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Screening for Depression Prevention: Identifying Adolescent Girls at High Risk for Future Depression

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Abstract

This study investigated a broad array of putative risk factors for the onset of major depression and examined their screening properties in a longitudinal study of 479 adolescent girls. Results indicated that the most potent predictors of major depression onset included subthreshold depressive symptoms, poor school and family functioning, low parental support, bulimic symptoms, and delinquency. Classification tree analysis revealed interactions between four of these predictors, suggesting qualitatively different pathways to major depression. Girls with the combination of elevated depressive symptoms and poor school functioning represented the highest risk group with a 40% incidence of major depression during the ensuing 4-year period. Results suggest that selected and indicated prevention programs should target these high-risk populations and seek to reduce these risk factors.

Keywords

depression; screening; prevention; risk factors; adolescent

Depression is one of the most prevalent mental disorders among adolescents with approximately 20% experiencing an episode of major depressive disorder (MDD) during adolescence (Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993; Newman et al., 1996). Adolescent depression predicts future suicide attempts, substance abuse, antisocial behavior, academic problems, marital difficulties, interpersonal problems, and unemployment (e.g., Newman et al. 1996; Reinherz, Giaconia, Hauf, Wasserman, & Silverman, 1999). Unfortunately, most depressed adolescents do not receive treatment (Newman et al., 1996). Thus, preventing depression is a key public health priority.

Mrazek and Haggerty (1994) distinguish between *universal* prevention programs administered to the entire population, *selected* programs administered to individuals at high-risk for future problems, and *indicated* programs administered to individuals who have symptoms of the disorder but are below diagnostic threshold. Selective depression prevention studies have targeted youth at elevated risk due to poverty, bereavement, divorce, parental depression, and negative cognition style, whereas indicated depression prevention programs have targeted adolescents with elevated depressive symptoms (Horowitz & Garber, 2006). Selective and indicated prevention programs are considered targeted prevention approaches.

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Although investigators have developed efficacious targeted depression prevention programs that produce enduring effects (e.g., Clarke et al., 2001; Gillham, Reivich, Jaycox, & Seligman, 1995; Stice, Burton, Bearman, & Rohde, 2006), no prospective studies have formally assessed the screening properties of various self-report measures that identify sub-populations at risk for MDD. This is an important lacuna because targeted prevention programs produce significantly larger effects than universal programs (Horowitz & Garber, 2006). Improving our understanding of how best to identify high-risk groups for targeted prevention programs would allow interventionists to focus on those at greater risk. Furthermore, Munoz, Le, Clarke, and Jaycox (2002) argue that it is imperative to identify groups at sufficiently high risk to provide adequate statistical power to test whether interventions prevent MDD onset. An improved ability to identify those at greatest risk may increase the effects of depression prevention programs.

Previous research on screening for adolescent depression has focused on identification of individuals with current MDD using self-report measures (Daviss et al., 2006; Reynolds, 1991; Roberts, Lewinsohn, & Seeley, 1991). Although prospective studies have identified risk factors for future MDD onset (e.g., Lewinsohn, Roberts, et al., 1994; Reinherz et al., 1993; Stice, Hayward, Cameron, Killen, & Taylor, 2000), no study has compared the screening properties of various risk factors for detecting future cases of adolescent MDD. Screening properties include sensitivity, specificity, positive predictive value (PPV), and *negative predictive value* (NPV).[1] An efficient method for evaluating the efficacy of a screener is through receiver operating characteristic (ROC) analysis, which examines the sensitivity and specificity of different cut-points of screening measures (Hsiao, Bartko, & Potter, 1989). By calculating the *area under the ROC curve* (AUC), it is possible to compare different screeners or the efficacy of the same screener with different populations. One can also use the ROC curve to select specific cut-points that maximize sensitivity and specificity.

Elucidating risk factors that identify youth at elevated risk for MDD should also advance knowledge regarding etiologic processes for this pernicious disorder. Major psychosocial theories of depression have focused on stressful life events (Compas, 1987), negative cognitions (Beck 1967), and interpersonal relationships (Joiner & Coyne, 1999). The stress exposure theory asserts that those exposed to stressful life events are more likely to experience depression due to disruptions in their routine or changes in perceived competence. Cognitive theories posit that individuals with negative beliefs or stable, internal attributions for failure or proneness towards rumination are more likely to become depressed. Interpersonal theories assert that individuals with deficits in interpersonal behaviors, attachment, or the social environment are more likely to experience depression. Consistent with these theories, stressful life events, negative cognitions, and problematic interpersonal relationships have been found to predict MDD in adolescence (Bohon, Stice, Burton, Fudell, & Nolen-Hoeksema, 2008; Burton, Stice, & Seeley, 2004; Garrison et al., 1997; Lewinsohn et al., 1994; Reinherz et al., 1993). Elevated depressive symptoms have also emerged as potent predictor of adolescent MDD (Lewinsohn et al., 1994; Stice et al., 2000). Although numerous risk factors have been suggested, we focused on those that had the strongest empirical support when this study began (e.g., negative life events, negative attributional style, depressive symptoms) and those that involved body image and eating disturbances (e.g., body dissatisfaction, bulimic symptoms), as data for the present report were drawn from a study on risk factors for eating pathology.

Given that females show a marked increase in MDD during adolescence (Hankin et al., 1998), researchers have argued that preventing depression among adolescent girls should be a

[1]Sensitivity refers to the proportion who screen positive among those who develop MDD; specificity refers to the proportion who screen negative among those who do not develop MDD; PPV refers to the proportion of future MDD cases among those who screen positive; and NPV refers to the proportion of those who do not develop MDD among those who screen negative.

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public health priority and that advances are needed in the accurate identification of adolescent girls at risk for MDD (Le, Munoz, Ippen, & Stoddard, 2003). There has also been interest in developing explanatory models for the precipitous rise in depression for girls during adolescence (e.g., Nolen-Hoeksema & Girgus, 1994). One possible explanation is the gender-additive model (Stice et al., 2000) which posits that girls face additional risk factors for depression beyond those shared with boys, which escalate in early adolescence and create additional pathways to depression. In support, initial body dissatisfaction and bulimic symptoms are stronger predictors of future MDD for adolescent girls relative to boys and these effects often emerged when controlling for gender non-specific risk factors (e.g., elevated depressive symptoms, stressful life events) (Bearman & Stice, in press; Stice et al., 2000).

Garber (2006) advocated for an examination of risk mechanisms that may elucidate the pathways linking risk factors to MDD onset, yet few prospective studies have examined how risk and protective factors interact to increase the risk for MDD. The identification of such nonlinear epigenesis illustrates potentially diverse pathways to depression, a process that has been referred to as equifinality (Cicchetti & Rogosch, 1996). Two multivariate models are the cognitive vulnerability model (Beck, 1967) and the stress-buffering model (Cohen & Wills, 1985). The cognitive vulnerability model posits that negative cognitions represent a diathesis that in combination with stressful events lead to negative thoughts, which in turn increase the risk for MDD. The stress-buffering model proposes that social support mitigates the relation between stressful life events and MDD onset. Some prospective investigations have provided support for these models (Bohon et al., 2008; Nolen-Hoeksema, Girgus, & Seligman, 1992), whereas others have not (Burton et al., 2004; DuBois, Felner, Brand, Adan, & Evans, 1992).

The primary aim of the present study is to investigate a broad array of putative risk factors for MDD onset and examine the screening properties for these measures that optimally predict MDD onset. The secondary aim is to conduct a classification tree analysis (CTA) to test for interactive effects between risk factors (Kraemer, Wilson, Fairburn, & Agras, 2002) and illuminate potential pathways to depression. CTA is sensitive to detecting interactions that may be nonlinear. To our knowledge, CTA has not been applied to predicting MDD onset in adolescents. The identification of interactive effects can both facilitate screening strategies of specific combinations of measures that identify groups of adolescent girls at risk for MDD and provide empirical support for potentially novel risk and protective mechanisms in etiologic models of depression.

Method

Participants

Participants were 496 adolescent females from public and private middle schools in a large city. They were in 10^{th} and 11^{th} grades at Time 1 (T1) for this report (*M* age = 15.5, *SD* = 0.67, range 15-18). The sample included 2% Asian, 7% African Americans, 68% Caucasians, 18% Hispanics, 1% Native Americans, and 4% who specified other or mixed racial heritage, which was representative of the ethnic composition of the schools from which we sampled. Average parental education was 29% high school graduate or less, 23% some college, 33% college graduate, and 15% graduate degree, which was representative of the metropolitan area. Of the 496 participants, 485 had data available at the beginning of the 4-year observation period, of which, six met criteria for MDD at the beginning of the observation period and were excluded. Thus, the reference sample for this report was 479 participants; the cumulative attrition rate across the 4-year observation period was 5.4%.

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Procedures

The study was described as an investigation of adolescent mental and physical health. Active parental consent was used, wherein an informed consent letter and stamped return envelope were sent to parents of eligible female students (a second mailing was sent to non-responders). This resulted in a participation rate of 56%, which was comparable to other school-recruited samples (e.g., 61% for Lewinsohn et al., 1994). Participants completed a survey and structured interview at baseline and four annual follow-ups. Female assessors with a bachelor or graduate degree in psychology conducted all interviews. Assessors initially attended 24 hours of training, in which they were taught interviewing skills, reviewed diagnostic criteria, observed simulated interviews, and role-played interviews. Assessors demonstrated high inter-rater agreement (kappa [*k*] > .80) with the project coordinator using 12 tape-recorded interviews conducted with individuals with and without major depression and eating disorders before collecting data. Assessors attended training workshops twice a year throughout the study to reduce interviewer drift. A randomly selected subset of participants (5% annually) completed a second interview by the same assessor to assess test-retest reliability and another randomly selected subset of participants (5% annually) completed a second interview with another blinded assessor to examine inter-rater agreement. Interviewers who showed poor test-retest reliability or inter-rater agreement were no longer permitted to collect data. Assessments took place at the school or at participants' residences. Participants were paid for completing each survey and interview (even if they elected to skip certain questions).

Measures

Parental and peer support—Perceived support from parents and peers was measured with 12 items from the 363-item Network of Relationships Inventory (Furman & Buhrmester, 1985). We selected items assessing companionship, guidance, intimacy, affection, admiration, and reliable alliance from parents/guardians and from peers (e.g., I could count on my parent (s) to be there when I needed them). Adolescents rated these items on 5-point scales ranging from 1 = *strongly disagree to* 5 = *strongly agree*. Items were averaged to form parent and peer support scales, as were those for the scales reported below unless otherwise indicated. These two scales have shown internal consistency (*M* α = .88), 1-month test-retest reliability (*M r* = .69), and participants who report lower support on these scales are at increased risk for future MDD onset (*M* OR = 1.8) (Furman, 1996; Stice, Ragan, & Randall, 2004).

Negative life events—The Major Life Events scale (Lewinsohn et al., 1994) assessed the occurrence of nine stressful events in the past year (e.g., *Did your parents get divorced or separated?*). Response options were 0 = *no,* 1 = *once*, and 2 = *at least twice*. This scale has shown 1-week test-retest reliability (*r* = .90) and participants with elevated scores on this scale are at greater risk for future MDD onset (*M* OR = 1.4) (Burton et al., 2004; Lewinsohn et al., 1994).

Attributional style—Sixteen items from the 60-item Adolescent Cognitive Style Questionnaire (ACSQ: Hankin & Abramson, 2002) assessed attributional style. The ACSQ presents positive and negative hypothetical events (e.g., *You take a test and get a bad grade*); participants rate the degree to which they believe the event is internal, stable, global, and signifies that the person is flawed using forced choice response options. We randomly selected 12 items for use in this study to minimize respondent burden. Healthy attributions for positive events are coded 1 (vs. 0) and healthy attributions for negative events are coded −1 (vs. 0). A square-root transformation was used to normalize this scale. This version of the ACSQ has shown internal consistency (α = .95) and 2-week test-retest reliability (*r* = .73) and participants with elevated scores show greater future increases in depressive symptoms (*r* = .16) (Bohon et al., 2008).

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Emotionality—Buss and Plomin's (1984) Emotionality Scale assessed temperamental negative affectivity. Using a modified response format than the original scale, participants indicated how much they agree with eight items regarding their tendency to become emotionally distressed or aroused (e.g., *I frequently get upset*) on 5-point response format ranging from 1 = *strongly disagree* to 5 = *strongly agree*. This scale has shown internal consistency (α = .82), 1-week test-retest reliability (*r* = .71 95% CI = .43 – 1.00) in a pilot study with adolescents (*N* = 25) and participants with elevated scores on this scale are at greater risk for future increases in bulimic symptoms (β = .20) (Buss & Plomin, 1984; Stice, 2001).

Perfectionism—Three items from the perfectionism scale of the Eating Disorder Inventory (Garner, Olmstead, & Polivy, 1983) assessed trait perfectionism. Participants indicated the truth-value of items regarding perfectionistic tendencies on 5-point response format ranging from 1 = *never* to 5 = *always*. This scale has shown internal consistency (α = .80) and 1-week test-retest reliability (*r* = .88) (Thiel & Paul, 2006).

Body dissatisfaction—The 8-item Body Dissatisfaction Scale (Stice, 2001) assesses satisfaction with body parts (e.g., stomach, hips) using response options ranging from 1 = *extremely satisfied* to 6 = *extremely dissatisfied*. This scale has shown internal consistency (α = .94), 3-week test-retest reliability (*r* = .90), and participants with elevated scale scores are at greater risk for future increases in bulimic symptoms (β = .25) (Stice, 2001).

Bulimic symptoms—Diagnostic items from the Eating Disorder Diagnostic Interview (Stice, Marti, Spoor, Presnell, & Shaw, 2008), a semi-structured interview, assessed DSM-IV bulimia nervosa symptoms. Items assessing the frequency of binge eating and compensatory behaviors in the past month and the severity of weight/shape concerns (0 = *no importance* to 6 = *supreme importance*) were summed to create an overall bulimic symptom composite. A square root transformation was used to normalize this variable. This symptom composite showed internal consistency (α = .92), 1-week test-retest reliability (*r* = .90), sensitivity to intervention effects, and participants with elevated scores on this scale are at increased risk for future MDD onset (OR = 1.7) (Stice et al., 2008; Stice, Burton et al., 2004).

Physical activity—The Past Year Leisure Physical Activity Scale assessed exercise (Aaron et al., 1995). Participants indicate whether they engaged in 24 activities more than 10 times over the past year; then they report how many months out of the year, days per week, and minutes per day they engaged in each endorsed activity. Items were weighted according to metabolic expenditure and summed to create an overall measure of past year caloric expenditure through exercise. This scale had shown internal consistency (α = .93), 1-month test-retest reliability (*r* = .79) and convergent validity with activity monitors (Aaron et al., 1995; Kimm et al., 2000).

Social adjustment—Family, peer, and school functioning was measured with 17 items from the 54-item Social Adjustment Scale-Self Report for Youth (Weissman, Orvaschel, & Padian, 1980) (response options: 1 = *never* to 5 = *always*). This adapted scale has shown internal consistency (*M* α = .77), 1-week test-retest reliability (*M r* = .83) and sensitivity to treatment (Burton & Stice, 2006; Stice et al., 2008). In a pilot study (*N* = 25), the family, peer, and school subscales showed 1-week test retest reliability (*r* = .82 [95% CI = .48 – .89]; *r* = .93 [95% CI = .78 – 1.00]; *r* = .82 [95% CI = .65 – 1.00]) and were only modestly inter-correlated (*r* = .11 to .31)

Delinquency—We selected 13 items that loaded on the Externalizing Scale for adolescent girls from the Child Behavior Checklist (Achenbach & Edelbrock, 1983). Participants reported the frequency of each behavior over the past year using a response format ranging from 1 =

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never to 5 = *always*. This scale has shown internal consistency (α = .88), 1-week test-retest reliability (*r* = .81, 95% CI = .59 – 1.00) in a pilot sample of adolescents (*N* = 25), correlated significantly with the full externalizing scale of the CBCL (*r* = .87) in a sample (*N = 87*) of female adolescents (Nolen-Hoeksema, Stice, Wade, & Bohon, 2007) and participants with elevated scores on this scale showed greater future increases in depressive symptoms (β = .39) (Measelle, Stice, & Hogansen, 2006).

Substance use—Ten items assessing substance use were adapted from Johnston, O'Malley, and Bachman (1989). A dolescents reported their frequency of consumption during the past 6 months of beer/wine/wine coolers, hard liquor, cigarettes, marijuana, stimulants, sedatives, inhalants, and hallucinogens (response options ranged from 0 = *never* to 6 = *3-7 times a week*), as well as the typical number of drinks consumed during a drinking episode and the number of cigarettes smoked daily (response options ranged from 0 to 21 or more). This scale has shown internal consistency (*r* = .87) and 1-month test-retest reliability (*r* = .94) (Burton, Stice, Bearman, & Rohde, 2007).

Depressive symptoms and diagnosis—An adapted version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS; Puig-Antich & Chambers, 1983), a semi-structured diagnostic interview, assessed DSM-IV MDD symptoms. Participants reported the peak severity of each symptom during the past year using response options ranging from 1 = *not at all* to 4 = *severe symptoms* (ratings of 3 and 4 constituted diagnostic level). Responses were used to determine whether the participant had met criteria for MDD over the past year at each assessment. Symptoms had to occur concurrently for a diagnosis. This version of the K-SADS depression interview has shown 1-week test-retest reliability (*k* = 1.00) and inter-rater agreement (*k* = 1.00) for depression diagnosis and internal consistency (*M* α = .81), 1-week test-retest reliability (*r* = .86) and inter-rater agreement (*r* = . 87) for the symptom composite in the present sample (Nolen-Hoeksema et al., 2007).

Results

Predictors of MDD Onset

Univariate and multivariate Cox regression models were used to predict the time to MDD onset during the 4-year follow-up. Of the 479 participants, 62 (13%) developed MDD. Fifteen of the 62 incident cases (24%) had a previous history of MDD at baseline and 17 (27%) experienced multiple MDD episodes during follow-up.

Univariate models—Table 1 presents results of univariate models predicting time to MDD onset. Eleven of the 18 predictors (61%) significantly predicted time to MDD at $p < .05$. Applying a more conservative Bonferroni correction ($p = .05/18 = .0028$), 8 of the 19 (44%) predictors would be significant. The proportional hazards assumption was met for all predictors. Past history of MDD did not moderate the association between any predictor and time to MDD onset.

Table 1 shows the hazard ratios, which reflect the increase in likelihood of developing MDD for each one-unit increase on the predictor. Because the interpretation of the hazard ratios for continuous variables depends on the scaling of the measure, we also report the generalized R^2 to facilitate direct comparisons of the magnitude of effects. The strongest effects emerged for depressive symptoms, poor school functioning, low parental support, poor family functioning, bulimic symptoms, and delinquency.

Multivariate model—Examination of correlations between predictors confirmed that there was only moderate co-linearity (all *r*'s were smaller than .51), suggesting multicolinearity

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would not produce unstable parameter estimates. All predictors, demographic factors, and past MDD history were entered simultaneously into the model in which only two variables showed significant unique effects: depressive symptoms (likelihood χ^2 [1, N = 479] = 9.59, p = .002) and poor school functioning (likelihood χ^2 [1, N = 479] = 4.36, p = .037).

Interactive Effects: Classification Tree Analysis (CTA)

CTA (Breiman, Friedman, Olshen, & Stone, 1984) tested whether variables interacted in predicting MDD onset. CTA provides a nonparametric alternative to linear and additive logistic models for dichotomous outcomes. Trees are fitted using a binary recursive partitioning approach that selects the optimal cut-point on the most potent risk factor for generating subgroups with differential risk for the outcome. This procedure is then repeated on the resulting subgroups until there are no remaining predictors that identify subgroups at significantly differential risk or the node sizes become too small. When different risk factors emerge for two branches from the same fork, it signifies an interaction.

Because variables with nonsignificant main effects could potentially interact significantly with other variables, all predictors were included in the CTA. The minimum node size was set at 20 to minimize Type I error and influential outliers. The CTA produced a classification tree with four forks and five terminal nodes (Figure 1). As would be expected based on the univariate analyses, depressive symptoms emerged as the most potent predictor of MDD onset. Girls with elevated depressive symptoms were at approximately five times greater risk for MDD than girls who reported less symptoms (MDD incidence rate = 28% vs. 6%; χ^2 (1, N = 479) = 42.63, p <.001). Low parental support emerged as the next risk factor, but only among those with low depressive symptoms (MDD incidence rate = 22% vs. 3%; χ^2 (1, N = 327) = 25.13, p <.001). In contrast, poor school functioning emerged as a risk factor only among those with elevated depressive symptoms (MDD incidence rate = 40% vs. 18%; χ^2 (1, N = 152) = 8.69, p = .003). The last significant predictor was bulimic symptoms among girls with high parental support and low depressive symptoms (MDD incidence rate = 13% vs. 2%; χ^2 (1, N = 276) = 4.267, p = .034), though only three incident MDD cases were identified via this pathway. Thus, the CTA revealed a two-way interaction between depressive symptoms and parental support, a two-way interaction between depressive symptoms and school functioning, and a three-way interaction between bulimic symptoms, parental support, and depressive symptoms. The CTA resulted in a 75% accuracy rate in predicting MDD onset.

Based on the CTA findings, we compared participants who developed MDD via different pathways on five clinical characteristics: time to MDD onset, episode duration, symptom severity, number of episodes during the follow-up period, and previous MDD history. Table 2 presents descriptive statistics for these characteristics for the following three pathway groups (a) elevated depressive symptoms with poor school functioning (n = 26), (b) elevated depressive symptoms without poor school functioning (n =16), and (c) low parental support without elevated depressive symptoms (n = 11); the pathway group that included bulimic symptoms was too small (n = 3) for meaningful comparisons. Because of the limited power associated with small groups, effect sizes (i.e., Cohen's d and odds ratios [OR]) were used to interpret group differences rather than statistical tests. The low parental support pathway group had a longer time to episode onset (d = .80), shorter episode duration (d = .52), lower percentage with recurrent episodes (OR = 2.25; 95% CI = 0.43 – 11.85), and a lower percentage with past MDD history (OR = 4.48; 95% CI = 0.52 – 38.76) compared to the two other pathway groups (combined). All three groups had comparable symptom severity ratings during the MDD episode.

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Screening Properties

To compare screening properties of the significant predictors, we used ROC analysis to select the cut-point that optimized rates of both sensitivity and specificity for predicting MDD onset (Table 3). The area under the ROC curve (AUC) provides an overall index of screening performance for continuous scale scores. In terms of effect size corresponding to Cohen's *d*, AUC values of .56, .64, and .71 represent small, medium, and large effects (Rice & Harris, 2005). Consistent with the Cox models, depressive symptoms had the best screening properties and a large effect size (AUC = .75). At the optimal cutoff score, depressive symptoms correctly classified 68% of MDD cases (sensitivity) and 74% of those who did not develop MDD (specificity) for an overall accuracy rate of 73%; 28% of those who screened positive developed MDD (PPV); 94% of those who screened negative did not develop MDD (NPV); and 32% of the sample screened positive. Poor school functioning, low parental support, poor family functioning, delinquency, and bulimic symptoms had medium size effects and moderate screening properties. The kappa values indicate that the degree of predictive power beyond chance is modest for the top five predictors and minimal for the remaining predictors.

We also examined whether the screening properties can be improved by combining multiple risk factors. Combining risk factors involves decision rules based on "and" and "or" logical operators. The "and" rule specifies that *all* of the risk factors in the combination are present to screen positive; the "or" rule specifies that *any* of the risk factors in the combination be present to screen positive. Table 4 presents screening properties for combinations of the three most potent risk factors using the ROC cut-points: depressive symptoms, poor school functioning, and low parental support. As can be seen, the "and" rule maximizes specificity (.85) and PPV (.35) at the expense of sensitivity (.53) and NPV (.93), whereas the "or" rule does the converse. A positive screen on any of the three risk factors yielded the highest sensitivity (.94) but lowest PPV (.20) and overall accuracy (.51).

Discussion

The first aim was to identify significant predictors of MDD onset and their screening properties in a sample of female adolescents across a 4-year follow-up. Eleven of the 18 variables (61%) significantly predicted MDD onset. Elevated depressive symptoms emerged as the strongest risk factor and best single screening measure, converging with prior findings (e.g., Lewinsohn et al., 1994; Stice et al., 2000). Roberts et al. (1991) reported that two depression questionnaires (Beck Depression Inventory and Center for Epidemiologic Studies – Depression scales) had sensitivities and specificities of approximately .80 for detecting concurrent MDD, which compares favorably to the .70 value in the current study for predicting future MDD onset. PPV was higher in the current study than in Roberts et al. (.28 versus .08-.10), presumably because the proportion of participants who developed MDD during a 4-year follow-up was higher than the point prevalence rate observed by Roberts et al. (13% versus 3%). Because PPV represents the risk of having or developing the disorder among those who screened positive on the basis of a given risk factor or set of risk factors, it is a useful index for researchers, clinicians, and other public health personnel for identifying high risk groups.

It is tempting to suggest that elevated depressive symptoms are simply a prodromal sign of MDD. However, the fact that most individuals with elevated symptoms do not develop MDD suggests this explanation is incomplete. It is also possible that the elevated depressive symptoms are residual effects from a previous episode, which is associated with recurrence. Yet, this interpretation is not consistent with the finding that depressive symptoms predicted MDD onset controlling for a history of MDD. A more likely explanation is that depressive symptoms may escalate into full MDD if other factors are present, such as impaired school functioning. An important priority is to determine the mechanisms by which elevated depressive symptoms increase risk for MDD onset. Future research should conduct more

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frequent and detailed follow-up of adolescents with elevated symptoms to understand the progression from elevated symptoms to diagnosis and should consider the influence of previous MDD episodes. Understanding why the majority of adolescents with elevated symptoms do not become depressed may also inform theories of protective factors and resiliency, and might implicate other factors that amplify the risk conveyed by elevated depressive symptoms (e.g., stress, demoralization, alternative forms of grief, physical illness). Such findings would have implications regarding the etiology of depressive disorders and potentially regarding conceptualization of the diagnosis of depression. Regardless, elevated depressive symptoms is a key screening criterion for indicated prevention programs that focus on individuals who are experiencing early signs of pathology but have not yet experienced onset of full MDD.

Poor school functioning also showed a significant unique relation to risk for MDD onset in the multivariate model, suggesting it may be fruitful for selected prevention programs to target individuals with academic difficulties. Other easily measured predictors also showed moderate screening properties, including low parental support, poor family functioning, delinquency, and bulimic symptoms. Although these risk factors had a lower PPV than elevated depressive symptoms and poor school functioning, many are easily assessed and may be useful for interventions that target such factors (e.g., bulimic symptoms or delinquency) as a means of reducing risk for future MDD. For example, Stice et al. (2008) found that eating disorder prevention programs can have the added benefit of reducing affective disturbances in female adolescents.

The second aim was to investigate the interactive effects between risk factors. Results indicated that the combination of elevated depressive symptoms and poor school functioning was especially predictive of future MDD. This finding may have emerged because poor school functioning is a product of more severe depressive symptoms or because specific depressive symptoms that contribute to school impairment (e.g., concentration difficulties and insomnia) may be important in predicting MDD onset. Alternatively, poor school functioning may lead to an exacerbation of depression or some third factor variable (e.g., ADHD) contributes to an increased risk for both poor school functioning and subsequent MDD. Good school functioning might also serve as a protective factor--high school can be stressful academically and interpersonally, but youth with elevated depressive symptoms who function well in school may be buffered against developing MDD. These findings might suggest that many adolescents experience periods of subthreshold depression symptoms, but that elevated symptoms need to result in functional impairments to trigger MDD.

Results also suggested that, in the absence of elevated depressive symptoms, low parental support or bulimic symptoms might be alternate pathways to MDD for adolescent girls. One interpretation of the results based on the effect sizes is that predictive effects for less potent risk factors emerged only when more potent risk factors are not operating. The predictive effects of low parental support emerged only for participants without elevated depressive symptoms and the predictive effects of eating pathology emerged only for participants without elevated depressive symptoms or parental support deficits. The evidence that there may be qualitatively different pathways to depression is novel. Results also suggested that adolescents who reach MDD through different pathways experience disorders that have different clinical presentations. Indeed, adolescents who did not have elevated depressive symptoms but had low parental support took almost twice as long to develop MDD as adolescents with elevated depressive symptoms (with or without school impairment). This subset of young women also had a much lower rate of previous MDD. Findings suggest that subthreshold depressive symptoms may represent both a prodromal stage of MDD (although the average time to MDD onset was still over one year for those with elevated symptoms) and a lingering aspect of previous MDD for approximately 30% of the participants who had elevated depressive symptoms. One study suggested that elevated depressive symptoms are a scar of depression in

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adolescents (Rohde, Lewinsohn, & Seeley, 1994), though another study that examined individuals before, during, and after an MDD episode suggested the elevated depressive symptoms precede MDD (Beevers, Rohde, Stice, & Nolen-Hoeksema, 2007). Future research should evaluate the degree to which MDD episodes emerging from various pathways have a different clinical course.

Regarding parental support, low perceived support may be particularly demoralizing, as it is often expected that parents will provide unconditional positive support. Moreover, adolescents who cannot count on instrumental and emotional parental support may experience more functioning difficulties or have fewer social resources during this high-risk developmental period. It was noteworthy that parental support deficits predicted MDD onset for those who did not have elevated depressive symptoms. A better understanding of the factors that led to the low support is needed. Low parental support could be a consequence of parental depression or a product of the adolescent's behavior (e.g., delinquency). Future research should test whether interventions that enhance parental support reduce risk for MDD onset in female adolescents.

The evidence that bulimic symptoms predicted MDD onset provides further support for the gender-additive model in explaining the elevated rate of depression for female adolescents (Stice et al., 2000). However, it is important to note that this variable emerged as significant predictor only in the absence of depressive symptoms and low parental support within the CTA model. Interestingly, these results suggest that high parental support does not buffer adolescent girls with bulimic symptoms from developing future MDD.

Several negative findings were noteworthy. Though negative cognitive style is central to etiological theories of depression (e.g., Beck, 1967), attributional style did not emerge as a significant predictor of MDD, either as a main effect or in interaction with either mild dysphoria (the differential activation hypothesis; Teasdale, 1983) or negative life events (the cognitive vulnerability risk mechanism; Beck, 1967). Although such putative risk/protective mechanisms cannot be disproved by CTA, they did not emerge as pathways to MDD in the current study.

Limitations of the Current Study

Although this study employed multiple-method data collection, used semi-structured interviews to assess MDD, examined the screening properties for an array of risk factors, and followed a large sample of young women over four years, limitations of this study should be noted. First, the sample consisted of only females, so findings should be generalized with caution to males. Second, the age of sample at the beginning of the study period (age range 15-18) was past the age at which the incidence of depression begins to escalate. Hence, the study findings may not inform on prevention efforts during the critical period of emerging adolescence. Third, several important depression risk factors were not assessed (e.g., pessimism, dysfunctional attitudes, interpersonal dependency, poverty, family conflict, coping skills, physical health, parental depression, child abuse, comorbidities) because of respondent burden concerns. The results from the multivariate models might have differed if additional risk factors had been included. Fourth, certain measures evidenced only moderate test-retest reliability in previous studies, which may have limited predictive power. Fifth, because one purpose of the study was to evaluate the ability to screen for future MDD, we focused on measures that could be assessed by self-report. Although self-report is susceptible to potential mood-state effects, it is one of most efficient and commonly used methods for large-scale screening. Lastly, the moderate recruitment rate suggests some caution should be exercised when generalizing the findings.

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Clinical and Research Implications

The present findings have important research and clinical implications. Because this is the first study to provide comprehensive screening information on numerous risk factors that can be used to identify female adolescents at high risk for MDD onset, the results can inform the design and methodology of future targeted prevention efforts. Consistent with past findings, elevated depressive symptoms emerged as the most potent risk factor for MDD which provides further support for indicated depression prevention programs. Other potent risk factors relevant to selective interventions include both contextual factors (e.g., low parental support) and individual characteristics (e.g., bulimic symptoms) that identify high-risk youth.

As recommended by Jaycox and associates (1994), to maximize the reach of targeted prevention programs, we should consider targeting multiple risk groups. For example, a screening strategy based on the study data would be to assess depressive symptoms, poor school functioning, and low parental support and identify those girls who are positive on any one of the three risk factors. Such a screening strategy would maximize sensitivity (94%), but would also increase sample heterogeneity and would be resource intensive due to the high percentage of girls who would be screened positive (60%) for targeted prevention efforts. Researchers could also use a serial screening strategy that focuses on combinations of risk factors to identify groups at highest risk for MDD if they were interested in maximizing the ability to detect intervention effects. For example, the highest PPV was obtained by requiring a positive screen on both elevated depressive symptoms and poor school functioning. Hence, if the goal of the screening effort is to maximize the incidence rate in order to improve statistical power for detecting intervention effects, then requiring elevated scores on the both depressive symptoms and poor school functioning would be a recommended strategy. However, this strategy would result in a sensitivity of only 53%. Clearly, when combining multiple risk factors for screening purposes, there are tradeoffs for employing the “or” versus “and” logical operator.

Findings also identified several malleable risk factors that could be targeted in prevention programs. Although we have offered potential directions for the design of prevention interventions, these models may be overly simplistic because the effects may be driven by omitted third variables, which is a possible confound for any prospective investigation. Nonetheless, the present findings may be helpful in suggesting hypotheses to be tested in future etiologic studies. Refining etiologic models to include gender-specific pathways to depression may advance our understanding of this heterogeneous disorder. Future studies should examine gender-specific pathways to MDD in samples with both male and female adolescents.

To our knowledge, this is the first study that has employed CTA to explore potential pathways to depression. Although it is constrained by the limited array of risk factors examined herein, CTA provides an efficient way of examining how various combinations of risk factors predict future conditions and is a useful compliment to standard multivariate approaches that are more limited in regard to testing the multitude of risk factor combinations. Because CTA should be considered as a hypothesis-generating rather than hypothesis-confirming approach (Kraemer et al., 2002), it will be important to cross-validate the findings in independent samples. An improved understanding of the various pathways to depression may permit the development of more effective prevention programs, as all MDD episodes create impairment. Nonetheless, future research might consider focusing on identifying the subset of future MDD episodes that are the most severe, recurrent, and impairing.

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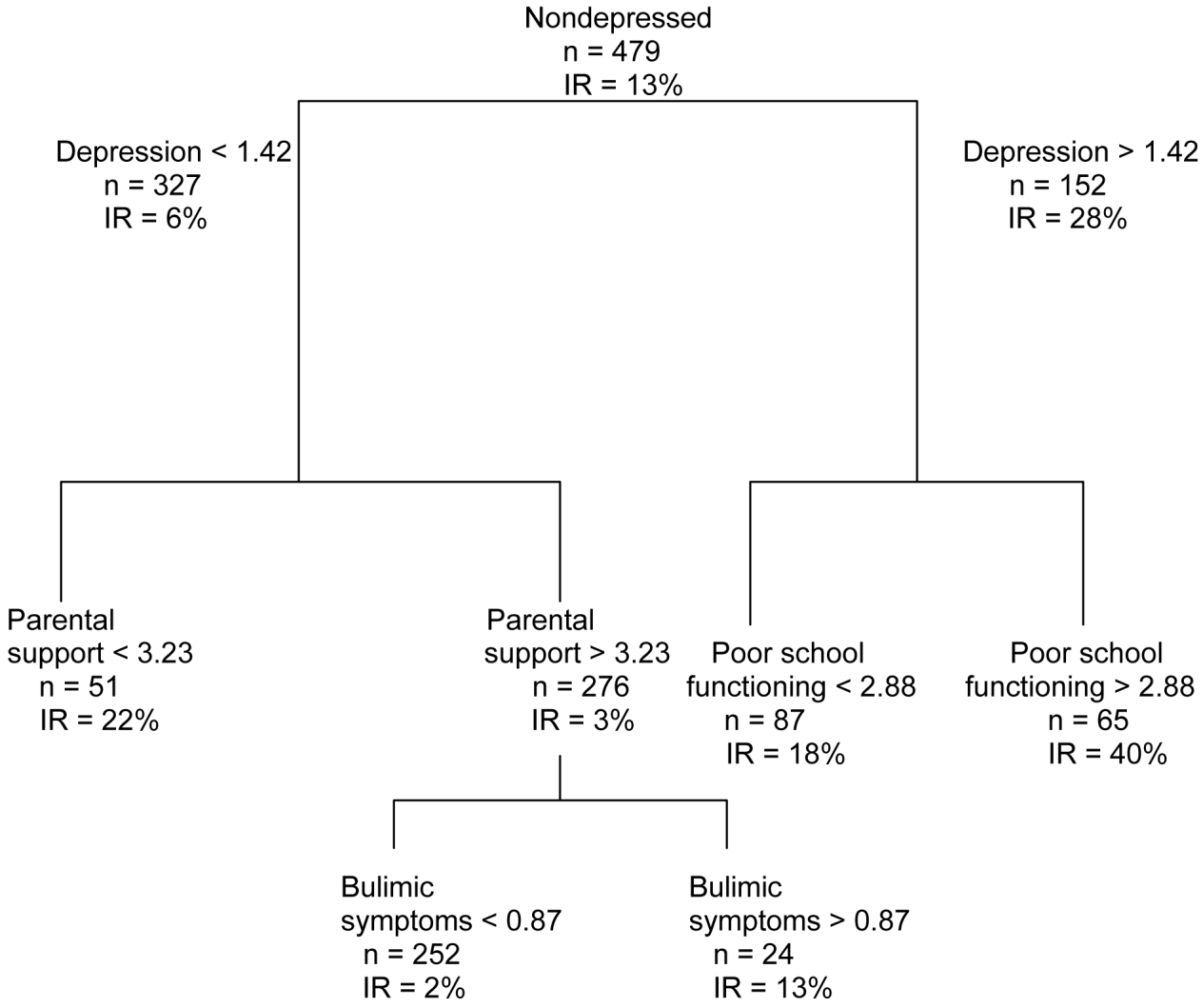

Figure 1.
Graphical depiction of the decision rules for the classification tree analysis predicting onset of major depression. The empirically derived cut-points are shown, along with the sample size and the incidence rate for major depression onset during the study period for each branch and node. IR = incidence rate of major depression.

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Table 1

Summary of Univariate Cox Models Predicting Onset of Major Depression

	Hazard ratio	Hazard ratio 95% CI		R^2	p-value
Race	1.00	0.81	1.24	.001	.984
Age	1.00	0.69	1.44	.000	.979
Parent education	0.90	0.73	1.11	.002	.337
Low parental support	1.70	1.35	2.15	.041	<.001
Low peer support	1.40	1.07	1.83	.008	.015
Negative life events	1.14	1.02	1.27	.011	.021
Negative attributional style	1.16	0.86	1.56	.001	.334
Emotionality	1.72	1.25	2.36	.023	.001
Perfectionism	1.06	0.86	1.31	.001	.586
Body dissatisfaction	1.54	1.19	2.00	.019	.001
Bulimic symptoms	3.42	1.96	5.96	.036	<.001
Low physical activity	1.13	1.01	1.27	.010	.030
Poor peer functioning	1.24	0.74	2.06	.001	.411
Poor family functioning	1.67	1.31	2.13	.038	<.001
Poor school functioning	2.26	1.64	3.11	.046	<.001
Delinquency	1.76	1.31	2.38	.032	<.001
Substance use	1.01	0.97	1.046	.000	.798
Depressive symptoms	5.87	3.48	9.880	.092	<.001

Note. CI = confidence interval.

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Table 2
Descriptive Statistics for Major Depressive Episode Characteristics by CTA Risk Groups

Characteristic	Depressive symptoms with poor school functioning (*n* = 26)	Depressive symptoms without poor school functioning (*n* = 16)	Low parental support without depressive symptoms (*n* = 11)
Time to onset (months)			
M (SD)	12.2 (11.1)	16.7 (16.4)	25.8 (17.1)
95% CI	7.7 – 16.7	7.9 – 25.4	14.3 – 37.4
Duration of episode (months)			
M (SD)	7.5 (6.4)	6.5 (4.2)	4.4 (3.2)
95% CI	4.9 – 10.1	4.3 – 8.7	2.2 – 6.5
Symptom severity			
M (SD)	2.52 (0.39)	2.58 (0.43)	2.55 (0.41)
95% CI	2.36 – 2.68	2.36 – 2.81	2.27 – 2.83
Multiple new episodes			
% (SE)	26.9 (8.7)	43.8 (12.4)	18.2 (11.6)
95% CI	9.9 – 43.9	19.5 – 68.1	0.0 – 41.0
Previous MDD history			
% (SE)	34.6 (9.3)	25.0 (10.8)	9.1 (8.7)
95% CI	16.3 – 52.9	3.8 – 46. 2	0.0 - 26.1

Note. CTA = Classification tree analysis; CI = confidence interval.

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Table 3

Screening Properties for the Significant Predictors of Major Depression Onset

Predictor	AUC	Cutoff score	Sens	Spec	PPV	NPV	Positive screen	Overall accuracy	κ
Depressive symptoms	.75	1.42	.68	.74	.28	.94	.32	.73	.26
Poor school functioning	.68	2.63	.65	.70	.24	.93	.35	.69	.20
Low parental support	.66	3.75	.61	.67	.22	.92	.37	.66	.16
Poor family functioning	.66	3.25	.63	.65	.21	.92	.39	.64	.15
Delinquency	.66	1.60	.68	.61	.21	.93	.42	.62	.15
Bulimic symptoms	.65	0.66	.60	.56	.17	.90	.46	.56	.07
Emotionality	.62	2.81	.57	.59	.17	.90	.43	.59	.08
Body dissatisfaction	.62	3.00	.57	.58	.17	.90	.44	.58	.07
Low peer support	.59	4.40	.52	.61	.17	.90	.40	.60	.07
Negative life events	.59	1.68	.63	.51	.16	.90	.51	.53	.06
Low physical activity	.58	3.50	.52	.57	.15	.90	.45	.56	.04

Note. AUC = area under the curve; Sens = sensitivity; Spec = specificity; PPV = positive predictive value; NPV = negative predictive value; Positive screen = proportion of sample screened positive at cutoff score; Overall accuracy = proportion of sample correctly classified; K = kappa statistic

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Table 4

Screening Properties for CTA Risk Factor Combinations

Risk factor combination / Logical operator rule	Sens-itivity	Spec-ificity	PPV	NPV	Positive screen	Overall accuracy
Depressive symptoms and/or poor school functioning						
“And” rule	.53	.85	.35	.93	.20	.81
“Or” rule	.79	.58	.22	.95	.47	.61
Depressive symptoms and/or low parental support						
“And” rule	.42	.91	.30	.86	.18	.80
“Or” rule	.87	.55	.22	.97	.51	.59
Poor school functioning and/or low parental support						
“And” rule	.37	.85	.26	.90	.18	.78
“Or” rule	.89	.52	.22	.97	.53	.57
Depressive symptoms and/or poor school functioning and/or low parental support						
“And” rule	.32	.90	.33	.90	.13	.84
“Or” rule	.94	.45	.20	.98	.60	.51

Note. CTA = Classification Tree Analysis; PPV = positive predictive value; NPV = negative predictive value; Positive screen = proportion of sample screened positive; Overall accuracy = proportion of sample correctly classified. The “and” rule specifies that *all* of the risk factors in the combination are present to screen positive; the “or” rule specifies that *any* of the risk factors in the combination are present to screen positive.