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| Authors C. Jacobi, E. Fittig, S. W. Bryson, D. Wilfley, H. C. Kraemer, and C. Barr Taylor | | | | | | | |
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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.* 1994*a*; Stice *et al.* 1998; Taylor *et al.* 1998; Mitchell *et al.* 2002).

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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

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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 collegeage 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 highrisk 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 collegeage 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 long-itudinally 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, 2.1% 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 \geq 47 has good predictive validity for ED caseness (Killen et~al.~1994~b, 1996; Jacobi et~al.~2004~a). Participants were considered potentially eligible for this study if they scored \geq 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-IVdiagnosed 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

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.* 1994*b*, 1996), the Eating Disorder Inventory (EDI) drive for thinness and bulimia subscales (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 by 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 δ 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 AUC <63% low, 64% \leq AUC <70% medium, and 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*

| Measure | Before baseline | | | | Baseline | | |
|--|-----------------|-------|---------|-----------------------------------|----------------|-------|---------|
| | W | р | AUC (%) | Measure | \overline{W} | р | AUC (%) |
| Season of birth | 2.23 | 0.131 | | WCS | 5.75 | 0.017 | 64.22 |
| Years in school | 2.15 | 0.143 | | EDE-Q Eating Concern scale | 12.81 | 0.000 | 65.91 |
| Parental education | 0.94 | 0.333 | | EDE-Q Shape Concern scale | 1.27 | 0.260 | |
| Ethnicity (minority status) | 2.85 | 0.091 | | EDE-Q Weight Concern scale | 4.12 | 0.042 | 61.58 |
| Negative comments about weight and shape from parents | 0.51 | 0.474 | | EDE-Q Restraint | 0.86 | 0.354 | |
| Negative comments about weight and shape from siblings | 3.62 | 0.057 | 60.59 | EDI Interoceptive Awareness | 2.66 | 0.103 | |
| Negative comments about weight and shape from peers or friends | 2.81 | 0.094 | | EDI Drive for Thinness | 6.00 | 0.014 | 65.85 |
| Negative comments about weight and shape from coach or teacher | 3.71 | 0.054 | 60.03 | EDI Bulimia | 4.58 | 0.032 | 55.99 |
| Negative comments about eating from parents | 0.94 | 0.332 | | EDI Perfectionism | 1.84 | 0.175 | |
| Negative comments about eating from siblings | 7.39 | 0.007 | 60.58 | Emotional eating | 0.91 | 0.339 | |
| Negative comments about eating from peers or friends | 3.74 | 0.053 | 60.81 | BMI | 0.29 | 0.591 | |
| Negative comments about eating from coach or teacher | 15.10 | 0.000 | 69.00 | Compensatory behavior | 7.61 | 0.006 | 66.34 |
| Family eating disorder | 2.58 | 0.109 | | Rosenberg self-esteem | 0.00 | 0.949 | |
| Parental depression | 1.41 | 0.260 | | Social support family | 0.09 | 0.770 | |
| Parental alcohol problems | 1.27 | 0.260 | | Social support friends | 0.01 | 0.919 | |
| Dieting in the family | 1.26 | 0.262 | | Social support significant others | 0.01 | 0.908 | |
| Encourage for dieting in the family | 0.28 | 0.599 | | COPE Denial | 0.57 | 0.452 | |
| Maximum weight student (Stunkard) | 0.24 | 0.627 | | COPE Substance Use | 2.40 | 0.121 | |
| Average parental weight (Stunkard) | 6.45 | 0.025 | 63.02 | COPE Emotional Support | 1.13 | 0.288 | |
| CTQ Emotional Abuse | 0.32 | 0.571 | | COPE Positive Reframing | 0.02 | 0.896 | |
| CTQ Sexual Abuse | 1.10 | 0.294 | | COPE Planning | 0.79 | 0.375 | |
| CTQ Emotional Neglect | 0.64 | 0.426 | | COPE Use of Humor | 2.27 | 0.132 | |
| Lifetime depression diagnosis | 13.46 | 0.000 | 67.21 | COPE religion | 0.01 | 0.909 | |
| Lifetime bipolar disorder ^a | | | | Life events occurrence | 2.19 | 0.139 | |
| Lifetime panic disorder diagnosis | 4.84 | 0.028 | 54.67 | Life events effect | 0.01 | 0.908 | |
| Lifetime agoraphobia ^a | | | | CES-D | 0.51 | 0.475 | |
| Lifetime social phobia ^a | | | | Alcohol use in the last month | 0.89 | 0.346 | |
| Lifetime obsessive compulsive disorder ^a | | | | Alcohol use in a week (usually) | 4.96 | 0.029 | 63.02 |
| Lifetime any anxiety disorder ^a | | | | Social impairment | 1.23 | 0.267 | |

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.

^a Cox regressions only performed for n > 5 cases.

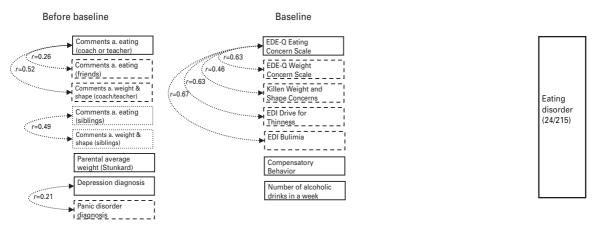


Fig. 1. Within-time analyses. Boxes with dashed lines indicate proxies, dotted lines indicate overlapping risk factors.

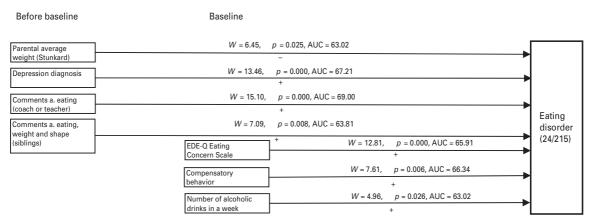


Fig. 2. Across-time analyses.

(p < 0.01). 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.* 2003).

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 ($\chi^2 = 17.73$, $p \le 0.000$). 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 $(\chi^2 = 19.1, p \le 0.000)$.

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 (4.2%) (χ^2 =31.9, $p \le 0.000$). 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.

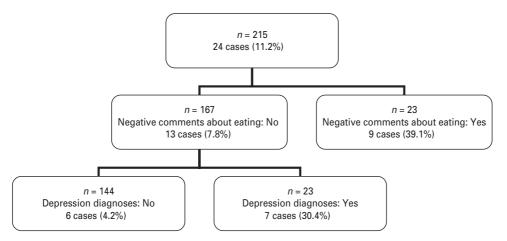


Fig. 3. Receiver operating characteristic (ROC) analysis.

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. 2004a; 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.* 2004*b*). 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

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.* 2004*b*). 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 very 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 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.

References

- Agras WS, Bryson S, Hammer LD, Kraemer HC (2007). Childhood risk factors for thin body preoccupation and social pressure to be thin. *Journal of the American Academy of Child and Adolescent Psychiatry* **46**, 171–178.
- Attie I, Brooksgunn J (1989). Development of eating problems in adolescent girls: a longitudinal study. *Developmental Psychology* **25**, 70–79.
- Bernstein D, Fink L (1998). Manual for the Childhood Trauma Questionnaire. The Psychological Corporation: New York.
- Bulik CM, Sullivan PF, Tozzi F, Furberg H, Lichtenstein P, Pedersen NL (2006). Prevalence, heritability, and prospective risk factors for anorexia nervosa. *Archives of General Psychiatry* **63**, 305–312.
- Carver CS (1997). You want to measure coping but your protocol's too long: consider the Brief COPE. *International Journal of Behavioral Medicine* **4**, 92–100.
- Clara IP, Cox BJ, Enns MW, Murray LT, Torgrudc LJ (2003).
 Confirmatory factor analysis of the Multidimensional Scale of Perceived Social Support in clinically distressed and student samples. *Journal of Personality Assessment* 81, 265–270
- Cooper Z, Fairburn CG (1987). The Eating Disorder Examination: a semi-structured interview for the assessment of the specific psychopathology of eating disorders. *International Journal of Eating Disorders* **6**, 1–8.
- Drenowski A, Hopkins SA, Kessler RC (1988). The prevalence of bulimia nervosa in the U.S. college student population. American Journal of Public Health 78, 1322–1325.
- Fairburn CG, Cooper Z, Doll HA, Welch SL (1999). Risk factors for anorexia nervosa: three integrated case-control comparisons. Archives of General Psychiatry 56, 468–476.
- Fairburn CG, Doll HA, Welch SL, Hay PJ, Davies BA, O'Connor ME (1998). Risk factors for binge eating

- disorder: a community-based, case-control study. *Archives of General Psychiatry* **55**, 425–432.
- Fairburn CG, Welch SL, Doll HA, Davies BA, O'Connor ME (1997). Risk factors for bulimia nervosa: a community-based case-control study. Archives of General Psychiatry 54 509–517.
- Field AE, Austin SB, Frazier AL, Gillman MW, Camargo CA, Colditz GA (2002). Smoking, getting drunk, and engaging in bulimic behaviors: in which order are the behaviors adopted? *Journal of the American Academy of Child and Adolescent Psychiatry* 41, 846–853.
- Fitzgibbon ML, Sanchez-Johnsen LAP, Martinovich Z (2003). A test of the continuity perspective across bulimic and binge eating pathology. *International Journal of Eating Disorders* **34**, 83–97.
- Garner DM, Olmsted MP (1984). Eating Disorder Inventory Manual. Psychological Assessment Resources, Inc.: Odessa, FL.
- Graber JA, Brooksgunn J, Paikoff RL, Warren MP (1994).Prediction of eating problems: an 8-year study of adolescent girls. *Developmental Psychology* 30, 823–834.
- Hayward C, Killen JD, Kraemer HC, Taylor CB (2000).
 Predictors of panic attacks in adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry* 39, 207–214.
- Hirshfeld-Becker DR, Micco JA, Simoes NA, Henin A (2008). High risk studies and developmental antecedents of anxiety disorders. *American Journal of Medical Genetics*. *Part C, Seminars in Medical Genetics* **148C**, 99–117.
- **Hoek HW, van Hoeken D** (2003). Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders* **34**, 383–396.
- Hudson JI, Hiripi E, Pope Jr. HG, Kessler RC (2007).
 The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry* 61, 348–358.
- Jacobi C (2005). Psychosocial risk factors for eating disorders. In *Eating Disorders Review* (ed. S. A. Wonderlich, J. E. Mitchell, M. de Zwaan and H. Steiger), pp. 59–85. Radcliffe Publishing Ltd: Oxford, UK.
- Jacobi C (2007). Comparing risk for onset and chronic course of eating disorders: what do we know about the individuals who have enduring eating disorder pathology? In 8th London International Eating Disorders Conference, London, UK.
- Jacobi C, Abascal L, Taylor CB (2004a). Screening for eating disorders and high-risk behavior: caution. *International Journal of Eating Disorders* 36, 280–295.
- Jacobi C, Fittig E (2010). Psychosocial risk factors for eating disorders. In Oxford Handbook of Eating Disorders (ed. W. S. Agras), pp. 123–135. Oxford University Press: New York.
- Jacobi C, Hayward C, de Zwaan M, Kraemer HC, Agras WS (2004b). Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. *Psychological Bulletin* 130, 19–65.
- **Johnson JG, McCutcheon S** (1980). Assessing life stress in older children and adolescents: preliminary findings with the Life Events Checklist. In *Stress and Anxiety*

- (ed. I. Sarason and C. Spielberger), pp. 115–125. Hemisphere Publishing Corporation: Washington, DC.
- Killen JD, Hayward C, Wilson DM, Taylor CB, Hammer LD, Litt I, Simmonds B, Haydel F (1994a).
 Factors associated with eating disorder symptoms in a community sample of 6th and 7th grade girls. *International Journal of Eating Disorders* 15, 357–367.
- Killen JD, Taylor CB, Hayward C, Haydel KF, Wilson DM, Hammer L, Kraemer H, Blair-Greiner A, Strachowski D (1996). Weight concerns influence the development of eating disorders: a 4-year prospective study. *Journal of Consulting and Clinical Psychology* 64, 936–940.
- Killen JD, Taylor CB, Hayward C, Wilson DM, Haydel KF, Hammer LD, Simmonds B, Robinson TN, Litt I, Varady A, Kraemer H (1994*b*). Pursuit of thinness and onset of eating disorder symptoms in a community sample of adolescent girls: a 3-year prospective analysis. *International Journal of Eating Disorders* **16**, 227–238.
- **Kraemer HC, Blasey CM** (2004). Centering in regression analyses: a strategy to prevent errors in statistical inference. *International Journal of Methods in Psychiatric Research* **13**, 141–151.
- Kraemer HC, Kazdin AE, Offord DR, Kessler RC, Jensen PS, Kupfer DJ (1997). Coming to terms with the terms of risk. Archives of General Psychiatry 54, 337–343.
- Kraemer HC, Kazdin AE, Offord DR, Kessler RC, Jensen PS, Kupfer DJ (1999). Measuring the potency of risk factors for clinical or policy significance. *Psychological Methods* 4, 257–271.
- Kraemer HC, Lowe KK, Kupfer DJ (2005). To Your Health: How to Understand What Research Tells Us About Risk. Oxford University Press: New York.
- Kraemer HC, Morgan GA, Leech NL, Gliner JA, Vaske JJ, Harmon RJ (2003). Measures of clinical significance. Journal of the American Academy of Child and Adolescent Psychiatry 42, 1524–1529.
- Kraemer HC, Stice E, Kazdin A, Offord D, Kupfer D (2001).
 How do risk factors work together? Mediators, moderators, and independent, overlapping, and proxy risk factors. American Journal of Psychiatry 158, 848–856.
- Leon GR, Fulkerson JA, Perry CL, Early-Zald MB (1995). Prospective analysis of personality and behavioral vulnerabilities and gender influences in the later development of disordered eating. *Journal of Abnormal Psychology* **104**, 140–149.
- **Leon GR, Fulkerson JA, Perry CL, Keel PK, Klump KL** (1999). Three to four year prospective evaluation of personality and behavioral risk factors for later disordered eating in adolescent girls and boys. *Journal of Youth and Adolescence* **28**, 181–196.
- Luce KH, Crowther JH (1999). The reliability of the Eating Disorder Examination – Self-Report Questionnaire Version (EDE-Q). *International Journal of Eating Disorders* 25, 349–351.
- Measelle JR, Slice E, Hogansen JM (2006). Developmental trajectories of co-occurring depressive, eating, antisocial and substance abuse problems in female adolescents. *Journal of Abnormal Psychology* **115**, 524–538.
- Mitchell JE, Myers TC, Glass JB (2002). Pharmacotherapy and medical complications of eating disorders in children

- and adolescents. Child and Adolescent Psychiatric Clinics of North America 11, 365–385.
- Moorhead DJ, Stashwick CK, Reinherz HZ, Giaconia RM, Streigel-Moore RM, Paradis AD (2003). Child and adolescent predictors for eating disorders in a community population of young adult women. *International Journal of Eating Disorders* 33, 1–9.
- Neumark-Sztainer D, Wall MM, Haines JI, Story MT, Sherwood NE, van den Berg PA (2007). Shared risk and protective factors for overweight and disordered eating in adolescents. *American Journal of Preventive Medicine* 33, 359–369.
- Orme JG, Reis J, Herz EJ (1986). Factorial and discriminant validity of the Center for Epidemiological Studies Depression (CES-D) scale. *Journal of Clinical Psychology* 42, 28–33.
- Pike KM, Hilbert A, Wilfley DE, Fairburn CG, Dohm FA, Walsh BT, Striegel-Moore RH (2007). Toward an understanding of risk factors for anorexia nervosa: a case-control study. *Psychological Medicine* **10**, 1–11.
- Plutchik R, van Praag HM (1987). Interconvertability of five self-report measures of depression. *Psychiatry Research* 22, 243–256.
- Rosen JC, Vara L, Wendt S, Leitenberg H (1990). Validity studies of the Eating Disorder Examination. *International Journal of Eating Disorders* **9**, 519–528.
- Safer DL, Telch CF, Agras WS (2001). Dialectical behavior therapy for bulimia nervosa. *American Journal of Psychiatry* 158, 632–634.
- Scher CD, Stein MB, Asmundson GJG, McCreary DR, Forde DR (2001). The Childhood Trauma Questionnaire in a community sample: psychometric properties and normative data. *Journal of Trauma and Stress* 14, 843–857.
- **Spoor ST, Stice E, Burton E, Bohon C** (2007). Relations of bulimic symptom frequency and intensity to psychosocial impairment and health care utilization: results from a community-recruited sample. *International Journal of Eating Disorders* **40**, 505–514.
- **Stice E, Killen JD, Hayward C, Taylor CB** (1998). Support for the continuity hypothesis of bulimic pathology. *Journal of Consulting and Clinical Psychology* **66**, 784–790.
- Striegel-Moore RH, Bulik CM (2007). Risk factors for eating disorders. *American Psychologist* **62**, 181–198.
- Striegel-Moore RH, Dohm FA, Kraemer HC, Taylor CB, Daniels S, Crawford PB, Schreiber GB (2003). Eating disorders in white and black women. *American Journal of Psychiatry* **160**, 1326–1331.
- Strober M, Freeman R, Bower S, Rigali J (1996). Binge eating in anorexia nervosa predicts later onset of substance abuse disorder: a 10-year prospective, longitudinal follow-up of 95 adolescents. *Journal of Youth and Adolescence* **25**, 519–532.
- Stunkard AJ, Sorenson T, Schulsinger F (1983). Use of the Danish adoption register for the study of obesity and thinness. In *The Genetics of Neurological and Psychiatric Disorders* (ed. S. S. Kety, L. P. Rowland, S. W. Sidman and S. W. Mathysee), pp. 115–120. Raven Press: New York.
- Taylor CB, Altman TM, Shisslak C, Bryson S, Estes LS, Gray N, McKnight KM, Kraemer H, Killen JD (1998).

- Factors associated with weight concerns in adolescents. *International Journal of Eating Disorders* **24**, 31–42.
- Taylor CB, Bryson S, Luce KH, Cunning D, Doyle AC, Abascal LB, Rockwell R, Dev P, Winzelberg AJ, Wilfley DE (2006). Prevention of eating disorders in at-risk college-age women. Archives of General Psychiatry 63, 881–888.
- Taylor CB, Bryson SW, Altman TM, Abascal L, Celio A, Cunning D, Killen JD, Shisslak CM, Crago M, Ranger-Moore J, Cook P, Ruble A, Olmsted ME, Kraemer HC, Smolak L (2003). Risk factors for the onset of eating disorders in adolescent girls: results of the McKnight longitudinal risk factor study. *American Journal of Psychiatry* 160, 248–254.
- Wade T, Treloar S, Martin NG (2008). Shared and unique risk factors between lifetime purging and objective binge eating: a twin study. *Australian and New Zealand Journal of Psychiatry* **42**, A26–A26.
- Wechsler H, Lee JE, Kuo MC, Lee H (2000). College binge drinking in the 1990s: a continuing problem. Results of the

- Harvard School of Public Health 1999 College Alcohol Study. *Journal of American College Health* **48**, 199–210.
- Weissman MM, Bothwell S (1976). Assessment of social adjustment by patient self-report. *Archives of General Psychiatry* **33**, 1111–1115.
- Wilfley DE, Bishop ME, Wilson TG, Agras WS (2007). Classification of eating disorders: toward DSM-V. *International Journal of Eating Disorders* **40**, 123–129.
- Wilson TG, Sysko R (2009). Frequency of binge eating episodes in bulimia nervosa and binge eating disorder: Diagnostic considerations. *International Journal of Eating Disorders* 42, 603–610.
- Wonderlich SA, Connolly KM, Stice E (2004). Impulsivity as a risk factor for eating disorder behavior: assessment implications with adolescents. *International Journal of Eating Disorders* **36**, 172–182.
- Zimet GD, Powell SS, Farley GK, Werkman S, Berkoff KA (1990). Psychometric characteristics of the Multidimensional Scale of Perceived Social Support. *Journal of Personality Assessment* 55, 610–617.