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Who is really at risk? Identifying risk factors for subthreshold and full syndrome eating disorders in a high-risk sample

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Who is really at risk? Identifying risk factors for subthreshold and full syndrome eating disorders in a high-risk sample

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Background. Numerous longitudinal studies have identified risk factors for the onset of most eating disorders (EDs). Identifying women at highest risk within a high-risk sample would allow for focusing of preventive resources and also suggests different etiologies.

Method. A longitudinal cohort study over 3 years in a high-risk sample of 236 college-age women randomized to the control group of a prevention trial for EDs. Potential risk factors and interactions between risk factors were assessed using the methods developed previously. Main outcome measures were time to onset of a subthreshold or full ED.

Results. At the 3-year follow-up, 11.2% of participants had developed a full or partial ED. Seven of 88 potential risk factors could be classified as independent risk factors, seven as proxies, and two as overlapping factors. Critical comments about eating from teacher/coach/siblings and a history of depression were the most potent risk factors. The incidence for participants with either or both of these risk factors was 34.8% (16/46) compared to 4.2% (6/144) for participants without these risk factors, with a sensitivity of 0.75 and a specificity of 0.82.

Conclusions. Targeting preventive interventions at women with high weight and shape concerns, a history of critical comments about eating weight and shape, and a history of depression may reduce the risk for EDs.

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Key words: Eating disorders, high-risk, longitudinal study, risk factors.

Introduction

Approximately 1–3% of the young adult female population suffer from full syndrome eating disorders (EDs), which include anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED) (Hoek & van Hoeken, 2003; Striegel-Moore et al., 2003; Hudson et al., 2007). Rates of subthreshold EDs have been reported to exceed those of full syndrome EDs, with the combined rates easily exceeding 4%, and subthreshold or partial syndrome EDs exist on a continuum with full syndrome EDs and represent similar levels of functional impairment (Fitzgibbon et al., 2003). ED attitudes and behaviors can have serious psychological and physical consequences (Killen et al., 1994a; Stice et al., 1998; Taylor et al., 1998; Mitchell et al., 2002).

Risk factors for EDs

In recent years, considerable progress has been made in identifying risk factors for EDs (Jacobi et al., 2004b; Striegel-Moore & Bulik, 2007). Because of the inconsistent use of the terms risk and risk factor, Kraemer et al., (1997) proposed exact definitions and methods for identifying risk and etiology factors. In this approach, precedence is a crucial criterion for the definition of risk factors. Accordingly, the most informative risk factor studies are longitudinal studies. This model has been used to confirm potential risk factors for AN, BN, BED, and syndromes including EDs not otherwise specified (EDNOS) (Jacobi et al., 2004b; Jacobi, 2005, 2007). Although the low incidence of AN has limited the usefulness of prospective studies to identifying risk factors for that disorder, several risk factors have been confirmed for BN and EDNOS. Of these, gender and weight/shape concerns are consistently the most replicated and most potent factors for identifying students at risk of developing an ED (Taylor et al., 2003; Jacobi et al., 2004b). Unfortunately, the majority of samples in previous
longitudinal studies were too small for consistent and meaningful risk factor detection of clinical disorders. The selection of subjects already at higher risk at the beginning of the study may therefore yield more promising results. No previous risk factor studies have examined risk factors within high-risk populations.

Based on these data, Taylor et al. (2006) used high weight and shape concerns to identify female college-age students at potential risk of EDs and to determine whether a brief psychosocial intervention could reduce risk. As part of the study design, most of the risk factors identified by Jacobi et al. (2004b) were included in the baseline analysis, with the assumption that these factors might identify subgroups of high-risk students who were most likely to develop EDs. Identifying such students is of both theoretical and practical importance. Theoretically, some factors may identify subgroups of high-risk students at highest risk and potentially different etiologies. Practically, these high-risk groups might benefit from targeted interventions, thus conserving preventive interventions for those most at risk.

Another limitation of previous risk factor studies is the lack of consideration of interactions among risk factors, information useful for improving the understanding of the etiology of the disorder and the development and effectiveness of preventive interventions. To address the different interactions among risk factors (i.e. overlapping factors, proxies, mediators, and moderators), additional definitions and methodological recommendations were proposed (Kraemer et al. 2001). In the context of EDs, this extended methodological approach has been applied in only two studies (Taylor et al. 2003; Agras et al. 2007). Accordingly, the aims of this study were (1) to identify risk factors and their interactions for ED onset in a high-risk sample of college-age women using the methods developed by Kraemer et al. (1997, 2001) and (2) to determine the most potent risk factors for ED onset (including sensitivity, specificity, and optimal cut-offs) in a high-risk sample.

Method

Design

Potential risk factors for the onset of EDs and interactions between these factors were assessed longitudinally over 3 years with assessments at years 1, 2 and 3. For the assessment of potential risk factors, the non-treatment study arm of a randomized controlled prevention trial for EDs was used (Taylor et al. 2006).

Participants

Participants were 236 college-age women from San Diego and San Francisco aged 18 to 30 years (mean = 20.8, s.d. = 2.6) originally recruited for participation in a randomized controlled prevention trial for EDs (for details see Taylor et al. 2006). Overall, 21 participants (8.9%) had no follow-up data and were not available for the survival analysis.

Mean body mass index (BMI) of participants was 23.7 (s.d. = 2.7). Ethnicity of the sample was 61.0% white, 21.1% African American, 8.5% Hispanic, 16.5% Asian, and 11.9% other. By year in school, the sample consisted of 33.8% freshman, 20.2% sophomore, 20.2% junior, 17.8% senior, and 8.0% graduate students.

The Weight Concerns Scale (WCS) was used to determine high-risk status. The WCS consists of five questions that assess worry about weight and shape, fear of gaining 3 pounds, last time on a diet, importance of weight, and feelings of fatness. The WCS has good test–retest reliability (r = 0.85); a score of ≥ 47 has good predictive validity for ED caseness (Killen et al. 1994b, 1996; Jacobi et al. 2004a). Participants were considered potentially eligible for this study if they scored ≥ 50 on the WCS, reported that they were moderately or very afraid of gaining 3 pounds, or reported that their weight was the most important thing in their life.

Women who met clinical criteria for a DSM-IV-diagnosed ED at baseline were excluded from the study. Additional exclusion criteria were a current subthreshold ED diagnosis obtained from the Eating Disorder Examination (EDE) interview or treatment for ED within the past 6 months, acute suicidal ideation and/or drug or alcohol abuse or dependence (see Taylor et al. 2006 for a more detailed description). At baseline, 49 (21%) participants endorsed sporadic binge eating or compensatory behaviors (vomiting, laxative use, diuretic use) in the previous 3 months, but not at a frequency that met diagnostic criteria for clinical or subthreshold EDs. Of these, 31 (13%) reported objective binge episodes (median = 4), 18 (8%) engaged in some kind of compensatory behavior (median = 1.5) and seven (3%) in both over the past 3 months.

The study was approved by the human subjects committees at each of the participating institutions, including Stanford University and San Diego State University.

Measures

Most of the potential risk factors were assessed at baseline and at 1-, 2- and 3-year follow-up. The following potential risk factors were assessed at baseline only: participant’s self-reported age, year in school, ethnicity, and mother’s and father’s highest level of education, maximum parental and maximum own
weight, negative comments about weight, shape and eating, parental psychopathology, childhood trauma, and previous own psychopathology. Maximum parental weight and maximum own weight were assessed using Stunkard’s figures (Stunkard et al. 1983).

Negative comments on weight, shape and eating and parental psychopathology were assessed using items from a risk factor interview developed by Fairburn et al. (1998). The items related to negative comments were: ‘Before you were 18, did anyone ever make negative comments about your shape or weight?’ and a similar item was used for eating. The participants rated this for all relatives, friends, peers, coaches, or teachers as ‘never,’ ‘a few comments’ or ‘repeated comments’. Parental (or primary caregiver) history of depression, alcohol use, and EDs were also assessed using the respective items from the risk factor interview (Fairburn et al. 1998).

Possible childhood maltreatment was assessed by the Childhood Trauma Questionnaire (CTQ; Bernstein & Fink, 1998; Scher et al. 2001).

Participants’ previous psychopathology was assessed using Section C of the SCID, which screens for the major DSM-IV diagnoses. In this model, any ‘yes’ answers are followed up with completion of the relevant module. However, the SCID current and past depression and anxiety modules were completed on everyone.

Case definition

The ED diagnoses and assessment of ED behaviors were made with the EDE interview adapted to include the diagnostic criteria for BED. The EDE (Cooper & Fairburn, 1987) is a semi-structured interview that generates ED diagnoses based on DSM-IV criteria. It has demonstrated high internal consistency, sensitivity to change, and inter-rater reliability (Rosen et al. 1990; Luce & Crowther, 1999). Diagnoses of AN, BN and BED corresponded with the DSM-IV and were consistent with previous studies (Taylor et al. 2003).

ED attitudes and behaviors were assessed using the WCS (Killen et al. 1994b, 1996), the Eating Disorder Inventory (EDI) drive for thinness and bulimia subcales (Garner & Olmsted, 1984), and the EDE Questionnaire (EDE-Q), a self-report version of the EDE (Luce & Crowther, 1999). Apart from these, the following other potential risk factors were assessed at all assessment points.

Social support was measured with the Multidimensional Scale of Perceived Social Support (Zimet et al. 1990), a 12-item self-report measure of perceived social support (Clara et al. 2003). The Center for Epidemiological Studies – Depression Scale (CES-D), a 20-item self-report questionnaire, was used to assess depressed mood (Orme et al. 1986). The CES-D has high internal consistency, adequate test–retest reliability, and convergent validity (Plutchik & van Praag, 1987). Coping strategies that participants typically use when facing stressful events were assessed by the 28-item measure Brief COPE (Carver, 1997).

Global self-esteem was assessed by the Rosenberg Self-Esteem Scale (RSE; Rosenberg, 1965). Current social (mal-)adjustment was assessed using the Social Adjustment Scale Self-Report (SAS-SR), modified for college participants (Weissman & Bothwell, 1976). The SAS-SR has good reliability and convergent validity with clinician ratings. Negative life events were assessed by asking students to note if any of 24 events (such as having a serious illness, or parental divorce) occurred in the past year and, if so, to rate their impact on their life as: none, some, moderate, great (Johnson & McCutcheon, 1980). Alcohol use was assessed by asking how many times in the past month the participant had four or more drinks on one occasion and how many drinks they usually have in a week (Wechsler et al. 2000).

Statistical analysis

The model for the identification of potential risk factors follows the methodological and statistical recommendations by Kraemer et al. (2001, 2005). In this model, potential risk factors are first ordered temporally according to the time period of their assessment. For the present study, the following time periods were determined: (1) pre-baseline (birth to early adulthood assessed retrospectively before onset of ED), and (2) baseline (with factors assessed prospectively). Because changes between baseline and the follow-ups have only theoretical value and little value for screening purposes, these factors were omitted from the analyses.

The analysis was carried out in three separate steps:

Step 1: The relationship between each of the potential risk factors and the outcome was assessed univariately according to Cox regression models. The significance level for these analyses was set at p < 0.05.

Step 2: Within each time period, the risk factors remaining from step 1 were examined pairwise in relation to the outcome using Cox regression models. Factors were examined and identified as independent, proxy or overlapping risk factors according to the definitions by Kraemer et al. (2001, 2005). Two factors (A and B) were considered as independent if they were uncorrelated (r < 0.2). Correlated factors were considered as proxies (B) if only A remained a predictor of the outcome, and as overlapping if both A and B predicted outcome in the bivariate model. Proxies were removed from further analyses, overlapping
factors were combined (e.g. into one factor using principal component analysis).

**Step 3:** Following the identification of independent and overlapping risk factors within time, independent risk factors, mediators and moderators were identified across time periods according to the procedure outlined above. All variables were centered according to the recommendations of Kraemer & Blasey (2004). The significance level for testing the moderator interaction was set at $p=0.01$ (Kraemer et al. 2005).

Potency of confirmed risk factors was first determined by odds ratios for binary variables and by Cohen’s $\delta$ for continuous variables. To enable comparisons of effect sizes for binary and continuous variables, the area under the curve (AUC) was also calculated (Kraemer et al. 2003). The standards used to categorize the AUC are: $<56\%$ very low, $56\% \leq \text{AUC} <63\%$ low, $64\% \leq \text{AUC} <70\%$ medium, and $\text{AUC} \geq 70\%$ large (Kraemer et al. 2003).

Finally, all confirmed risk factors were entered into a receiver operator characteristics (ROC) analysis to determine optimal cut-offs and also sensitivity and specificity of the most potent risk factors (Kraemer et al. 1999) (www.stanford.edu/~yesavage/ROC.html).

**Results**

**Onset of EDs**

Over the course of the study, 24 out of 215 participants (11.2%) were classified as subthreshold or full clinical ED cases. Of the participants with full EDs, one (0.5%) fulfilled criteria for BN, 11 (5.1%) for subthreshold BN, and seven (3.3%) for subthreshold BED. Five (2.3%) participants were classified as cases for EDs on the basis of reporting entering treatment for an ED. One of them with a BMI of 17.6 entered treatment and might have been anorectic. Seven of the 24 participants classified as cases endorsed sporadic binge eating and/or compensatory behaviors below the threshold of full or subthreshold cases already at baseline (four participants binging only, two participants purging only, and one participant both).

**Comparison between drop-outs and completers**

Differences between participants who completed all follow-up assessments and those who did not were tested by $t$ tests. Participants who completed all follow-up assessments did not differ from those who dropped out before the completion of all follow-up assessments on any of the sociodemographic variables, eating-related variables, or general psychopathology.

**Step 1: Univariate analyses**

Before baseline, the following variables were related significantly and positively to ED onset (Table 1): comments about eating by coach or teacher, comments about eating by friends, comments about eating by siblings, comments about weight and shape by coach or teacher, comments about weight and shape by siblings, previous diagnoses of depression, and previous panic disorder diagnoses. Lower parental weight predicted ED onset. At baseline, the following variables were related significantly and positively to ED onset: EDE-Q Eating Concern, EDE-Q Weight and Shape Concerns, weight concerns, EDI Drive for Thinness, EDI Bulimia, compensatory behavior, and number of alcoholic drinks per week.

Table 1 also displays potential risk factors that were not significant in the univariate analyses and were then omitted from further analyses.

**Step 2: Within-time analyses**

In the pairwise comparisons, the following overlapping risk factors and proxies were identified (Fig. 1): before baseline, both comments about eating by friends and comments about weight and shape by coach or teacher turned out to be proxies for comments about eating by coach or teacher. Because comments about weight and shape by siblings and comments about eating by siblings turned out to be overlapping factors, they were combined into one factor that was independent of the factor comments about eating by coach or teacher. Furthermore, a history of panic disorder was a proxy for a history of depression diagnosis.

At baseline, EDE-Q Weight Concern, Weight and Shape Concerns, EDI Drive for Thinness and EDI Bulimia turned out to be proxies for EDE-Q Eating Concern, whereas EDE-Q Eating Concern and compensatory behaviors turned out to be overlapping factors ($r=0.20$). Because they cover different aspects of eating problems (i.e. attitudes and behaviors), they were retained as separate factors.

**Step 3: Across-time analyses**

Risk factors were examined across time periods to determine mediators, moderators, and proxies (Fig. 2). No proxies were found across time periods. None of the factors could be confirmed as moderator or mediator according to our preset criteria regarding correlations between risk factors ($r>0.20$) and the required significance levels for testing the interactions.
Table 1. Univariate relationships between potential risk factors and outcome

<table>
<thead>
<tr>
<th>Measure</th>
<th>Before baseline</th>
<th>Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>W</td>
<td>p</td>
</tr>
<tr>
<td>Season of birth</td>
<td>2.23</td>
<td>0.131</td>
</tr>
<tr>
<td>Years in school</td>
<td>2.15</td>
<td>0.143</td>
</tr>
<tr>
<td>Parental education</td>
<td>0.94</td>
<td>0.333</td>
</tr>
<tr>
<td>Ethnicity (minority status)</td>
<td>2.85</td>
<td>0.091</td>
</tr>
<tr>
<td>Negative comments about weight and shape from parents</td>
<td>0.51</td>
<td>0.474</td>
</tr>
<tr>
<td><strong>Negative comments about weight and shape from siblings</strong></td>
<td>3.62</td>
<td>0.057</td>
</tr>
<tr>
<td>Negative comments about weight and shape from peers or friends</td>
<td>2.81</td>
<td>0.094</td>
</tr>
<tr>
<td>Negative comments about weight and shape from coach or teacher</td>
<td>3.71</td>
<td>0.054</td>
</tr>
<tr>
<td>Negative comments about eating from parents</td>
<td>0.94</td>
<td>0.332</td>
</tr>
<tr>
<td>Negative comments about eating from siblings</td>
<td>7.39</td>
<td>0.007</td>
</tr>
<tr>
<td>Negative comments about eating from peers or friends</td>
<td>3.74</td>
<td>0.053</td>
</tr>
<tr>
<td>Negative comments about eating from coach or teacher</td>
<td>15.10</td>
<td>0.000</td>
</tr>
<tr>
<td>Family eating disorder</td>
<td>2.58</td>
<td>0.109</td>
</tr>
<tr>
<td>Parental depression</td>
<td>1.41</td>
<td>0.260</td>
</tr>
<tr>
<td>Parental alcohol problems</td>
<td>1.27</td>
<td>0.260</td>
</tr>
<tr>
<td>Dieting in the family</td>
<td>1.26</td>
<td>0.262</td>
</tr>
<tr>
<td>Encourage for dieting in the family</td>
<td>0.28</td>
<td>0.599</td>
</tr>
<tr>
<td>Maximum weight student (Stunkard)</td>
<td>0.24</td>
<td>0.627</td>
</tr>
<tr>
<td><strong>Average parental weight (Stunkard)</strong></td>
<td>6.45</td>
<td>0.025</td>
</tr>
<tr>
<td>CTQ Emotional Abuse</td>
<td>0.32</td>
<td>0.571</td>
</tr>
<tr>
<td>CTQ Sexual Abuse</td>
<td>1.10</td>
<td>0.294</td>
</tr>
<tr>
<td>CTQ Emotional Neglect</td>
<td>0.64</td>
<td>0.426</td>
</tr>
<tr>
<td><strong>Lifetime depression diagnosis</strong></td>
<td>13.46</td>
<td>0.000</td>
</tr>
<tr>
<td>Lifetime bipolar disorder*</td>
<td>4.84</td>
<td>0.028</td>
</tr>
<tr>
<td>Lifetime panic disorder diagnosis*</td>
<td>4.84</td>
<td>0.028</td>
</tr>
<tr>
<td>Lifetime agoraphobia*</td>
<td>4.84</td>
<td>0.028</td>
</tr>
<tr>
<td>Lifetime social phobia*</td>
<td>4.84</td>
<td>0.028</td>
</tr>
<tr>
<td>Lifetime obsessive compulsive disorder*</td>
<td>4.84</td>
<td>0.028</td>
</tr>
<tr>
<td>Lifetime any anxiety disorder*</td>
<td>4.84</td>
<td>0.028</td>
</tr>
</tbody>
</table>

W, Wald statistic; AUC, area under the curve; WCS, Weight Concerns Scale; EDE-Q, Eating Disorder Examination Questionnaire; EDI, Eating Disorder Inventory; BMI, body mass index; CTQ, Childhood Trauma Questionnaire (CTQ); CES-D, Center for Epidemiological Studies – Depression Scale.

Bold indicates significant univariate relationships.

* Cox regressions only performed for n > 5 cases.
When interactions between risk factors were examined across time periods, factors identified in the within-time analyses were confirmed as independent risk factors (Fig. 2).

### Potency and cut-off determination

Effect sizes (AUCs) for previous depression diagnosis, negative comments by coach or teacher, negative comments by siblings, EDE-Q Eating Concern, compensatory behavior, and changes in negative life events can be classified as in the medium range, whereas effect sizes for the remaining risk factors were small or very small (previous panic disorder diagnosis) (Kraemer et al. 2001).

In the ROC analysis (Fig. 3), the best predictor was negative comments by coach or teacher, with a prevalence of 39.1% in those participants endorsing comments versus 7.8% in those not endorsing comments ($\chi^2 = 17.73, p \leq 0.000$). The optimal cut-off was ‘1’, equivalent to a few or repeated comments versus none. In participants not reporting negative comments, the best predictor was depression diagnoses, with a prevalence of 30.4% in participants with a positive diagnosis of depression ever versus 4.2% in participants without depression diagnosis ever ($\chi^2 = 19.1, p \leq 0.000$).

The prevalence of EDs in participants endorsing either negative comments or a depression diagnosis was 34.8% compared to participants not endorsing any of these predictors ($\chi^2 = 31.9, p \leq 0.000$). The sensitivity of either negative comments or depression diagnosis was 0.75, specificity 0.82 [positive predictive value (PPV) = 0.35].

### Discussion

This is the first prospective study examining risk factors for EDs and their interactions across time periods in a high-risk sample based on the methodology proposed by Kraemer et al. (2001). The study is of theoretical importance as it adds insight into the nature of EDs and of practical importance as two items proved to have high sensitivity and specificity, allowing for preventive resources to be used more efficiently.
In this high-risk group of college-age women (i.e. high weight and shape concerns), we found an 11% onset rate of full or subthreshold EDs, which is consistent with rates of 10% and 12% found in risk factor studies of adolescents (Killen et al. 1994a, 1996). Comparable to what has been found in the majority of longitudinal studies, most of the cases in our study were subthreshold (Jacobi et al. 2004; Jacobi & Fittig, 2010). No full cases of AN and only one full case of BN were found. Of the large number of potential risk factors included, only a few turned out to be predictive of ED onset when interactions between factors were examined both within and across time periods. Sixteen of the 88 potential risk factors originally included were confirmed as risk factors, seven of these turned out to be proxies, two were overlapping factors, and seven were independent risk factors.

Of the seven independent risk factors, a history of depression was one of the two factors with the highest potency (AUC = 67.21) for predicting ED onset. Although a history of depression has not as yet been examined as a risk factor prospectively, negative emotionality and neuroticism, both of which are probably proxies for depression, have been confirmed as predictors of eating disturbances and disorders in most of the longitudinal studies of ED onset (Attie & Brooksgunn, 1989; Graber et al. 1994; Leon et al. 1995, 1999; Killen et al. 1996; Moorhead et al. 2003; Taylor et al. 2003; Bulik et al. 2006). Further support for this factor comes from cross-sectional case–control studies with retrospective assessment of depression diagnosis, which found up to sevenfold higher rates of pre-morbid depression compared to healthy controls (Fairburn et al. 1997, 1998, 1999; Pike et al. 2007).

A history of negative comments from a coach or teacher about eating and a history of negative comments about eating, weight and shape by siblings were two other risk factors predicting ED onset. Although longitudinal evidence for these factors is fairly weak, there is evidence from cross-sectional studies that a history of critical comments about shape, weight and eating by the family was significantly more prevalent (two- to sixfold risk) in patients with EDs (AN, BN and BED) compared to healthy controls (Fairburn et al. 1997, 1998, 1999). Similarly, a study of Australian twins found that retrospectively assessed parental comments about weight were associated with onset of both objective binge eating and self-induced vomiting (Wade et al. 2008). In a longitudinal study by Neumark-Sztainer et al. (2007), weight teasing by family was one of the strongest predictors of various outcomes including binge eating and being overweight in a large group of adolescent girls at the 5-year follow-up. A history of a lower parental average weight was the final risk factor assessed pre-baseline found to predict ED onset. This finding somewhat contradicts an earlier finding where bulimic patients, when compared to healthy controls, reported higher rates of parental obesity before the onset of their ED (Fairburn et al. 1997). On one hand, it seems plausible that a parent with a lower average weight increases pressures to be thin and thus promotes dieting, weight and shape concerns, and subsequent EDs in the child. On the other hand, parental weight assessed by the Stunkard figures in our study was still in the normal range in both groups and the potency of this factor was fairly small.

At baseline, the EDE-Q Eating Concern scale, level of compensatory behaviors, and number of alcohol drinks in a week predicted ED onset. The factor ‘weight and shape concerns’ is the most potent and consistently supported risk factor for ED onset on the basis of longitudinal research (Jacobi et al. 2004b). Because it overlaps or correlates highly with EDE-Q Eating Concern, it is not surprising that it predicts onset even in the high-risk sample. In the randomized factors, two were overlapping factors, and seven were independent risk factors.

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Of the seven independent risk factors, a history of depression was one of the two factors with the highest potency (AUC = 67.21) for predicting ED onset. Although a history of depression has not as yet been examined as a risk factor prospectively, negative emotionality and neuroticism, both of which are probably proxies for depression, have been confirmed as predictors of eating disturbances and disorders in most of the longitudinal studies of ED onset (Attie & Brooksgunn, 1989; Graber et al. 1994; Leon et al. 1995, 1999; Killen et al. 1996; Moorhead et al. 2003; Taylor et al. 2003; Bulik et al. 2006). Further support for this factor comes from cross-sectional case–control studies with retrospective assessment of depression diagnosis, which found up to sevenfold higher rates of pre-morbid depression compared to healthy controls (Fairburn et al. 1997, 1998, 1999; Pike et al. 2007).

A history of negative comments from a coach or teacher about eating and a history of negative comments about eating, weight and shape by siblings were two other risk factors predicting ED onset. Although longitudinal evidence for these factors is fairly weak, there is evidence from cross-sectional studies that a history of critical comments about shape, weight and eating by the family was significantly more prevalent (two- to sixfold risk) in patients with EDs (AN, BN and BED) compared to healthy controls (Fairburn et al. 1997, 1998, 1999). Similarly, a study of Australian twins found that retrospectively assessed parental comments about weight were associated with onset of both objective binge eating and self-induced vomiting (Wade et al. 2008). In a longitudinal study by Neumark-Sztainer et al. (2007), weight teasing by family was one of the strongest predictors of various outcomes including binge eating and being overweight in a large group of adolescent girls at the 5-year follow-up. A history of a lower parental average weight was the final risk factor assessed pre-baseline found to predict ED onset. This finding somewhat contradicts an earlier finding where bulimic patients, when compared to healthy controls, reported higher rates of parental obesity before the onset of their ED (Fairburn et al. 1997). On one hand, it seems plausible that a parent with a lower average weight increases pressures to be thin and thus promotes dieting, weight and shape concerns, and subsequent EDs in the child. On the other hand, parental weight assessed by the Stunkard figures in our study was still in the normal range in both groups and the potency of this factor was fairly small.

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trial, participants with high levels of compensatory behaviors at one site had onset of EDs at the 2-year follow-up of 30.4% (Taylor et al. 2006). Alcohol abuse has been found to predict ED onset (Killen et al. 1996; Field et al. 2002; Wonderlich et al. 2004). Other studies also found ED symptoms to be predictive of alcohol use (Strober et al. 1996; Measelle et al. 2006). One hypothesis is that a subset of women with EDs who use substances and binge eating to cope with distress (Safer et al. 2001) are more likely than non-bulimic or bingeing women to have difficulties with affect regulation. If so, it would be expected that higher alcohol use, as an indication of dysfunctional coping, might predict ED onset.

Most of the potential risk factors measured in our study were based on those confirmed in the meta-analysis (Jacobi et al. 2004b). Only a few factors were not measured: acculturation, pubertal timing, some personality factors, and neuroticism. The present study differs from most of the studies included in the meta-analysis with regard to risk status, age and sample size. Although sample sizes were usually larger among the studies in the meta-analysis, high-risk samples were not assessed, and mainly adolescents were studied. However, with the two exceptions of lower parental weight and compensatory behavior, risk factors found in this high-risk college-age sample are in accordance with factors from the meta-analysis.

On the basis of the most potent risk factors, the two questions identified in the ROC analysis could serve as a useful two-step screen. The first step would be to use the WCS to identify college-age students with high weight and shape concerns. The risk of developing an ED in this sample would be about 10%. The next step would be to select students with high weight concerns who endorsed either a history of negative comments about eating or a history of depression. Based on the ROC analysis, the final model using these two questions as a screen would have a reasonably high sensitivity (0.75) and specificity (0.82).

Data from this study, combined with that from existing literature, allow us to estimate the at-risk population in a college-age population. About 25% of college-age women have weight and shape concerns, placing them at some risk (10%) of developing an ED (Drenowski et al. 1988; Killen et al. 1996). Of these, assuming the current sample represents a typical population, about a third would be at high risk. Within this very high-risk group, about a third would develop an ED. Accordingly, in a sample of 100 college-age women, about 25 could be classified as high risk. Of these 25, eight or nine would be at very high risk, and of these, two or three would develop an ED for an incidence rate of 2–3%.

There are several limitations to this study. Cases were limited primarily to (subthreshold) BN and BED and thus risk factors may not be equally relevant for AN. Although students did not have an ED in the first 6 months before the trial, a few might have had a lifetime history before that time frame. However, the prevalence of a past history of ED is too small for us to determine whether the intervention might have had an effect on preventing relapse.

Some of the variables could only be obtained retrospectively. In addition, we cannot fully rule out that the risk status of included participants, even though they do not fulfill criteria for an ED at baseline, may have affected their recall of some childhood or current experiences and feelings. The sample involved individuals who were interested in an intervention to reduce weight and shape concerns and to improve body image. The specificity of the risk factors for EDs was not tested in this study. However, some of the factors (e.g. negative affect/depression) represent confirmed risk factors for other disorders (Hayward et al. 2000; Hirshfeld-Becker et al. 2008).

In recent years, there has been some debate about the validity of the frequency criterion for BN. Some authors have suggested relaxing the frequency criterion by adopting a once a week or even lower (≥2 times/month) threshold in DSM-V (e.g. Spoor et al. 2007; Wilfley et al. 2007; Wilson & Sysko, 2009). Although evidence-based cut-offs are still to be determined by future research (Wilson & Sysko, 2009), preventive interventions may need to attempt to reduce any binge eating and compensatory behaviors even if below subthreshold disorders.

Although the application of a systematic risk factor approach (Kraemer et al. 1997, 2001) has proved effective in separating correlates from prospective factors and in addressing interactions among risk factors, there are also limitations to this approach. Proxy factors or risk factors that are correlated may be important but are excluded if they are less strongly associated with the outcome. However, it would not make sense, as implied by the statistical model, to recommend teachers and coaches to focus on not making critical comments only about eating. Similarly, it is not clear why a history of depression would be more important than current depression or depressed mood; the latter would need to be addressed in an intervention. In similar populations, slight changes in distribution of depression scores or prevalence of current or past depression could make any of these variables proxies to others. An ROC analysis, although indicating which groups might benefit from intervention, suffers from the same issue. Depending on slight changes in distribution, other factors might have emerged as important.
Overall, the results of this study identify a group of students within an already high-risk sample who are very likely to develop an ED. Preventive interventions addressing weight and shape concerns should be expanded to focus on issues of affect and affect regulation and on the effects of negative comments about eating and shape.

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Declaration of Interest

None.

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