A Randomized Trial of a Group Cognitive Intervention for Preventing Depression in Adolescent Offspring of Depressed Parents

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Background: Adolescent offspring of depressed parents are at high risk for development of depression. Cognitive restructuring therapy holds promise for preventing progression to depressive episodes.

Methods: A randomized, controlled trial was conducted to prevent depressive episodes in at-risk offspring (aged 13-18 years) of adults treated for depression in a health maintenance organization (HMO). Potential adult cases were found by reviewing the HMO pharmacy records for dispensation of antidepressant medication and the mental health appointment system. Medical charts were reviewed for a depression diagnosis. Recruitment letters signed by treating physicians were mailed to adults. Eligible offspring had subdiagnostic depressive symptoms insufficient to meet full DSM-III-R criteria for affective disorder and/or a past mood disorder. These youth were randomized to usual HMO care (n=49) or usual care plus a 15-session group cognitive therapy prevention program (n=45).

Results: We detected significant treatment-by-time (program) effects for the Center for Epidemiological Studies Depression Scale ($P=.005$) and the Global Assessment of Functioning scores ($P=.04$). Survival analysis of incident major depressive episodes during a median 15-month follow-up found a significant advantage ($P=.003$) for the experimental condition (9.3% cumulative major depression incidence) compared with the usual-care control condition (28.8%).

Conclusion: A brief, group cognitive therapy prevention program can reduce the risk for depression in the adolescent offspring of parents with a history of depression.

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Depression is common among adolescents, with a point prevalence estimated at 3% to 8%.1 By 18 years of age, as many as 25% of adolescents have had at least 1 depressive episode.2 Most adults with recurrent depression have their initial depressive episodes as teenagers,3 suggesting that adolescence is an important developmental period in which to intervene.

Research on the treatment of youth depression supports the efficacy of medication4 and psychotherapy.5-7 Emerging evidence also exists that psychosocial interventions may prevent depression.8-10 Recently, intervention research has focused on groups at high risk for affective disorder, including children and adolescents with a depressed parent, in whom depression is up to 6 times more likely to develop than in other children.11,12 Most offspring studies were noninterventional, limited to investigations of relative risk and longitudinal course.12 Recently, however, Beardslee and colleagues8 conducted a trial of a family-based, cognitive intervention for offspring of depressed parents. They found positive effects on functional outcomes of parent and child, but did not report effects for depression diagnoses.

Another frequently studied risk group includes individuals who do not meet full criteria for a DSM affective episode, but who report significant “subsyndromal” depressive symptoms. “Full-blown” depression is more likely to develop in individuals with subsyndromal symptoms,13-15 who have been the subject of several targeted prevention interventions.5-10

This report presents the results from a new prevention trial conducted with the subsyndromal adolescent offspring of parents treated for depression. This study was similar to the investigation by Beardslee et al8 in that both trials attempted to treat or prevent depression in the at-risk offspring, and both used manual-based, psycho-educational interventions. However, Beardslee et al8 used an individual,
SUBJECTS AND METHODS

SUBJECTS AND RECRUITMENT

The sampling frame included approximately 410,000 members enrolled in the Kaiser Permanente Northwest health maintenance organization (HMO), centered around Portland, Ore. The study research center is located within the HMO but is self-governed. The Human Subjects Committee for the HMO approved all study procedures.

To refresh the available pool of depressed parents and youth, case finding was conducted in 6 separate cohorts across a 2-year recruitment period (September 1994 through October 1996). Each cohort began by identifying those adults aged 30 to 65 years who had been HMO members for at least 6 months, had covered dependents aged 13 to 18 years, and lived reasonably close to the study center.

To generate the initial pool of depressed adults, we searched the HMO computerized pharmacy database for adults who had received at least 2 dispensations of an antidepressant medication within the past 12 months. Nearly all HMO member prescriptions are filled at HMO pharmacies. Across all cohorts, this step identified 4706 potentially depressed parents. We also searched the mental health appointment database, identifying 1316 adults with at least 2 mental health visits in the past 12 months. Approximately 17% of adults (n=1003) were identified using both methods, resulting in a nonoverlapping total of 5954 potentially depressed adults.

Medical chart reviews determined that 3935 of these adults (66.1%) had a depression diagnosis and/or symptoms recorded in association with the medication prescription or the mental health visits. Each parent’s physician mailed introductory letters to those adults they judged appropriate for the study (n=2995). A postpaid refusal postcard was returned by 438 adults (13.3%). Study staff attempted to telephone the remaining adults for a brief screening of study criteria. The adolescent offspring of 2083 contacted adults were also asked about their interest in participating. Interested families were invited for an intake evaluation; a written consent was signed by the parent(s) and the adolescent. Up to 2 qualifying siblings from each family could participate. Interviews were scheduled with 744 families (966 youth) and completed with 481 parents (64.7%) and 551 adolescents (57.0%). This assessment confirmed the parent diagnosis of depression and assessed adolescent psychiatric diagnoses, symptoms, and psychosocial functioning.

To qualify for the study, parents could either be in a current episode of major depression and/or dysthymia, or have had an episode in the past 12 months that touched chronologically on their current course of antidepressant medication and/or psychotherapy. The latter subjects were those who had effectively responded to their depression treatment, were no longer depressed, but were still taking medication or receiving treatment. Parent diagnosis of depression and other disorders was assessed using the Family Schedule for Affective Disorders and Schizophrenia (F-SADS), modeled after the Family History Research Diagnostic Criteria. Parents were asked a series of structured questions probing for the presence or absence of symptoms of psychiatric disorders listed in the DSM-III-R. Interviewers assigned appropriate psychiatric diagnoses according to DSM-III-R decision rules. The F-SADS demonstrated excellent interrater reliability for lifetime diagnoses in another study (Peter Lewinsohn, PhD, unpublished data, August 1994), with κ = 0.92 for unipolar depression and κ = 0.93 for nonaffective disorders.

Of 591 interviewed youth/parent dyads, 79 were disqualified, usually because the interviewed parent did not meet criteria for major depression or dysthymia (n = 70). The remaining 472 youth were classified into 1 of 3 mutually exclusive depression severity groups based on adolescent information gathered during the intake assessment.

A high-severity depression group (n = 116; 24.6%), called depressed youth, consisted of adolescents with current DSM-III-R diagnoses of major depressive disorder (MDD) and/or dysthymia. A separate randomized treatment trial was conducted with this sample but is not reported herein.

A medium-severity depression group (n = 123; 26.1%) was called subsyndromal youth, who reported some subdiagnostic levels of depressive symptoms that were insufficient to meet full criteria for a DSM-III-R affective diagnosis, and/or had a Center for Epidemiological Studies-Depression Scale (CES-D) score of greater than 24. These youth are the focus of this report.

A low-severity depression group was identified (n = 233; 49.4%), called resilient youth, with no significant depression symptoms and no history of depressive disorder. These youth were not subsequently followed up in this study.

We conducted independent, planned comparisons of baseline data, contrasting the depressed and subsyndromal youth vs the resilient youth, and the depressed vs the subsyndromal youth. Compared with the resilient youth, the depressed and subsyndromal youth were significantly older (P = .003), less likely to be enrolled in school (P = .02), and more often female (P < .001, an expected result given the greater likelihood of depression among female subjects in community samples). Compared with the subsyndromal youth, the depressed youth were older (P = .003) and had more special education classes per week (P = .05). Compared with the resilient families, the parents of depressed and subsyndromal youth had lower educational levels for the parent in the household with the highest educational attainment level (P = .05) and were less likely to be their biological parents (P = .042).

ASSESSMENTS

Unless otherwise noted, all parent and youth instruments were administered at intake (baseline), after treatment, and at follow-up assessments nominally planned for 12 and 24 months after the end of treatment (actual median completion dates were 53 and 107 weeks posttreatment). Assessors had psychology master’s degrees and were trained in the use of the diagnostic instruments and received weekly supervision from one of us (G.N.C.) for the duration of the study. Assessors were unaware of the experimental condition of interviewed subjects.

Parent Assessment

The Achenbach Child Behavior Checklist (CBCL) consists of several social competence items and 113 youth behavior and emotional problem items that parents rate as
being not true, sometimes or somewhat true, or very or often true.21,22 The CBCL has good test-retest reliability, and discriminates between clinic-referred and nonreferred children.23 Scores are reported for an extracted depression subscale created to match DSM-III-R criteria for major depression (the CBCL-D24) and the standard CBCL externalizing and internalizing symptom subscales.

Adolescent Assessment

The SADS for School-Age Children, Epidemiological Version (K-SADS-E25) was administered to adolescents26 to obtain DSM-III-R diagnoses. Interviewers completed a rigorous program in the use of the K-SADS-E and F-SADS and demonstrated a minimum interrater reliability level17 of k = 0.80 in 2 practice interviews before conducting study assessments. A random 20% of interviews were rerated by a senior diagnostic interviewer. Interrater reliability for the assessments. A random 20% of interviews were rerated by a senior diagnostic interviewer. Interrater reliability for the K-SADS-E was excellent for mood disorders (k = 0.87 for current diagnoses, k = 1.00 for past diagnoses at baseline, and k = 0.90 for diagnoses made at follow-up assessments) and good for all other nonaffective disorders (k = 0.74, k = 0.67, and k = 0.90, respectively).

The CES-D19 is a self-report measure of the frequency of 20 depressive symptoms during the past week. It has good psychometric properties when used with adults27,28 and adolescents.30,31 Parents and adolescents completed self-rated versions during separate administrations of the CES-D.

Interviewers completed a 14-item “extracted” version of the Hamilton Depression Rating Scale (HAM-D).32,33 We extrapolated HAM-D item scores from corresponding K-SADS-E depression symptom ratings. Reliability for the extracted HAM-D ranged from r = 0.87 to r = 0.94 in adults32; α = .83 for adolescents.24

Interviewers rated severity of impairment using the Global Assessment of Functioning scale (GAF). The GAF scores range from 1 to 90 and include behavioral examples that serve as anchor points. Scores below 40 reflect major impairment.

Youth in both conditions were free to continue or initiate use of any other health services. We used computerized HMO data systems to obtain data on extraexperimental health services, including, eg, inpatient and outpatient services, prescriptions, and emergency department visits. Research staff interviewed subjects monthly regarding health care services obtained from non-HMO sources.

PROCEDURES

Qualifying subsyndromal youth were randomized to conditions using a blocked procedure to ensure that group assignments were never significantly imbalanced. Group assignment was preprinted using a computer program and sealed in sequentially numbered envelopes, which were opened in sequential order by the project coordinator (M.P.). All subjects, regardless of their degree of future participation, were considered part of the study from the point of randomization (an intent-to-treat design). Of 123 qualifying and invited youth, 29 declined to participate before randomization. Of the remaining youth, 43 were randomized to the experimental condition and 49 to the usual-care control condition. The 94 randomized youth did not differ from the 29 declining youth by parent age or sex, youth sex, or youth baseline GAF, HAM-D, or CBCL-D scores. However, randomized youth had higher baseline CES-D scores (mean of 24.4 vs 19.2; P = .01) and were roughly a year younger than the nonrandomized youth (mean age of 14.6 vs 15.3 years; P = .003).

Compared with the initial pool of potential cases (N = 3374 youths), randomized subsyndromal youth were an average of 5 months older (P = .04) and were more likely to be female (60% of subjects [56/94] vs 46% of the initial pool [1558/3375]; P = .01).

Experimental Intervention

The prevention program34 was an abbreviated version of an adolescent depression treatment program35 that had been tested in a series of previous controlled outcome investigations.5,36 The intervention consisted of 15 one-hour sessions for groups of 6 to 10 adolescents, led by a therapist with a master’s degree who was trained in the approach. Sessions were conducted at the HMO clinic offices. Adolescents were taught cognitive restructuring techniques37,38 to identify and challenge irrational unrealistic or overly negative thoughts, with a special focus on beliefs related to having a depressed parent (eg, “I’m doomed to become depressed because my father is depressed, and there’s nothing I can do about it.”). The prevention program had been tested in a previous randomized trial35 in which it reduced the risk for future depressive episodes in youth with subdiagnostic depressive symptoms. Youth attended an average of 9.5 treatment sessions (median, 12; range, 0-15) and had completed homework assignments at an average of 46% of the sessions they attended.

We conducted 3 separate parent informational meetings at the beginning, middle, and end of each adolescent group. Parents were informed about the general topics discussed and skills taught in the adolescent groups and the rationale for their use. The parents’ own depression was not directly discussed in these parent meetings.

All intervention sessions were audiotaped, and 2 or 3 sessions were randomly selected from each group and rated by a senior supervisor on a 10-item fidelity scale39 to assess therapist adherence to the study protocol. Mean therapist compliance was 95.9% (SD, 3.9%; range, 90.0%-100.0%) across 15 rated sessions, indicating excellent compliance.

Usual-Care Control Condition

All youth, whether randomized to receive the experimental cognitive-behavioral treatment intervention or not, were permitted to initiate or continue any nonstudy mental health or other health care services provided by the HMO and/or outside health care providers (including antidepressant medication, of which there was very little). This usual care in the comparison group constituted the control condition. Information regarding the degree and type of usual-care treatment was collected as described above, for examination as a potential covariate of outcome.

Of the 94 randomized participants, 2 did not participate in any of the follow-up interviews. Four, 9, and 16 participants did not participate in the posttreatment and the 12- and 24-month diagnostic interviews, respectively. We found few baseline or treatment interaction

Continued on the next page
family-based intervention, provided by psychiatrists or primary care providers, and offered at a similar level of intensity to all youth regardless of depression severity level. In contrast, this study examined the effectiveness of a group cognitive-behavioral treatment program provided by therapists with master’s degrees. Furthermore, this investigation categorized offspring into 3 depression severity groups (low, medium, and high severity), and matched the intervention dose to the severity of the presenting depression.

This report presents the overall design of the study (Figure 1), and the outcome findings for the group with medium-severity depression, ie, adolescents who presented with subsyndromal depression symptoms but had no active depression episodes. The aim of the randomized trial with these youth was to prevent progression to future episodes of major depression.

CHARACTERISTICS OF THE EXPERIMENTAL GROUPS

The 2 experimental conditions did not differ at baseline on rates of current and past psychiatric disorder or on any other key demographic, depression severity, functioning, or other psychosocial measure (Table 1 and Table 2), after correction for multiple comparisons.

DEPRESSION OUTCOMES

Three continuous depression measures (the CES-D, the CBCL-D, and the HAM-D) were examined during follow-up using survival analyses and using Pearson chi analyses for cross-sectional comparisons at particular assessment points. All significance tests were 2-tailed, with an alpha of .05 (P < .05).

Table 1. Comparison of Experimental Conditions by Baseline Demographics, Psychopathology, and Psychosocial Constructs

<table>
<thead>
<tr>
<th>Youth demographics</th>
<th>Control Group (n = 47)</th>
<th>Treatment Group (n = 40)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>14.7 (1.5)</td>
<td>14.4 (1.4)</td>
<td>.23</td>
</tr>
<tr>
<td>No. (%) female</td>
<td>32 (65)</td>
<td>24 (53)</td>
<td>.24</td>
</tr>
<tr>
<td>No. (%) nonwhite</td>
<td>2 (4)</td>
<td>8 (18)</td>
<td>.03</td>
</tr>
<tr>
<td>Parent demographics</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age, y</td>
<td>40.6 (5.1)</td>
<td>42.4 (5.4)</td>
<td>.09</td>
</tr>
<tr>
<td>No. (%) female</td>
<td>38 (78)</td>
<td>37 (82)</td>
<td>.57</td>
</tr>
<tr>
<td>No. (%) nonwhite</td>
<td>1 (2)</td>
<td>4 (8.9)</td>
<td>.14</td>
</tr>
<tr>
<td>No. (%) married</td>
<td>40 (80)</td>
<td>30 (66.7)</td>
<td>.10</td>
</tr>
<tr>
<td>No. (%) college graduate</td>
<td>12 (24)</td>
<td>16 (35.6)</td>
<td>.24</td>
</tr>
<tr>
<td>No. (%) employed</td>
<td>41 (84)</td>
<td>37 (82.2)</td>
<td>.85</td>
</tr>
<tr>
<td>Psychopathology scores†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CES-D</td>
<td>23.8 (10.3)</td>
<td>25.2 (8.7)</td>
<td>.48</td>
</tr>
<tr>
<td>HAM-D</td>
<td>3.1 (3.2)</td>
<td>3.2 (3.4)</td>
<td>.96</td>
</tr>
<tr>
<td>No. of K-SADS diagnoses</td>
<td>0.5 (0.8)</td>
<td>0.4 (0.8)</td>
<td>.40</td>
</tr>
<tr>
<td>GAF</td>
<td>75.0 (8.7)</td>
<td>73.3 (9.4)</td>
<td>.36</td>
</tr>
<tr>
<td>CBCL-depression</td>
<td>6.8 (4.1)</td>
<td>6.8 (5.3)</td>
<td>.04</td>
</tr>
<tr>
<td>CBCL-internalizing</td>
<td>14.8 (8.5)</td>
<td>17.8 (10.7)</td>
<td>.14</td>
</tr>
<tr>
<td>CBCL-externalizing</td>
<td>12.4 (8.0)</td>
<td>14.4 (11.1)</td>
<td>.30</td>
</tr>
</tbody>
</table>

*Unless otherwise indicated, data are given as mean (SD).
†CES-D indicates Center for Epidemiological Studies–Depression Scale; HAM-D, Hamilton Depression Rating Scale; K-SADS, Schedule for Affective Disorders and Schizophrenia for School-Age Children; GAF, Global Assessment of Functioning; and CBCL, Achenbach Child Behavior Checklist.
Although there has been increasing interest in the role of children’s and adolescents’ depression in the overall burden of mental illness 

Table 2. Prevalence of Current and Past Psychiatric Disorder for Subsyndromal Youth at the Baseline Assessment*

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. (%) of Youth</th>
<th>Control Group (n = 49)</th>
<th>Experimental Group (n = 45)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Past</td>
<td>Current</td>
<td>Past</td>
</tr>
<tr>
<td>No diagnoses</td>
<td>9 (18.4)</td>
<td>32 (65.3)</td>
<td>9 (20.0)</td>
</tr>
<tr>
<td>Affective disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major depression</td>
<td>33 (67.3)</td>
<td>0</td>
<td>30 (66.7)</td>
</tr>
<tr>
<td>Dysthymia</td>
<td>0</td>
<td>0</td>
<td>3 (6.7)</td>
</tr>
<tr>
<td>Bipolar NOS</td>
<td>0</td>
<td>0</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Anxiety disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phobias</td>
<td>2 (4.1)</td>
<td>7 (14.3)</td>
<td>0</td>
</tr>
<tr>
<td>Anxiety without phobia, PTSD</td>
<td>0</td>
<td>6 (12.2)</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Separation anxiety</td>
<td>3 (6.1)</td>
<td>1 (2.0)</td>
<td>4 (8.9)</td>
</tr>
<tr>
<td>Panic disorder</td>
<td>2 (4.1)</td>
<td>1 (2.0)</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Disruptive behavior</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADHD</td>
<td>2 (4.1)</td>
<td>2 (4.1)</td>
<td>2 (4.4)</td>
</tr>
<tr>
<td>Conduct disorder</td>
<td>1 (2.0)</td>
<td>1 (2.0)</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Oppositional defiant</td>
<td>2 (4.1)</td>
<td>2 (4.1)</td>
<td>0</td>
</tr>
<tr>
<td>Substance abuse</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Marijuana dependence</td>
<td>1 (2.0)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Adjustment disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bereavement</td>
<td>2 (4.1)</td>
<td>2 (4.1)</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Adjustment disorder</td>
<td>4 (8.2)</td>
<td>1 (2.0)</td>
<td>0</td>
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<tr>
<td>Eating disorders</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eating disorder</td>
<td>0</td>
<td>0</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Eating disorder NOS</td>
<td>0</td>
<td>2 (4.1)</td>
<td>1 (2.2)</td>
</tr>
<tr>
<td>Psychotic disorder NOS</td>
<td>0</td>
<td>2 (4.1)</td>
<td>0</td>
</tr>
</tbody>
</table>

* NOS indicates not otherwise specified; PTSD, posttraumatic stress disorder; and ADHD, attention-deficit/hyperactivity disorder.
pressed days, between-group differences were tested using Poisson regression. The Wald $\chi^2$ for the difference was 760.62 ($P < .001$).

NONAFFECTIVE OUTCOMES

There were no significant effects for any of the nonaffective continuous mental health outcome measures, such as the CBCL internalizing and externalizing scales, and the sum of other, nonaffective diagnoses at each assessment point (Table 3).

There were no significant differences in time to the development of a new episode of a nonaffective disorder (Table 4). Although the cumulative estimated incidence of new, nonaffective episodes was higher in the control group than the intervention group at 12 (15.9% vs 4.9%, respectively) and 24 months after treatment (24.3% vs 19.5%, respectively), this difference was never statistically significant.

FUNCTIONING OUTCOMES

Results of random-effects regression analyses indicated significant program effects for the GAF ($P = .04$), although the GAF scores were not significantly different across conditions at any of the specific follow-up assessment points.

DOSE-OUTCOME EFFECTS

We examined the relationship between dose (defined here as number of intervention sessions) and each major outcome variable, restricting the sample to just the experimental youth. We were unable to detect significant dose effects for any measures.

COMMENT

We obtained significant preventive effects in this at-risk group of offspring, across continuous and diagnostic depression outcome variables, suicide symptoms, and functioning. The adjusted risk for development of depres-

Table 3. Random Effects Regression Outcome Results

<table>
<thead>
<tr>
<th>Measure, Treatment</th>
<th>Mean (SD) RER</th>
<th>Treatment by Time, Significance†</th>
<th>$P$ Value</th>
<th>$F$</th>
</tr>
</thead>
<tbody>
<tr>
<td>CES-D</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention</td>
<td>25.2 (8.7)</td>
<td>17.8 (8.7)</td>
<td>15.1 (10.0)</td>
<td>19.5 (9.8)</td>
</tr>
<tr>
<td>Usual care</td>
<td>23.8 (10.3)</td>
<td>22.5 (11.3)</td>
<td>21.5 (13.6)</td>
<td>19.9 (10.4)</td>
</tr>
<tr>
<td>HAM-D</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention</td>
<td>3.2 (3.4)</td>
<td>1.8 (2.1)</td>
<td>1.5 (2.7)</td>
<td>2.2 (2.9)</td>
</tr>
<tr>
<td>Usual care</td>
<td>3.1 (3.2)</td>
<td>2.9 (4.6)</td>
<td>2.6 (4.9)</td>
<td>2.6 (4.8)</td>
</tr>
<tr>
<td>K-SADS-E suicide symptom total</td>
<td>0.34 (1.14)</td>
<td>0.14 (0.54)</td>
<td>0.08 (0.35)</td>
<td>0.20 (0.68)</td>
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<tr>
<td>Usual care</td>
<td>0.17 (0.72)</td>
<td>0.20 (0.72)</td>
<td>0.44 (1.05)</td>
<td>0.13 (0.47)</td>
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<tr>
<td>No. other current diagnoses</td>
<td>0.4 (0.8)</td>
<td>0.2 (0.4)</td>
<td>0.1 (0.4)</td>
<td>0.4 (0.8)</td>
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<tr>
<td>Intervention</td>
<td>0.5 (0.8)</td>
<td>0.3 (0.9)</td>
<td>0.4 (0.8)</td>
<td>0.3 (0.6)</td>
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<td>Usual care</td>
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<td></td>
</tr>
<tr>
<td>CBCL-depression</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention</td>
<td>8.8 (5.3)</td>
<td>7.8 (5.5)</td>
<td>7.2 (5.7)</td>
<td>8.0 (6.5)</td>
</tr>
<tr>
<td>Usual care</td>
<td>6.8 (4.1)</td>
<td>6.4 (3.5)</td>
<td>5.4 (3.9)</td>
<td>3.9 (2.9)</td>
</tr>
<tr>
<td>CBCL-internalizing</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intervention</td>
<td>17.8 (10.7)</td>
<td>14.7 (10.3)</td>
<td>14.2 (11.4)</td>
<td>14.9 (12.4)</td>
</tr>
<tr>
<td>Usual care</td>
<td>14.8 (8.5)</td>
<td>12.9 (7.0)</td>
<td>10.5 (7.3)</td>
<td>8.7 (5.4)</td>
</tr>
<tr>
<td>CBCL-externalizing</td>
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<td></td>
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<tr>
<td>Intervention</td>
<td>14.4 (11.1)</td>
<td>12.7 (9.8)</td>
<td>10.6 (9.6)</td>
<td>12.2 (11.6)</td>
</tr>
<tr>
<td>Usual care</td>
<td>12.4 (8.0)</td>
<td>12.7 (9.6)</td>
<td>9.6 (6.5)</td>
<td>7.6 (5.6)</td>
</tr>
<tr>
<td>GAF</td>
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<tr>
<td>Intervention</td>
<td>73.3 (9.4)</td>
<td>77.8 (12.5)</td>
<td>81.6 (8.7)</td>
<td>78.8 (11.0)</td>
</tr>
<tr>
<td>Usual care</td>
<td>75.0 (8.7)</td>
<td>78.2 (9.6)</td>
<td>79.3 (12.8)</td>
<td>79.9 (9.9)</td>
</tr>
</tbody>
</table>

*†RER indicates random effects regression. Abbreviations are described in the second footnote to Table 1. n = 94.

†Indicates program effect. F indicates type III $F$ statistic for fixed effect of treatment by time.

Figure 2. Cumulative proportion of youth remaining nondepressed for experimental and control groups during the 2-year follow-up. Treatment conditions are described in the “Procedures” subsection of the “Subjects and Methods” section.
sion in the control group was more than 5 times that of
the prevention group. Preventive effects were clinically
significant‡; that is, large enough to be considered mean-
ingful in the real world. The 8.0% total annual rate of
depression in the experimental group approximated the
6.5% naturalistic total annual incidence rate of depres-
sion with a depressed parent, and the interven-
tion was provided in the schools rather than in
an HMO. However, the prevention program was essen-
tially the same in both trials.

Despite these generally positive findings, there were
several limitations of this study. By the 2-year follow-
up, the conditions were beginning to converge on many
measures, suggesting that preventive effects faded over
time. Clearly, the effects were more proximal than dis-
tal. What can be done to sustain the protective effects of
the intervention? Despite previous negative findings re-
garding continuation treatment, periodic boosters fol-
lowing acute intervention may be warranted to sustain
preventive effects.

Another limitation of this program was the multi-
stage case-finding mechanism used to identify poten-
tially depressed parents; this method would be difficult
to duplicate in many settings. However, alternatives such
as provider and family referrals could be substituted.

Another limitation was that only a small number of
subjects were enrolled out of a large initial pool of po-
tential subjects. The enrolled subjects were older and more
often female, raising generalization issues. The rela-
tively low randomization rates also raise concern about
patient interest in preventive services. However, low par-
ticipation in a research study (with its associated assess-
ment burden) is not necessarily equivalent to lack of in-
terest in prevention.

Another issue relates to the very heart of outreach
and prevention. This study systematically recruited fami-
lies that were not actively seeking care for the target youth
from the HMO. This is a different way of doing business
for a large health care system, although well-known ex-
ceptions include outreach for vaccinations, well-infant
visits, and mammograms. However, outreach and pre-
vention of this type may be particularly fruitful for health
care systems with a known, enrolled membership. Pro-
active mental health care can yield more functional and
less distressed members, and if these services cost the same
or less than the traditional approach, then health care sys-
tems are already financially and in terms of member sat-
sification.

However, there is also a cautionary side to out-
reach. Well-meaning but poorly implemented outreach
programs risk being perceived by families as overly in-
trusive or risk inviting families to participate in un-
wanted services. Mindful of this, we were cautious in our
contacts with parents and did not presume that they or
their offspring were depressed. Our telephone scripts ac-
nowledged in advance that many people receive so-
called antidepressant medication for many other legiti-
mate uses (eg, insomnia, smoking cessation, neuralgia).

Has this preventive intervention been sufficiently
tested to warrant adoption in nonresearch settings? We
believe that some additional questions remain to be an-
swered. Another important hurdle would be replication
of our findings by an independent group, preferably with a
more diverse sample than ours.45

Ideally, we would like to know something about the
dissemination of this intervention in completely natu-
ralistic settings. How important is fidelity to the inter-
vention? Must the program be delivered essentially as writ-
ten to achieve similar results?39 These and related
questions could be answered in early naturalistic dis-
semination efforts, rather than necessitating another ran-
donized trial.

**CONCLUSIONS**

Depression risk can be substantially lessened with a brief
group program, which in this study’s high-risk sample
reduced depression symptoms and episode rates to the
community-normal range. Future analyses will examin-

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**Table 4. Survival Analyses for Affective and Nonaffective Episodes During Follow-up**

<table>
<thead>
<tr>
<th>Follow-up Point, mo†</th>
<th>Affective episodes</th>
<th>Nonaffective episodes</th>
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<tbody>
<tr>
<td></td>
<td>Wilcoxon‡§</td>
<td>P Value</td>
</tr>
<tr>
<td>12</td>
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<tr>
<td>18</td>
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<tr>
<td></td>
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<td>.33</td>
</tr>
<tr>
<td></td>
<td>0.62</td>
<td>.43</td>
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</table>

<table>
<thead>
<tr>
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<th>Life-Table Analyses</th>
<th>Cox Regression Analyses</th>
<th>Hazards Ratio (95% Confidence Interval)</th>
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</tbody>
</table>

*P = 0.05.*

†Follow-up points are from the posttreatment assessment, which was an average of 2 months after the baseline interview.
‡df = 1.
§n = 92.
ine the impact of this program on use of health care resources and costs.

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