17-year follow-up, but childhood pica, digestive problems, eating conflicts, not eating, disinterest in food, picky eating, struggles around eating, eating too slowly, and unpleasant meals did not (Kotler et al. 2001). Controlling for baseline eating disorder symptoms, negative affect correlated with lifetime diagnoses of threshold or subthreshold bulimia nervosa but perfectionism, BMI, impulsivity, and family functioning did not (Tyrka et al. 2002).

In sum, the most consistently identified risk factors are dieting, which predicted bulimia nervosa onset in six out of six studies; thin-ideal internalization, body dissatisfaction, and negative affect, which predicted bulimia nervosa onset in three out of four studies; and fasting, which predicted bulimia nervosa onset in two out of two studies. Results are largely consistent with accepted etiologic theories that focus on cultural pressure for thinness, body dissatisfaction, and dieting. Support was mixed for other risk factors. BMI predicted bulimia nervosa onset in only one out of four studies. Eating too little during childhood, alcohol use, low interoceptive awareness, psychiatric symptoms, mental health utilization, and impaired social functioning predicted bulimia nervosa onset in only one study each. Bulimia nervosa onset was not predicted by perfectionism, maturity fears, interpersonal distrust, dysfunctional family dynamics, peer dieting, exercise, parental separation, childhood pica, digestive problems, eating conflicts, not eating, disinterest in food,

parental education, healthy eating, and loss of control over eating. Thus, results provided little or no support for certain broadly accepted risk factors for bulimia nervosa (e.g., perfectionism).

Risk Factors for Binge Eating Disorder Onset

Only two studies investigated baseline factors that predict future onset of threshold or subthreshold binge eating disorder. Social pressure for thinness predicted onset of threshold or subthreshold binge eating disorder over a seven-year follow-up, but thin-ideal internalization, body dissatisfaction, dieting, and negative affect/depressive symptoms did not (Stice et al. 2011). Thin-ideal internalization, body dissatisfaction, dieting, denial of the costs of pursuit of the thin ideal, loss of control when eating, impaired social functioning, mental health utilization, and negative affect predicted binge eating disorder onset over a three-year follow-up in a high-risk sample of young women with body dissatisfaction, but parental education, healthy eating, exercise, positive expectancies about thinness, fasting, excessive exercise, BMI, and substance use did not (Stice et al. 2015).

The findings across these two studies were inconsistent, potentially because the first study involved a representative sample, whereas the latter study involved a high-risk sample with a higher binge eating disorder onset incidence, which increased sensitivity. Another difference that may have explained variation in effects is that the baseline assessment occurred at age 13 in the first study versus age 18 in the second study.

Risk Factors for Purging Disorder Onset

Only two studies investigated baseline risk factors that predict future onset of purging disorder. Thin-ideal internalization, body dissatisfaction, and dieting predicted purging disorder onset over a seven-year follow-up, but social pressure to be thin, negative affect, and depressive symptoms did not (Stice et al. 2011). Lower parental education, thin-ideal internalization, positive expectancies about thinness, body dissatisfaction, dieting, denial of the costs of pursuing the thin ideal, fasting, impaired social functioning, and negative affect predicted purging disorder onset over a three-year follow-up in a high-risk sample of young women with body dissatisfaction, but healthy eating, exercise, loss of control when eating, excessive exercise, mental health utilization, BMI, and substance use did not (Stice et al. 2015).

Only body dissatisfaction and dieting predicted purging disorder onset in two studies, though low parental education, thin-ideal internalization, positive expectancies regarding thinness, denial of the costs of pursuing the thin ideal, fasting, and impaired social functioning predicted onset in one study each. Social pressure for thinness, healthy eating, exercise, loss of control over eating, excessive exercise, mental health utilization, BMI, and substance use each did not show significant predictive effects in a single study. Again, the mixed findings may have occurred because the first study examined a representative sample and conducted baseline assessments at age 13, whereas the second examined a high-risk sample and conducted the baseline assessment at age 18.

Risk Factors for Onset of Any Eating Disorder

Although it is important to investigate risk factors that predict future onset of specific eating disorders, there is also value in identifying risk factors that predict onset of any eating disorder, as this might inform the design of prevention programs for the full spectrum of eating disorders.

Only five prospective studies have investigated baseline risk factors that predict future onset of any eating disorder. Drive for thinness and dieting predicted onset of eating disorders not otherwise specified over a one-year follow-up, but BMI, body dissatisfaction, ineffectiveness, perfectionism,

interpersonal distrust, interoceptive awareness, psychiatric symptoms, family psychiatric disorders, parental obesity, and family conflicts did not (Santonastaso et al. 1999). Low self-esteem, body dissatisfaction, escape/avoidance coping, and low family social support predicted onset of any eating disorder over a two-year follow-up, but baseline BMI did not (Ghaderi & Scott 2001). Social pressure to be thin and body dissatisfaction predicted onset of any eating disorder over a three-year follow-up, but substance use, parental pressure for thinness, social support, negative life events, and school performance did not (McKnight 2003). Receiving negative comments about eating from siblings, coaches, and teachers; parental overweight; a history of major depression and panic disorder; body dissatisfaction; thin-ideal internalization; and alcohol use predicted onset of any eating disorder over a three-year follow-up among a high-risk sample of young women who endorsed weight concerns, but receiving negative comments about weight, family eating disorders, parental depression and substance use disorders, emotional and sexual abuse, anxiety disorders, dieting, interoceptive awareness, perfectionism, emotional eating, BMI, low self-esteem, social support deficits, coping stategies, negative life events, depressive symptoms, and social impairment did not (Jacobi et al. 2011). Perceived pressure to be thin, thin-ideal internalization, body dissatisfaction, dieting, and negative affectivity predicted onset of any eating disorder over a four-year follow-up, but BMI did not (Rohde et al. 2015).

Four studies assessed risk factors during a developmental period that predates the typical emergence of bulimia nervosa but did not confirm that all participants were free of an eating disorder when the baseline data were collected. Physical neglect and sexual abuse during childhood were correlated with onset of any eating disorders during adolescence (Johnson et al. 2002). Parental separation, solitary eating, and reading teen magazines predicted onset of any eating disorder over an 18-month follow-up, but family history of psychiatric disorders, family stressful life events, and hours of television viewing did not (Martinez-Gonzalez et al. 2003). Controlling for initial eating disorder symptoms, low parental support, negative affect, and body dissatisfaction correlated with eating disorder diagnoses two years later but socioeconomic status, eating disorder in siblings, dysfunctional family dynamics, low peer social support, suicidal ideation/attempts, self-injury, and self-esteem did not (Beato-Fernandez et al. 2004). Female sex, higher childhood BMI, family stress, and social withdrawal correlated with onset of any eating disorder except anorexia nervosa over 3- to 20-year follow-up, but paternal BMI, maternal BMI, maternal drug use, intelligence, self-efficacy, depressive symptoms, self-esteem, social problems, externalizing behaviors, maternal psychiatric problems, and body dissatisfaction and alcohol use did not (Allen et al. 2009, 2014).

In sum, the most consistently identified risk factors are body dissatisfaction, which predicted any eating disorder onset in five out of seven studies; negative affect, which predicted any eating disorder onset in three out of three studies; and thin-ideal internalization, perceived pressure for thinness, dieting, and family support deficits, which predicted any eating disorder onset in two out of two studies. Findings for other factors were more mixed. Maladaptive coping, social problems, and a history of psychiatric disorders and emotional/sexual abuse predicted any eating disorder onset in one out of two studies. Low self-esteem predicted any eating disorder onset in one out of four studies. Elevated BMI predicted any eating disorder onset in one out of five studies. Several risk factors predicted any eating disorder onset in a single study, including solitary eating, reading teen fashion magazines, female sex, social withdrawal, and negative comments about eating. Other factors did not predict any eating disorder onset, including parental pressure for thinness, hours of television viewing, social support deficits, dysfunctional family dynamics, family history of psychiatric disorders, suicidal ideation/attempts, self-injury, emotional eating, self-efficacy, interoceptive awareness, perfectionism, school performance, negative life events, substance use, externalizing symptoms, parental substance use, parental obesity, family/sibling

eating disorders, intelligence, and socioeconomic status. Thus, the risk factors that predicted onset of any eating disorder were more similar to those for bulimia nervosa and binge eating disorder than for anorexia nervosa, which may have occurred because the former two diagnoses have a higher incidence than the latter diagnosis. It was also noteworthy that several widely accepted risk factors were not supported in these studies (e.g., media use, dysfunctional family dynamics, and emotional eating).

CLASSIFICATION TREE ANALYSES PREDICTING ANY EATING DISORDER ONSET

Several prospective studies used classification tree analyses in an exploratory fashion to investigate interactions between risk factors. Classification tree analysis represents a recursive partitioning analytic method that identifies the most potent predictor of a dichotomous event in the full sample (e.g., eating disorder onset) and selects the optimal cut point on that measure (based on sensitivity and specificity) for identifying groups at differential risk for the event (Breiman et al. 1984). This procedure is repeated in a recursive fashion with each successively identified subgroup until there are no remaining risk factors that identify groups at significantly differential risk for the event. If different risk factors emerge for two subgroups from the same parent node, this signifies an interaction between the risk factors involved (i.e., the predictive effects of one risk factor depend on the level of another risk factor). Such interactions may identify qualitatively distinct pathways to a psychiatric disorder. If qualitatively distinct vulnerability groups exist, it may be possible to improve the yield of prevention efforts by targeting different risk factors for these distinct vulnerability groups. Two benefits to classification tree analysis are that it can detect nonlinear interactions and that it specifies cut points on the risk factors, which facilitates identification of high-risk subgroups. One limitation of this data-driven analytic technique is that the model can be overfit to the data, which has prompted some investigators to require that the minimum node size be at least 20 participants and that all splits be statistically significant to reduce the likelihood that this problem will occur (Stice et al. 2002). One can also apply a Bonferroni correction to maintain alpha at $p = 0.05$ across splits (Stice et al. 2012b).

An early classification tree analysis predicted onset of recurrent binge eating over a two-year follow-up among adolescent girls (Stice et al. 2002). Appearance overvaluation was the first predictor: 20% of girls in the upper 50% of appearance overvaluation showed binge eating onset versus 2% for those with lower appearance overvaluation (see **Figure 1**). The fact that this variable emerged on the first split indicates that it had greater predictive power in the full sample than did the other variables in the model, which included dieting, BMI, body dissatisfaction, perceived pressure to be thin, depressive symptoms, self-esteem, emotional eating, modeling of eating-disordered behaviors, and low peer social support. Among girls with low appearance overvaluation, 9% of those in the upper 25% of depression scores showed binge eating onset versus 0% for those with lower depression. Among girls with high appearance overvaluation, 27% of those with a BMI of 18 or greater showed binge eating onset versus 0% for those with a lower BMI. Among girls with high appearance overvaluation and a BMI >18, 42% showed onset of binge eating if they were in the upper 40% of dieting versus 17% for those with lower dieting. Thus, results revealed a four-way interaction between appearance overvaluation, depression, BMI, and dieting. Findings suggested that an elevated BMI amplified the predictive relation between appearance overvaluation and binge eating onset. This amplifying interaction indicates that the attitudinal risk factor of appearance overvaluation only operates among adolescent girls who have an age- and gender-adjusted BMI that places them in the slightly overweight range; thus, for girls who conform to the thin ideal, appearance overvaluation was not associated with binge eating

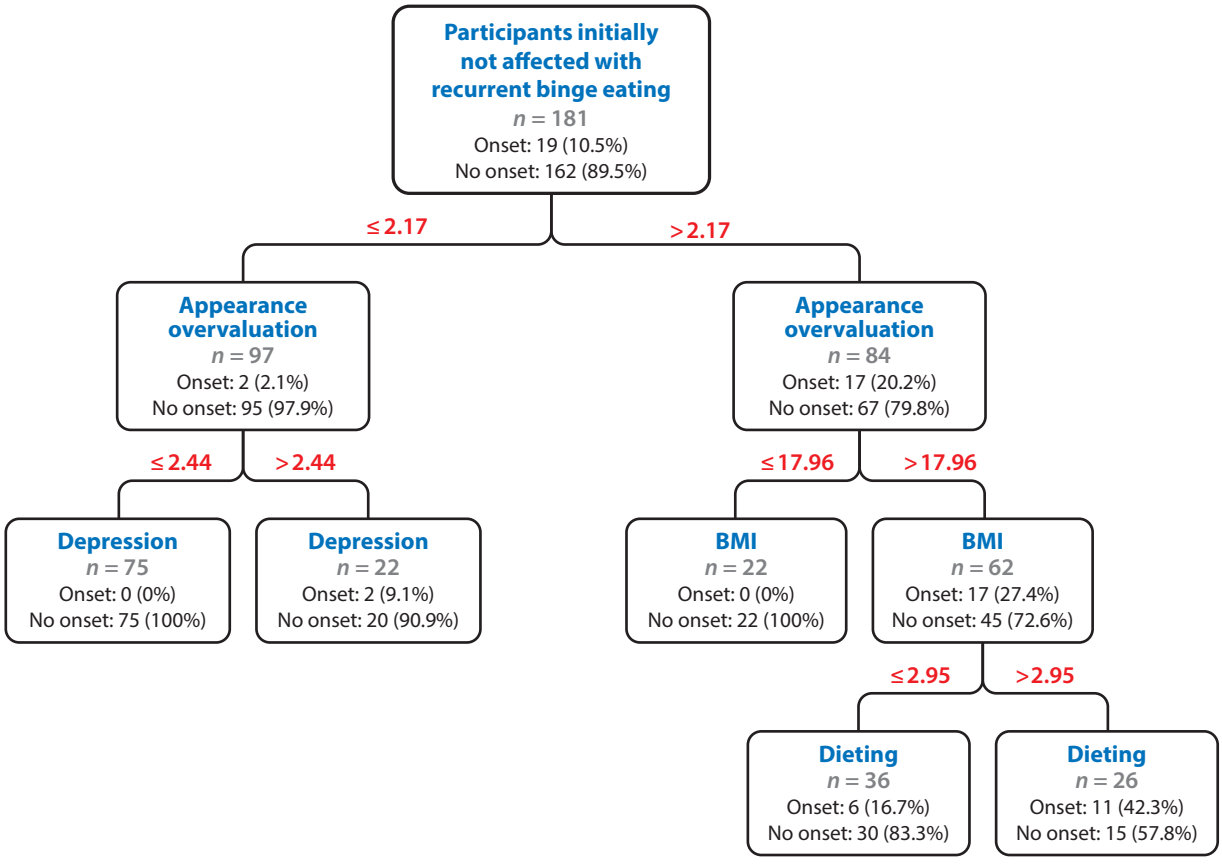

Figure 1

Classification tree analysis decision rules from Stice et al. (2002). The empirically derived cut points are shown, along with the sample size and the incidence and probability for recurrent binge eating onset during the follow-up period for each branch and node. Abbreviation: BMI, body mass index.

onset. Further, results indicated another amplifying interaction wherein dieting increased the predictive effects of the combination of appearance overvaluation and elevated BMI, with almost half the participants with this triple confluence of risk factors showing binge eating onset. Last, results suggested an alternative pathway interaction wherein among adolescent girls with lower appearance overvaluation, elevated depression emerged as a risk pathway, theoretically because depression increases the reward value of food or people turn to eating for mood improvement.

A second classification tree analysis predicted onset of any eating disorder over a seven-year follow-up among early adolescent girls (Stice et al. 2011). One unique feature of this report is that it used the risk factor values measured the year before onset of the eating disorder rather than those measured at baseline, thus capturing more proximal predictive effects. Further, the classification tree analysis predicted survival time to onset of an eating disorder rather than simply whether the participant showed eating disorder onset, illustrating the flexibility of this analytic approach. Body dissatisfaction emerged as the first predictor; 24% of girls in the highest 25% of body dissatisfaction showed eating disorder onset versus 6% among girls with lower body dissatisfaction (see **Figure 2**). Body dissatisfaction had a greater predictive effect than perceived pressure to be

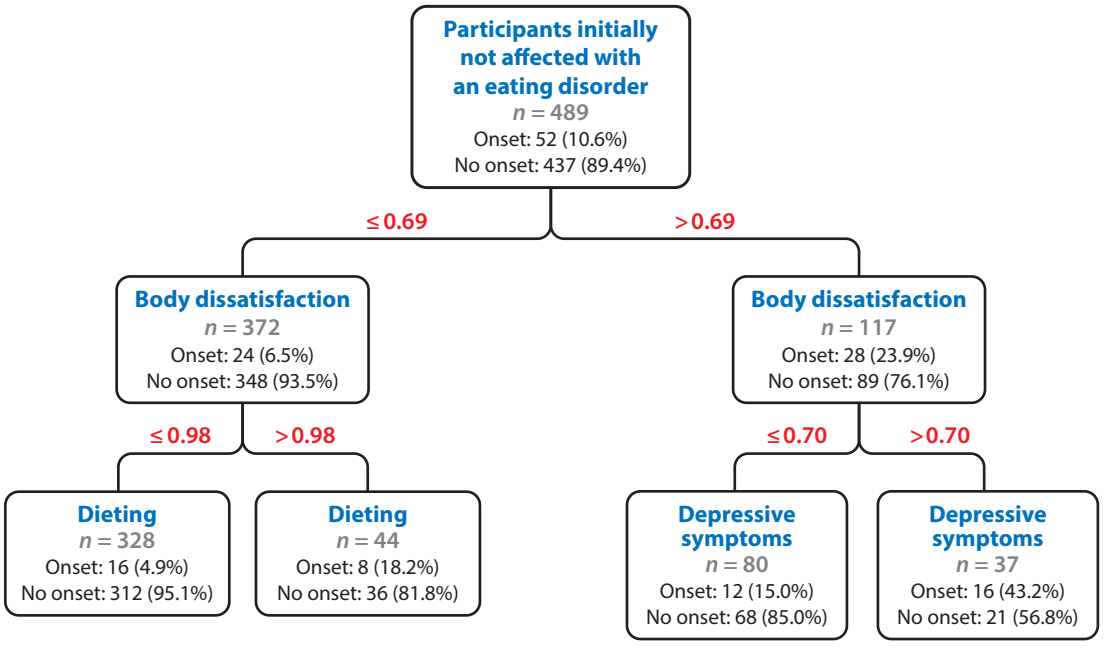

Figure 2

Classification tree analysis decision rules from Stice et al. (2011). The empirically derived cut points are shown, along with the sample size and the incidence and probability for any eating disorder onset during the follow-up period for each branch and node. Note that all predictors were standardized.

thin, thin-ideal internalization, dieting, negative affectivity, and depressive symptoms. Among participants with elevated body dissatisfaction, having a depressive symptom score in the top 33% of the distribution emerged as a predictor; 43% showed eating disorder onset if they had both elevated body dissatisfaction and depressive symptoms versus only 15% if they had only elevated body dissatisfaction. Among participants with low body dissatisfaction, having a dieting score in the upper 12% of the distribution emerged as a predictor; 18% showed eating disorder onset if they had elevated dieting versus 5% with lower dieting. Thus, results imply a three-way interaction between body dissatisfaction, depressive symptoms, and dieting. Results revealed one amplifying interaction in which the predictive effects of body dissatisfaction on eating disorder onset were greater for adolescents with elevated depression. There was also evidence of an alternative pathway interaction in which dieting emerged as a predictor of eating disorder onset, but only in the absence of body dissatisfaction.

A third classification tree analysis predicted onset of any eating disorder over a three-year follow-up among young women with weight concerns in the control condition from an eating disorder prevention trial (Jacobi et al. 2011). Negative comments about eating from a coach or teacher showed the strongest predictive effect; 39% of young women who reported a negative comment about eating showed eating disorder onset versus 8% of those who did not report negative comments. The predictive effects for this risk factor were greater than those for negative comments about eating from siblings, average parental weight, a history of depression, a history of panic disorder, weight concerns, eating concerns, drive for thinness, bulimic symptoms, compensatory behaviors, and alcohol use. For young women who did not receive comments about their eating, a history of depression emerged as a predictor: Those with a history of depression showed an

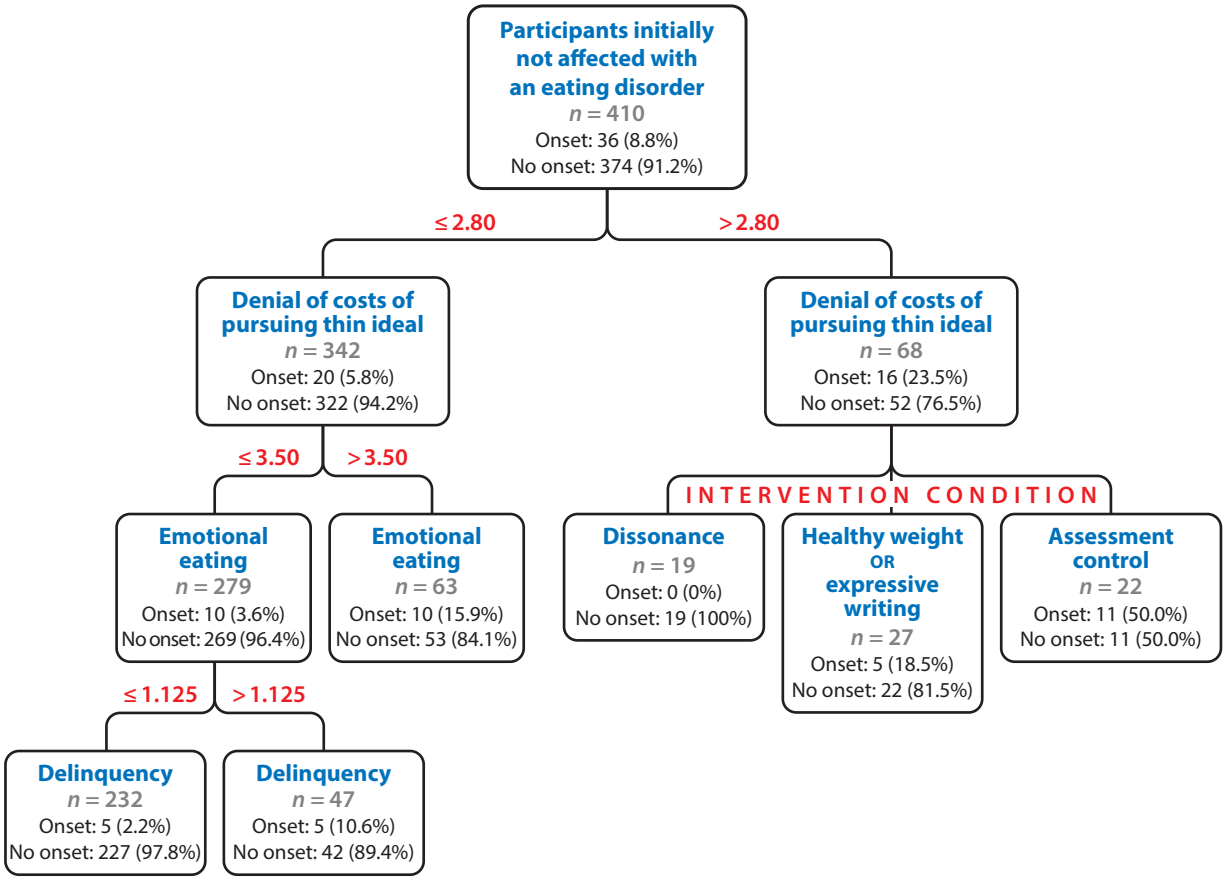

Figure 3

The decision rules from Stice et al. (2012a). The empirically derived cut points are shown, along with the sample size and the incidence and probability for any eating disorder onset during the follow-up for each branch and node.

eating disorder incidence of 30% versus 4% for those without a history of depression. Thus, these findings suggested an alternative pathway interaction between negative comments about eating and depression in the prediction of any eating disorder onset, wherein the predictive effects of depression only emerge in the absence of negative comments about eating.

A fourth classification tree analysis predicted onset of any eating disorder over a three-year follow-up among adolescent girls and young women with elevated body dissatisfaction in an eating disorder prevention trial (Stice et al. 2012a). In contrast to the Jacobi et al. (2011) study, an intervention condition was included in the model, as were participants from each condition. The most potent predictor was denial of the costs of pursuing the thin ideal; 23% of participants in the highest 16% of denial scores showed eating disorder onset versus 6% with lower denial scores (see **Figure 3**). The predictive effect for this risk factor was more potent than for perceived pressure to be thin, thin-ideal internalization, appearance overvaluation, body dissatisfaction, body image distress, readiness to improve body satisfaction, BMI, perceived weight norms, dieting, negative affect, emotional eating, subjective reward from eating, social rejection, externalizing symptoms, substance use, and condition. Among participants with higher denial scores, intervention condition emerged as a predictor: 0% of participants who completed a dissonance-based eating

disorder prevention program showed eating disorder onset versus 19% of those who completed two alternative eating disorder prevention programs and 50% of assessment-only controls. Among participants with lower denial scores, 16% showed eating disorder onset if they were in the upper 18% of emotional eating scores versus 4% for those with lower emotional eating scores. Among participants with lower denial scores coupled with lower emotional eating scores, 11% showed eating disorder onset if they were in the upper 17% of externalizing symptoms versus 2% for those with lower externalizing symptoms. Thus, results revealed a four-way interaction between denial of costs of pursuing the thin ideal, intervention condition, emotional eating, and externalizing symptoms, wherein emotional eating and intervention condition moderated the effects of denial of the costs of pursuing the thin ideal, and externalizing symptoms moderated the effects of emotional eating. Results suggested one mitigating interaction in which the predictive effects of denial scores were reduced if participants completed an effective eating disorder prevention program. One alternative pathway interaction suggested that the predictive effects of emotional eating emerged only for participants with low denial scores. Another alternative pathway interaction implied that the predictive effects of externalizing symptoms emerge only for participants with low denial and low emotional eating scores.

A fifth classification tree analysis predicted onset of any eating disorder (except anorexia nervosa) over a three- to six-year follow-up in a large sample of adolescents (K. Allen, S. Byrne, R. Crosby, E. Stice, manuscript under review). Female sex was the most potent predictor: 18% of females versus 4% of males showed eating disorder onset. The predictive effect for female sex was larger than for BMI, eating/weight/shape concerns, dieting, depression, externalizing symptoms, and internalizing symptoms. Interestingly, elevated eating/weight/shape concerns emerged as a predictor of eating disorder onset for both sexes, and two cut points emerged for this predictor for both sexes, indicating quadratic relations. The incidence of eating disorder onset was 1.2%, 1.5%, and 17.1% for males with low, moderate, and high eating/weight/shape concerns, and 1.8%, 8.2%, and 45.1% for females with low, moderate, and high eating/weight/shape concerns. Thus, the quadratic effect for eating/weight/shape concerns was much stronger for females. Among females with medium eating/weight/shape concerns, externalizing symptoms emerged as a significant predictor: 12% of females in the highest 60% of externalizing symptoms showed eating disorder onset versus 3% of those with lower externalizing symptoms. Thus, results revealed an amplifying interaction between sex and eating/weight/shape concerns, wherein sex amplified the predictive effects of elevated eating/weight/shape concerns on eating disorder onset. There was also evidence of an alternative pathway interaction wherein externalizing symptoms emerged as a predictor of eating disorder onset among females if they had moderate eating/weight/shape concerns.

In sum, classification tree analyses identified four amplifying interactions. The first indicated that elevated BMI amplified the predictive effects of appearance overvaluation on future binge eating onset, which represents the sole interaction wherein a biological factor amplified the predictive effects of a culturally based attitudinal risk factor. The second indicated that dieting amplified the predictive relations of elevated appearance overvaluation and elevated BMI to future binge eating onset, suggesting that the confluence of appearance overvaluation, elevated BMI, and dieting markedly increase risk for binge eating. The third indicated that depression amplified the predictive relation between body dissatisfaction and eating disorder onset, implying that negative affect may play a larger role in amplifying the effects of other risk factors than in showing main effects. The fourth indicated that sex amplified the relation of eating/weight/shape concerns to future onset of eating disorders, which suggests that this attitudinal risk factor may partially account for the risk conveyed by female sex.

Classification tree analyses also identified six alternative pathway interactions. The first interaction indicated that elevated depression predicted binge eating onset only in the absence of

appearance overvaluation. The second implied that dieting predicted eating disorder onset only among body-satisfied youth. The third indicated that depression predicted eating disorder onset solely among adolescents who did not receive a negative comment about their eating. The fourth indicated that the predictive effects of emotional eating for eating disorder onset emerged only among adolescents who did not deny the costs of pursuing the thin ideal. The fifth implied that the predictive effects of externalizing symptoms solely emerged for participants with a combination of low denial of the costs of pursuing the thin ideal and low emotional eating. The sixth indicated that the predictive effects of externalizing symptoms emerged only in the absence of elevated eating/weight/shape concerns. Each of these alternative pathway interactions may identify qualitatively distinct vulnerability pathways to eating disorder onset.

Finally, the classification tree analyses identified only one mitigating interaction. Specifically, evidence indicated that the predictive relation between denial of the costs of pursuing the thin ideal and eating disorder onset emerged only among youth who did not complete an eating disorder prevention program that decreased thin-ideal internalization.

Variation existed both in terms of what risk factor had the greatest predictive power and in the pathways implicated in these studies. This may have resulted because classification tree analysis overfits the models to the data. However, two other factors likely contributed to the variation in findings. First, each study examined a different set of factors, virtually precluding the possibility of more consistent findings. Second, the studies investigated relatively distinct samples, including a mixed-sex representative community sample, early adolescent girls recruited from public and private high schools, adolescent girls from private schools, and young women at high risk for eating disorder onset by virtue of having weight concerns or body dissatisfaction recruited for body acceptance/eating disorder prevention trials. The samples also differed in terms of country of origin and length of follow-up. As noted, variation in sampling frames across risk factor studies likely explained some of the inconsistent findings. Nonetheless, the exploratory nature of classification tree analyses should be kept in mind when interpreting these results.

MULTIVARIATE ETIOLOGIC MODELS OF EATING DISORDER DEVELOPMENT

Numerous investigators have proposed multivariate etiologic models that specify how multiple risk factors may work in concert to predict eating disorder onset. However, only a few have been tested with prospective data, and it appears that no study has tested whether any of these models predict onset of eating disorders. It is vital to elucidate how the various risk factors identified in the prospective studies reviewed previously work together to predict eating disorder onset.

Several etiologic theories have an interactive focus, hypothesizing that certain risk factors amplify the predictive effects of other risk factors. One interactive etiologic model postulated that the confluence of dietary restriction and negative affect would predict binge eating onset, based on the logic that both are risk factors for binge eating (Ruderman 1986). Recent neuroscience findings reveal that both negative affect and caloric deprivation increase the reward value of food (Lemmens et al. 2011, Stice et al. 2013), implying that individuals with the highest level of negative affect that is coupled with caloric restriction should be at greatest risk for binge eating. One prospective study did not find a significant interaction between dieting and negative affect in the prediction of binge eating onset (Stice et al. 2000). Moreover, four of the classification tree analyses included both dieting and negative affect as predictors (i.e., K. Allen, S. Byrne, R. Crosby, E. Stice, manuscript under review; Stice et al. 2002, 2011, 2012a), but none found that dieting and negative affect interacted in the prediction of eating disorder onset. However, a study that used a larger sample found that elevated dieting significantly interacted with low self-esteem in an additive fashion to

predict binge eating onset in females during young adulthood (2.9% of variance explained) but not during adolescence; this interactive effect did not emerge in either developmental period for males (Goldschmidt et al. 2012). This study also found that elevated dieting significantly interacted with elevated depression in an additive fashion to predict binge eating onset in males during young adulthood (0.5% of variance explained) but not adolescence; this interactive effect did not emerge in either developmental period for females. Thus, research has provided little support for this interactive model.

Theorists have posited that chronic exposure to thin models promotes body dissatisfaction, dieting, negative affect, and bulimic symptoms (Levine & Smolak 1996, Thompson et al. 1999). Yet despite the fact that most young women in Western culture are exposed to thin models, only 13% develop an eating disorder, suggesting it would be useful to identify individual difference factors that increase vulnerability to the effects of social pressures for thinness. One study experimentally manipulated exposure to a magazine containing thin models to assess the impact on body satisfaction and related outcomes and to examine moderators that identify youth who are more vulnerable to the effects of exposure to thin models (Stice et al. 2001). This experiment was serendipitous in that the participants requested a magazine subscription to compensate them for completing surveys in a study on risk factors that predict eating disorder symptom emergence. The investigators therefore held a randomized lottery for a subscription to the most popular magazine read by adolescent girls: *Seventeen*. Thus, 50% of the participants received a 15-month subscription to *Seventeen*. A manipulation check confirmed that youth assigned to the *Seventeen* condition showed an increase in the amount of time spent reading fashion magazines, but growth curve models, which are a powerful and flexible method of identifying predictors of change in continuous outcomes, indicated that there were no significant main effects on increases in thin-ideal internalization, body dissatisfaction, dieting, negative affect, and bulimic symptoms. Yet an interaction indicated that receiving *Seventeen* resulted in increases in negative affect for adolescents with initially elevated perceived pressure to be thin, but not for adolescents with lower perceived pressure scores (3.6% of variance explained). A second interaction indicated that *Seventeen* resulted in increases in negative affect for adolescents with initial body dissatisfaction, but not for those with initial body satisfaction (2.3% of variance explained). Further interactions indicated that receiving *Seventeen* resulted in increases in body dissatisfaction, dieting, and bulimic symptoms for adolescents with initial deficits in social support, but not for those adolescents with higher social support (2.3%, 1.3%, and 1.4% of variance explained, respectively, in these outcomes). Results suggest that initial elevations in perceived pressure to be thin, body dissatisfaction, and deficits in social support amplify the effects of exposure to fashion magazines on body dissatisfaction, dieting, negative affect, and bulimic symptoms, suggesting that the adverse effects of exposure to fashion magazines emerge only for initially vulnerable youth.

By extension, the moderators of the effects of exposure to *Seventeen* suggest that interventions that reduce perceived pressure to be thin and body dissatisfaction and increase social support may mitigate the adverse effects of exposure to thin models. In line with this implication, a randomized prevention trial found that completing a dissonance-based body acceptance intervention that reduced thin-ideal internalization and body dissatisfaction eliminated the negative effects of experimental exposure to thin models on body satisfaction and negative affect (in an ostensibly independent experiment) that emerged in control participants who did not complete the body acceptance intervention (Halliwell & Diedrichs 2014).

Vohs and colleagues (1999) propose that the confluence of perfectionism, body dissatisfaction, and low self-esteem predicts the emergence of bulimia nervosa. The combination of body dissatisfaction and perfectionism theoretically results in effective weight control behaviors for individuals with high self-esteem because such individuals are likely to view overweight as a temporary,

changeable situation. In contrast, perfectionistic individuals with low self-esteem putatively respond to overweight with less effective coping skills. The perfectionism × body dissatisfaction × self-esteem interaction predicted increases in bulimic symptoms over a five-week period (0.29% variance explained, $p = 0.048$; Vohs et al. 2001) and over a nine-month period (0.06% variance explained, $p = 0.028$; Vohs et al. 1999) among young women. However, a third independent prospective test of this interactive model in the prediction of increases in bulimic symptoms over a one-year follow-up was not able to replicate the predictive effects of the three-way interaction despite the fact that this study had a larger sample than the previous two studies and hence greater sensitivity (0.04% variance explained, $p = 0.71$; Shaw et al. 2004).

Scholars have also proposed that negative emotionality might interact with weight concerns to predict onset of eating pathology, as negative affectivity could increase the motivation for using unhealthy weight control techniques in response to weight dissatisfaction (Keel & Forney 2013). Somewhat consistent with this hypothesis, one classification tree analysis found that depression amplified the predictive effects of body dissatisfaction on future eating disorder onset (Stice et al. 2011).

Other etiologic theories focused on mediational relations that attempt to characterize multiple pathways to eating disorder onset. The dual-pathway model posits that social pressure to be thin and personal internalization of this appearance ideal contribute to body dissatisfaction, which in turn increases dietary restriction and negative affect, which predict onset of binge eating and unhealthy compensatory behaviors (Stice 2001). One prospective study that used growth curve models found that elevated perceived pressure to be thin and thin-ideal internalization predicted future increases in body dissatisfaction over a two-year follow-up, elevated body dissatisfaction predicted increases in dieting and negative affect over a two-year follow-up, and elevated dieting and negative affect predicted increases in bulimic symptoms over a two-year follow-up (Stice 2001). The dual-pathway model accounted for 23% of the variance in growth in bulimic symptoms, controlling for the effects of baseline symptoms. Additional studies provided prospective tests of several of the hypothesized relations in the dual-pathway model. Perceived pressure to be thin and thin-ideal internalization predicted future onset of body dissatisfaction, bulimic symptoms, and any eating disorder; body dissatisfaction predicted future increases in dieting and onset of bulimic symptoms and threshold/subthreshold bulimia nervosa; and dieting and negative affect predicted future onset of bulimic symptoms, threshold/subthreshold bulimia nervosa, and any eating disorder (Allen et al. 2012, Fairburn et al. 2005, Favaro et al. 2003, Field et al. 1999, Killen et al. 1996, McKnight 2003, Patton et al. 1999, Santonastaso et al. 1999, Stice & Agras 1998, Stice & Whitenton 2002). Thus, to date, prospective support has emerged for the dual-pathway model, and the effect sizes are clinically meaningful, but no research has tested whether it predicts future onset of eating disorders.

Smith and colleagues tested mediational models focusing on predicting increases in binge eating and purging behaviors. The first model hypothesizes that elevated ineffectiveness causes expectations that dieting and thinness will lead to life improvement, which in turn predicts future increases in binge eating and purging behaviors (Combs et al. 2010). Combs and colleagues (2010) confirmed that elevated ineffectiveness predicted future increases in the expectation that dieting and thinness will lead to life improvement, which did predict future increases in binge eating but did not predict purging behaviors. However, the mediational effect explained only 0.26% of the variance in change in binge eating. A second report tested the hypothesis that elevated negative urgency causes increases in the expectancy that eating reduces negative affect, which in turn causes increases in binge eating (Pearson et al. 2012). Pearson and colleagues (2012) confirmed this hypothesis, yet the mediational effect explained only 0.29% of the variance. A third report tested the hypothesis that negative affect causes increases in depressive symptoms, which in turn causes

KEY GAPS IN THE LITERATURE ON RISK FACTORS FOR EATING DISORDER ONSET

1. Few prospective studies have investigated factors that predict future onset of specific eating disorders.
2. No prospective studies have investigated factors that predict future onset of diagnostic levels of eating disorder symptoms (e.g., weekly binge eating for a period of three months).
3. Very few studies have examined the temporal sequencing of the emergence of empirically established risk factors.
4. No study has provided a comprehensive test of potential interactions between a broad range of risk factors.
5. No study has tested the ability of a multivariate interactive or mediational etiologic model to predict future onset of an eating disorder.
6. Research on the validity of scales used to assess putative risk factors is limited.

increases in binge eating (Pearson et al. 2015). This hypothesis was also confirmed; however, the mediational effect explained only 0.22% of the variance. These results underscore the importance of considering the amount of variance explained by effects when evaluating multivariate models. Indeed, it is possible that reporter bias (reliance on a single reporter) or method bias (use of surveys to collect all data) account for these very small mediational effects.

One multivariate model of the maintenance of bulimia nervosa has been tested prospectively. The cognitive behavioral model of bulimia nervosa maintenance proposes that low self-esteem causes overvaluation of weight and shape, which causes elevated dietary restriction, which in turn leads to binge eating and purging behaviors (Fairburn et al. 1993). However, weight and shape overvaluation did not predict persistence of binge eating or compensatory behaviors, and neither self-esteem nor dieting predicted persistence of these symptoms (Fairburn et al. 2003). Thus, results provided little support for this maintenance model of bulimia nervosa.

In sum, the studies reviewed have begun to advance knowledge regarding how risk factors work together to predict future increases in eating disorder symptoms. However, most of these studies generated null findings or explained only a small portion of the variance, and few incorporated many of the risk factors identified in the prospective studies that focused on univariate effects of the risk factors.

GAPS IN THE LITERATURE AND METHODOLOGICAL CHALLENGES TO TESTING MULTIVARIATE INTERACTIVE AND MEDIATIONAL ETIOLOGIC MODELS OF THE EATING DISORDERS

This review has revealed several key literature gaps (see sidebar Key Gaps in the Literature on Risk Factors for Eating Disorder Onset). The first is that few prospective risk factor studies have examined factors that predict future onset of diagnostic interview–assessed eating disorders, particularly anorexia nervosa, binge eating disorder, and purging disorder. It will be important for future studies predicting these outcomes to evaluate a broad range of risk factors implicated in theory or past risk factor studies because the limited overlap in the risk factors examined makes it difficult to determine whether predictive effects replicate. A particularly noteworthy lacuna is that virtually no research has tested whether biological factors, such as individual differences in neural structure or function, predict future onset of these eating disorders. It might be useful to conduct longitudinal follow-ups of youth at elevated risk for eating disorders with a low incidence (e.g., middle school girls with a low BMI for identifying risk factors for anorexia nervosa; middle school girls with body dissatisfaction for identifying risk factors for bulimia nervosa). If additional

prospective studies that identify risk factors that predict future onset of various eating disorders are published, it will eventually be possible to conduct meta-analytic summaries of the findings, which would be informative because the summaries would focus on empirical effect sizes rather than statistical significance, which is sample-size dependent, and would allow the elucidation of moderators that predict variation in effect sizes.

A second gap is that no large prospective study has investigated factors that predict onset of diagnostic threshold levels of eating disorder symptoms summarized in the fourth or fifth editions of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV or DSM-5). Such analyses may be revealing given that certain symptoms (e.g., binge eating, compensatory behaviors) are components of more than one eating disorder. It is therefore possible that the predictors of the emergence of diagnostic threshold levels of eating disorder symptoms may yield results that replicate better than analyses predicting onset of eating disorder syndromes.

A third gap is that little research has attempted to determine the temporal sequencing of the emergence of risk factors that predict eating disorder onset; however, establishing the sequencing would be challenging because many risk factors are continuous variables that show gradual increases over development (Rohde et al. 2015). A related issue is that insufficient attention has been paid to the prospective relations between risk factors. Although several etiologic models postulate that certain risk factors emerge before others, which then subsequently predict later eating disorder onset, no research has established that clinically meaningful levels of the upstream risk factors emerge before clinically meaningful levels of the downstream risk factors or that clinically meaningful levels of the risk factors emerge before eating disorder onset.

A fourth gap is that few studies have systematically tested for interactions between risk factors in the prediction of future eating disorder onset. It will be particularly important to test for interactions between biological risk factors, individual-difference factors, and cultural risk factors. Although classification tree analyses are a practical step in this direction, it might be useful to complement those studies with logistic regression or survival models that test for all possible two-way interactions between risk factors. Admittedly, these analyses would be exploratory and therefore best considered to be hypothesis generating, but with sufficient attention to independent replication, they should advance knowledge regarding etiologic processes.

A fifth gap is that no study has tested the ability of a multivariate interactive or mediational model to predict future onset of eating disorders. The prospective tests of the multivariate models predicted change in symptoms, which does not provide a definitive test that the risk factors temporally precede onset of clinically significant eating disorders. Ideally, the predictive effects of various models would be examined with the same data. A related limitation is that many of the etiologic models that have been evaluated do not account for meaningful variance. This suggests we have yet to identify many of the risk factors that predict eating disorder onset. Alternatively, the minimal amount of variance explained might imply that the way we test etiologic theory has limitations. For instance, the predictive effect of risk factors may be relatively immediate, which would suggest that greater attention should focus on time-varying levels of the risk factors that are lagged over a shorter predictive period rather than testing whether risk factors at baseline predict emergence of eating disorders over a multiyear follow-up. Further, not all individuals show emergence of eating disorders during the same developmental period, which could place an upper limit on the predictive validity of etiologic models. Perhaps rather than anchoring analyses around the baseline assessment, it would be more profitable to anchor prospective analyses around the event of eating disorder onset and focus on levels of putative risk factors immediately before that period (e.g., Stice et al. 2011), which could improve the ability to account for more variance in eating disorder onset.

A final topic that may be obscuring the ability to elucidate etiologic processes is the construct validity of the risk factors. Some risk factors may be tapping the same latent variable. For

example, drive for thinness, thin-ideal internalization, and positive expectancies regarding dieting and thinness scales may all tap the same latent variable. Likewise, body dissatisfaction, weight concern, and shape concern scales may likewise tap the same latent construct. Other risk factors may be proxy measures for the true risk factor. Indeed, certain widely studied risk factors do not appear to assess the constructs they purport to measure. Critically, despite the centrality of dieting to various etiologic models of eating disorders, the field does not appear to have a valid measure of dietary restriction. Individuals with high versus low scores on all widely used dieting scales do not consume fewer calories according to objective measures of intake during single eating episodes (Hetherington et al. 2000, Ouwens et al. 2003, Sysko et al. 2007), multiple eating episodes (Jansen et al. 2003, Martin et al. 2005, Rolls et al. 1997, Sysko et al. 2005), and over 2- to 12-week observation periods (Bathalon et al. 2000, Stice et al. 2007). It will be impossible to examine the role of dieting without a valid measure.

BUILDING EMPIRICALLY BASED INTERACTIVE AND MEDIATIONAL ETIOLOGIC MODELS

Given the limited knowledge of interactive and mediational etiologic models that predict eating disorder onset, it seems useful to outline a procedure for advancing knowledge regarding how multiple risk factors work together to predict disorder onset. The first logical step is to determine which risk factors reliably predict future onset of diagnostic interview–assessed eating disorders and diagnostic levels of eating disorder symptoms. It would be best to examine predictors of the emergence of eating disorders or symptom dimension in separate models because predictors of these outcomes may differ. It would be ideal to examine a broad range of factors, including biological ones, such as elevated reward region sensitivity and inhibitory control deficits. It might be best to first test whether baseline levels of the factors predict onset of the outcomes over follow-up, excluding those that have already shown onset by baseline, as this information is vital for identifying subgroups for selective prevention programs. However, there would also be utility in testing lagged models that examine the predictive effects of the factors one assessment before the emergence of eating disorders or symptom dimension, as the more proximal levels of the factors may have greater predictive power than baseline levels. Although it might be reasonable to set aside factors that do not account for at least 2% of the variance in onset of these outcomes, this may be premature because such risk factors could interact with others in predicting eating disorder onset.

It seems necessary to use separate lines of inquiry to investigate interactive and mediational relations among the risk factors. With regard to interactions, the next step would be to conduct exploratory tests regarding interactive effects between the potential risk factors. It seems reasonable to systematically test for all two-way interactions between each pair of factors in predicting of onset of eating disorders or symptom dimensions in logistic regression or proportional hazard models. Theoretically, the latter are more sensitive as they can detect variation in timing of event onset as well as differences in the incidence of event onset. However, because those models are not optimal for detecting nonlinear interactions, there may be value in conducting focused classification tree analyses that include each pair of potential risk factors, as this would provide an optimally sensitive method of detecting nonlinear interactions. Admittedly, this data-driven approach would yield chance findings, which means that it would be important to conduct independent tests to determine which of the interactions replicate. Split-half replication would be another option. It would also be useful to conduct classification tree analyses with all of the potential risk factors in a data set, as results may reveal more complex interactions between predictors that identify qualitatively distinct vulnerability pathways to onset of eating disorders or symptom

dimensions. However, it would be important to conduct confirmatory tests of the resulting classification tree. This should be possible if one dichotomizes the risk factors as indicated in the results from the classification tree analyses and then uses logistic regression or hazards models to test for the main and interactive effects specified in the classification tree analysis results.

With regard to advancing knowledge on mediational relations between risk factors, a somewhat different line of inquiry is needed. A useful first step might be to focus on establishing the temporal order of risk factor emergence. One potentially fruitful approach would be to use receiver operator characteristic models, which are essentially classification tree analyses models in which only one predictor is entered, to identify thresholds of risk factors that optimally predict onset of eating disorders or symptom dimensions. One could use these data to characterize the ages at which girls typically show emergence of the pathology-predictive levels of the risk factors. This information should guide development of mediational etiologic models. The second step would be to test whether earlier emerging risk factors predict subsequent onset of pathology-predictive levels of risk factors that emerge later in development. An alternative would be to test whether the earlier emerging risk factors predict subsequent increases in the development of downstream continuous risk factors (see Stice 2001).

Given that it is always possible that some omitted confound predicts both the putative risk factor and eating pathology in nonexperimental observational prospective studies, it is vital to confirm the causal status of ostensive risk factors experimentally. For ethical reasons, it is preferable to reduce the ostensive risk factors in randomized prevention trials. Ideally, these prevention trials will focus on single risk factors, rather than multiple risk factors, to permit more precise inferences. It would also be ideal if these trials used a credible alternative intervention that contains nonspecific factors, but not the active ingredients that theoretically should reduce the risk factor (e.g., a supportive-expressive group condition) as a comparison condition because expectancies and demand characteristics inherent to randomized prevention trials may result in reductions in outcomes in the active intervention condition but not in assessment-only control conditions. Such a program of research should simultaneously provide a definitive experimental test of etiologic theory and advance knowledge regarding the design of more effective eating disorder prevention programs. A few prevention trials have evaluated interventions that sought to reduce single risk factors [e.g., thin-ideal internalization (Stice et al. 2008b), body dissatisfaction (Bearman et al. 2003), and negative affect (Burton et al. 2007)]. These trials have provided support for the roles of thin-ideal internalization and body dissatisfaction in the etiology of eating disorders but have generated mixed support for the role of negative affect in the etiology of eating disorders. Randomized prevention trials have also revealed that interventions that promote dietary restriction for weight control do not increase eating disorder symptoms (Groesz & Stice 2007, Presnell & Stice 2003), as suggested by restraint theory. Indeed, a prevention program that promotes gradual lasting reductions in caloric intake and lasting increases in physical activity for the attainment and maintenance of a healthy weight is the only prevention program that has significantly reduced eating disorder onset over a multiple-year follow-up in two trials (Stice et al. 2008b, 2012b). The fact that prospective studies have found that individuals who reported dieting showed elevated future onset of eating disorders (Killen et al. 1996; Patton et al. 1999; Rohde et al. 2015; Santonastaso et al. 1999; Stice et al. 2008a, 2011), but that interventions that increase dietary restriction (confirmed by reductions in directly measured BMI) reduced future eating disorder symptoms and eating disorder onset, suggests that the predictive effects of dieting may reflect the operation of a proxy risk factor (e.g., body dissatisfaction that prompts desires to reduce caloric intake). The evidence that an intervention that promotes reductions in caloric intake and exercise, with the goal of balancing caloric intake with expenditure, reduced eating disorder onset implies that overeating is a risk factor for eating disorder onset. It will be vital for future studies to investigate this possibility.

By extension, randomized prevention trials also offer a rigorous method for investigating interactions between risk factors. A 2 × 2 factorial prevention trial could evaluate the main and interactive effects of prevention programs that reduce two separate risk factors (e.g., negative affect and impulsivity). Likewise, a randomized prevention trial that targets an upstream risk factor might provide a rigorous experimental test of a mediational model. One could test whether intervention versus control participants show greater reductions in the targeted upstream risk factor (e.g., body dissatisfaction), eating disorder onset, and any mediators that are thought to account for the relation of the targeted upstream risk factor and eating pathology (e.g., negative affect).

CONCLUSIONS

Although marked advances in the understanding of risk factors that predict onset of eating disorders have occurred over the past two decades, the understanding of how various risk factors work together to predict emergence of eating disorders is still limited. Large prospective risk factor studies, in conjunction with randomized prevention trials that confirm the causal role of these risk factors in the emergence of eating disorders, have the potential to improve our ability to design more effective prevention programs for these pernicious disorders and advance knowledge regarding which high-risk populations to target with selective prevention programs. Ultimately, if we are able to broadly implement prevention programs that reduce future onset of eating disorders, we will reduce the population prevalence of these conditions.

DISCLOSURE STATEMENT

The author is not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

LITERATURE CITED

Allen K, Byrne S, Crosby R, Stice E. 2015. Sex-specific pathways to binge eating and purging eating disorders: classification tree analysis. Manuscript under review

Allen K, Byrne S, Forbes D, Oddy W. 2009. Risk factors for full- and partial-syndrome early adolescent eating disorders: a population-based pregnancy cohort study. *J. Am. Acad. Child Adolesc. Psychiatry* 48:800–9

Allen K, Byrne S, McLean N. 2012. The dual-pathway and cognitive-behavioral models of binge eating: prospective evaluation and comparison. *Eur. Child Adolesc. Psychiatry* 21:51–62

Allen K, Byrne S, Oddy H, Crosby R. 2013. Eating disorders in adolescents: prevalence, stability, and psychosocial correlates in a population-based sample of male and female adolescents. *J. Abnorm. Psychol.* 122(3):720–32

Allen K, Byrne S, Oddy W, Schmid U, Crosby R. 2014. Risk factors for binge eating and purging eating disorders: differences based on age of onset. *Int. J. Eat. Disord.* 47:802–12

Arcelus J, Mitchell A, Wales J, Nielsen S. 2011. Mortality rates in patients with anorexia nervosa and other eating disorders: a meta-analysis of 36 studies. *JAMA Psychiatry* 68:724–31

Bathalon G, Tucker K, Hays N, Vinken A, Greenberg A, et al. 2000. Psychological measures of eating behavior and the accuracy of 3 common dietary assessment methods in healthy postmenopausal women. *J. Clin. Nutr.* 71:739–45

Bearman SK, Stice E, Chase A. 2003. Effects of body dissatisfaction on depressive and bulimic symptoms: a longitudinal experiment. *Behav. Ther.* 34:277–93

Beato-Fernandez L, Rodriguez-Cano T, Belmonte-Llario A, Martinez-Delgado C. 2004. Risk factors for eating disorders in adolescents: a Spanish community-based longitudinal study. *Eur. Child Adolesc. Psychiatry* 13:287–94

Black C, Wilson T. 1996. Assessment of eating disorders: interview versus questionnaire. *Int. J. Eat. Disord.* 20:43–50

Breiman L, Friedman JH, Olshen RA, Stone CJ. 1984. *Classification and Regression Trees*. Belmont, CA: Wadsworth

Burton E, Stice E, Bearman SK, Rohde P. 2007. Experimental test of the affect-regulation theory of bulimic symptoms and substance use: a randomized trial. *Int. J. Eat. Disord.* 40(1):27–36

Cnattingius S, Hultman C, Dahl M, Sparen P. 1999. Very preterm birth, birth trauma, and the risk of anorexia nervosa among girls. *JAMA Psychiatry* 56:634–38

Combs J, Smith G, Flory K, Simmons J, Hill K. 2010. The acquired preparedness model of risk for bulimic symptom development. *Psychol. Addict. Behav.* 24:475–86

Crow S, Peterson C, Swanson S, Raymond N, Specker S, et al. 2009. Increased mortality in bulimia nervosa and other eating disorders. *Am. J. Psychiatry* 166:1342–46

Decaluwe V, Braet C. 2004. Assessment of eating disorder psychopathology in obese children and adolescents: interview versus self-report questionnaire. *Behav. Res. Ther.* 42:799–811

Fairburn CG, Cooper Z, Doll HA, Davies BA. 2005. Identifying dieters who will develop an eating disorder: a prospective, population-based study. *Am. J. Psychiatry* 162(12):2249–55

Fairburn CG, Marcus MD, Wilson GT. 1993. Cognitive-behavioural therapy for binge eating and bulimia nervosa: a comprehensive treatment manual. In *Binge Eating: Nature, Assessment, and Treatment*, ed. CG Fairburn, GT Wilson, pp. 361–404. New York: Guilford

Fairburn CG, Stice E, Cooper Z, Doll HA, Norman PA, O'Connor ME. 2003. Understanding persistence of bulimia nervosa: a five-year naturalistic study. *J. Consult. Clin. Psychol.* 71:103–9

Favaro A, Ferrara S, Santonastaso P. 2003. The spectrum of eating disorders in young women: a prevalence study in a general population sample. *Psychosom. Med.* 65(4):701–8

Field AE, Camargo CA Jr, Taylor CB, Berkey CS, Colditz GA. 1999. Relation of peer and media influences to the development of purging behaviors among preadolescent and adolescent girls. *Arch. Pediatr. Adolesc. Med.* 153(11):1184–89

Ghaderi A, Scott B. 2001. Prevalence, incidence and prospective risk factors for eating disorders. *Acta Psychiatr. Scand.* 104:122–30

Goldschmidt A, Wall M, Loth K, Le Grange D, Neumark-Sztainer D. 2012. Which dieters are at risk for the onset of binge eating? A prospective study of adolescents and young adults. *J. Adolesc. Health* 51:86–92

Groesz M, Stice E. 2007. An experimental test of the effects of dieting on bulimic symptoms: the impact of eating episode frequency. *Behav. Res. Ther.* 45:49–62

Halliwell E, Diedrichs P. 2014. Testing a dissonance body image intervention among young girls. *Health Psychol.* 33:201–4

Hetherington M, Bell A, Rolls B. 2000. Pleasure and monotony: effects of repeat exposure on pleasantness, preference and intake. *Br. Food J.* 102:507–21

Jacobi C, Fittig E, Bryson S, Wilfley D, Kraember H, Taylor C. 2011. Who is really at risk? Identifying risk factors for subthreshold and full syndrome eating disorders in a high-risk sample. *Psychol. Med.* 41:1939–49

Jansen A, Theunissen N, Slechten K, Nederkoorn C, Boon B, et al. 2003. Overweight children overeat after exposure to food cues. *Eat. Behav.* 4:197–209

Johnson J, Cohen P, Kasen S, Brook J. 2002. Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *Am. J. Psychiatry* 159:394–400

Keel P, Forney J. 2013. Psychosocial risk factors for eating disorders. *Int. J. Eat. Disord.* 46:433–39

Killen JD, Taylor CB, Hayward C, Haydel KF, Wilson DM, et al. 1996. Weight concerns influence the development of eating disorders: a 4-year prospective study. *J. Consult. Clin. Psychol.* 64(5):936–40

Kotler L, Cohen P, Davies M, Pine D, Walsh T. 2001. Longitudinal relationships between childhood, adolescent, and adult eating disorders. *J. Am. Acad. Child Adolesc. Psychiatry* 40:1434–40

Lemmens S, Rutters F, Born J, Westerterp-Plantenga M. 2011. Stress augments food wanting and energy intake in visceral overweight subjects in the absence of hunger. *Physiol. Behav.* 103:157–63

Levine MP, Smolak L. 1996. Media as a context for the development of disordered eating. In *The Developmental Psychopathology of Eating Disorders*, ed. L Smolak, MP Levine, R Striegel-Moore, pp. 183–204. Mahwah, NJ: Erlbaum

Martin C, Williamson D, Geiselman P, Walden H, Smeets M, et al. 2005. Consistency of food intake over four eating sessions in the laboratory. *Eat. Behav.* 6:365–72

Martínez-González M, Gual P, Lahortiga F, Alonso Y, Irala-Estévez J, Cervera S. 2003. Parental factors, mass media influences, and the onset of eating disorders in a prospective population-based cohort. *Pediatrics* 111:315–20

McKnight I. 2003. Risk factors for the onset of eating disorders in adolescent girls: results of the McKnight longitudinal risk factor study. *Am. J. Psychiatry* 160(2):248–54

Ouwens M, van Strien T, van der Staak C. 2003. Tendency toward overeating and restraint as predictors of food consumption. *Appetite* 40:291–98

Patton GC, Johnson-Sabine E, Wood K, Mann AH, Wakeling A. 1990. Abnormal eating attitudes in London school girls—a prospective epidemiological study: outcome at twelve month follow-up. *Psychol. Med.* 20(2):383–94

Patton GC, Selzer R, Coffey C, Carlin JB, Wolfe R. 1999. Onset of adolescent eating disorders: population based cohort study over 3 years. *BMJ* 318(7186):765–68

Pearson CM, Combs JL, Zapolski TC, Smith GT. 2012. A longitudinal transactional risk model for early eating disorder onset. *J. Abnorm. Psychol.* 121:707–18

Pearson CM, Zapolski TC, Smith GT. 2015. A longitudinal test of impulsivity and depression pathways to early binge eating onset. *Int. J. Eat. Disord.* 48(2):230–37

Presnell K, Stice E. 2003. An experimental test of the effect of weight-loss dieting on bulimic pathology: tipping the scales in a different direction. *J. Abnorm. Psychol.* 112(1):166–70

Rohde P, Stice E, Marti N. 2015. Development and predictive effects of eating disorder risk factors during adolescence: implications for prevention efforts. *Int. J. Eat. Disord.* 48:187–98

Rolls B, Castellanos V, Shide D, Miller D, Pelkman C, et al. 1997. Sensory properties of a nonabsorbable fat substitute did not affect regulation of energy intake. *J. Clin. Nutr.* 65:1375–83

Ruderman A. 1986. Dietary restraint: a theoretical and empirical review. *Psychol. Bull.* 99:247–62

Santonastaso P, Friederici S, Favaro A. 1999. Full and partial syndromes in eating disorders: a 1-year prospective study of risk factors among female students. *Psychopathology* 32(1):50–56

Shaw H, Stice E, Springer D. 2004. Perfectionism, body dissatisfaction, and self-esteem in predicting bulimic symptomatology: lack of replication. *Int. J. Eat. Disord.* 36:41–47

Stice E. 2001. A prospective test of the dual-pathway model of bulimic pathology: mediating effects of dieting and negative affect. *J. Abnorm. Psychol.* 110(1):124–35

Stice E, Agras WS. 1998. Predicting onset and cessation of bulimic behaviors during adolescence: a longitudinal grouping analysis. *Behav. Ther.* 29(2):257–76

Stice E, Akutagawa D, Gaggar A, Agras WS. 2000. Negative affect moderates the relation between dieting and binge eating. *Int. J. Eat. Disord.* 27(2):218–29

Stice E, Burger K, Yokum S. 2013. Caloric deprivation increases responsivity of attention and reward regions to intake, anticipated intake, and images of palatable foods. *NeuroImage* 67:322–30

Stice E, Cooper J, Schoeller D, Tappe K, Lowe M. 2007. Are dietary restraint scales valid measures of moderate- to long-term dietary restriction? Objective biological and behavioral data suggest not. *Psychol. Assess.* 19:449–58

Stice E, Davis K, Miller N, Marti CN. 2008a. Fasting increases risk for onset of binge eating and bulimic pathology: a 5-year prospective study. *J. Abnorm. Psychol.* 117:941–46

Stice E, Marti N, Durant S. 2011. Risk factors for onset of eating disorders: evidence of multiple risk pathways from an 8-year prospective study. *Behav. Res. Ther.* 49:622–27

Stice E, Marti N, Rohde P. 2013. Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *J. Abnorm. Psychol.* 122:445–47

Stice E, Marti N, Spoor S, Presnell K, Shaw H. 2008b. Dissonance and healthy weight eating disorder prevention programs: long-term effects from a randomized efficacy trial. *J. Consult. Clin. Psychol.* 76:329–40

Stice E, Presnell K, Spangler D. 2002. Risk factors for binge eating onset: a prospective investigation. *J. Health Psychol.* 21:131–38

Stice E, Rohde P, Gau J, Shaw H. 2012a. Effect of a dissonance-based prevention program on risk for eating disorder onset in the context of eating disorder risk factors. *Prev. Sci.* 13:129–39

Stice E, Rohde P, Shaw H, Gau J. 2015. Risk factors for future onset of anorexia nervosa, bulimia nervosa, binge eating disorder, and purging disorder: results from a prospective high-risk study. Manuscript under review

Stice E, Rohde P, Shaw H, Marti CN. 2012b. Efficacy trial of a selective prevention program targeting both eating disorder symptoms and unhealthy weight gain among female college students. *J. Consult. Clin. Psychol.* 80(1):164–70

Stice E, Spangler D, Agras WS. 2001. Exposure to media-portrayed thin-ideal images adversely affects vulnerable girls: a longitudinal experiment. *J. Soc. Clin. Psychol.* 20:271–89

Stice E, Whitenton K. 2002. Risk factors for body dissatisfaction in adolescent girls: a longitudinal investigation. *Dev. Psychol.* 38:669–78

Swanson S, Crow S, Le Grange D, Swendsen J, Merikangas K. 2011. Prevalence and correlates of eating disorders in adolescents: results from the National Comorbidity Survey Replication Adolescent Supplement. *JAMA Psychiatry* 68:714–23

Sysko R, Walsh BT, Schebendach J, Wilson GT. 2005. Eating behaviors among women with anorexia nervosa. *J. Clin. Nutr.* 82:296–301

Sysko R, Walsh BT, Wilson GT. 2007. Expectancies, dietary restraint, and test meal intake among undergraduate women. *Appetite* 49:30–37

Thompson J, Heinberg LJ, Altabe M, Tantleff-Dunn S. 1999. *Exacting Beauty: Theory, Assessment and Treatment of Body Image Disturbance*. Washington, DC: Am. Psychol. Assoc.

Tyrka A, Waldron I, Graber J, Brooks-Gunn J. 2002. Prospective predictors of the onset of anorexic and bulimic syndromes. *Int. J. Eat. Disord.* 32:282–90

Vohs KD, Bardone AM, Joiner TE, Abramson LY, Heatherton TF. 1999. Perfectionism, perceived weight status, and self-esteem interact to predict bulimic symptoms: a model of bulimic symptom development. *J. Abnorm. Psychol.* 108:695–700

Vohs KD, Voelz ZR, Pettit JW, Bardone AM, Katz J, et al. 2001. Perfectionism, body dissatisfaction, and self-esteem: an interactive model of bulimic symptom development. *J. Soc. Clin. Psychol.* 20:476–97