Impact of Comorbidity on a Cognitive-Behavioral Group Treatment for Adolescent Depression

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ABSTRACT

Objective: Examine hypotheses concerning the negative impact of lifetime psychiatric comorbidity on participation in, and benefit from, a cognitive-behavioral group treatment for depression in adolescents (e.g., greater severity at intake, less recovery and more recurrence, less participation in treatment). Method: Across two previous studies conducted between 1986 and 1993, 151 depressed adolescents (aged 14–18) were randomly assigned to one of three treatment conditions (two active treatments and a waitlist control) and followed for 24 months posttreatment. Forty percent of participants had one or more lifetime comorbid diagnoses at intake. Results: Comorbid anxiety disorders were associated with higher depression measure scores at intake and greater decrease in depression scores by posttreatment. Overall lifetime comorbidity was unrelated to diagnostic recovery, but lifetime substance abuse/dependence was associated with slower time to recovery. Participants with attention-deficit and disruptive behavior disorders were more likely to experience depression recurrence posttreatment. Associations between comorbidity and participation or therapy process measures were nonsignificant. Conclusions: Although some outcomes were worse for some comorbid diagnoses, the reassuring overall conclusion is that the presence of psychiatric comorbidity is generally not a contraindication for the use of structured group cognitive-behavioral interventions for depressed adolescents. J. Am. Acad. Child Adolesc. Psychiatry, 2001, 40(7):795–802. Key Words: depression, adolescents, treatment, comorbidity.

Several studies have documented the existence of efficacious treatments for adolescent depression (e.g., Brent et al., 1998; Reynolds and Coats, 1986; Stark et al., 1987). Our own program of research has shown the Adolescent Coping With Depression course (CWD-A) (Clarke et al., 1990), a group cognitive-behavioral program, to be efficacious in treating depression in older (14–18 years of age) community adolescents. In two controlled clinical trials (Clarke et al., 1999; Lewinsohn et al., 1990), active treatments were found to be superior to a waitlist control. Contrary to expectation, the two active treatments (one of which included a separate group for parents) did not significantly differ in treatment efficacy.

In the present study, we use data from these previous trials to examine the impact of psychiatric comorbidity on participation in, and benefit from, a cognitive-behavioral group treatment for depression in adolescents. Although the experimental design excluded youths with some forms of current comorbidity (e.g., conduct disorder), a substantial proportion of the participants had current or past comorbid disorders. Our general hypothesis is that comorbidity, even the presence of past psychiatric disorders, negatively affects treatment in various ways.

In childhood and adolescence, psychiatric comorbidity with depression appears to be the rule rather than the exception. In both clinic and community samples, 40% to 90% of adolescents with major depressive disorder (MDD) have other psychiatric disorders (e.g., Rohde et al., 1991). Disorders that are most frequently comorbid with MDD include dysthymia, anxiety disorders, disruptive behavior disorders, and psychoactive substance abuse/dependence (e.g., Nottelmann and Jensen, 1995).

Comorbidity between mental disorders probably affects the etiology and prognosis of each condition. Compared with adolescents and adults with a single psychiatric disorder, individuals with comorbid conditions appear to have
an earlier depression onset (e.g., Lewinsohn et al., 1994), experience more associated problems, such as suicidal behavior (e.g., Lewinsohn et al., 1995; Reich et al., 1993), have higher rates of mental health treatment utilization but poorer response to treatment (e.g., Brent et al., 1998; Emslie et al., 1998; Lewinsohn et al., 1995), and may be at increased risk for depression recurrence after recovery (e.g., Emslie et al., 1998).

The presence of comorbidity also raises questions concerning the timing and selection of treatment. For example, does it matter which disorder is treated first? Are treatments focused on depression effective for depressed patients with comorbidity—both in terms of immediate response to treatment and the long-term maintenance of treatment gains? If not, how should treatment be modified? Almost all empirically supported treatment manuals focus on a single disorder, paying little attention to comorbid conditions. However, Newman et al. (1998) suggest that comorbid cases are likely to require more broadly focused approaches to intervention. Consistent with this recommendation, the CWD-A program provides training in a number of skills (e.g., social skills, relaxation, cognitive restructuring, problem solving) that are useful in many contexts, including the treatment of other psychiatric disorders.

Objectives of the present study grew out of our concern with the above-mentioned issues. Specifically, we examine six general ways in which comorbidity could impact various aspects of our adolescent depression intervention. Our first hypothesis (H1) is that comorbidity will negatively affect the depressed adolescent by increasing the severity of his or her depression or functional impairment at the onset of treatment.

Our second hypothesis (H2) is that the presence of comorbidity will result in less, or slower, depression recovery, both by the end of acute treatment and within the 24-month follow-up period. In previous research involving one of our two clinical trials (Clarke et al., 1992), the presence of nonaffective comorbid disorders was not associated with diagnostic recovery or reduction in Beck Depression Inventory (BDI) (Beck et al., 1961) scores from intake to posttreatment, although the total number of past diagnoses (affective and nonaffective) was significantly correlated with less reduction in BDI scores from intake to posttreatment.

In addition to a predicted association between comorbidity and lower depression recovery (i.e., a main effect), the presence of comorbidity may also increase the magnitude of differences between treatment conditions (i.e., an interaction between treatment conditions and comorbidity). Brent et al. (1998) found that individual cognitive-behavioral therapy was superior to both systemic-behavioral family therapy and nondirective supportive therapy among depressed adolescents with comorbid anxiety disorder; among depressed adolescent patients with no comorbid anxiety, treatment differences were minor. Thus, our third hypothesis (H3) is that the “adolescent plus parent” condition will be superior to the “adolescent only” condition in the comorbid sample. Previously, we had been unable to show that the two active conditions differed, although we predicted that the addition of parental involvement would result in greater response.

Our fourth hypothesis (H4) is that depressed adolescents with comorbidity will show less participation in treatment, less consistent attendance, lower ratings on therapy homework completion, and lower levels of cohesiveness with the treatment group. Poor participation in treatment might act as a mediator for any negative treatment outcome effects associated with comorbidity.

Fifth, we predict (H5) that even if the participant recovers from her or his depression, a lifetime history of comorbidity will be associated with higher rates of depression recurrence within the 24-month follow-up period.

Ideally, we would evaluate whether depression-focused treatment significantly reduced the onset of new comorbid disorders. However, given that all of our participants were eventually treated, we are unable to address this issue directly. Instead, we examine two related issues in our sixth hypothesis. First, adolescents with comorbidity will show greater problems in daily functioning during follow-up (H6a). Second, among depressed youths with comorbidity, those who fail to recover from their index depression will be more likely to experience continuing or new nonaffective disorders than will comorbid adolescents who recover from their depression during treatment (H6b). Consistent with this second prediction, Hasin et al. (1996) found that recovery of one disorder in adult patients with comorbid depression and alcoholism was associated with recovery in the other condition.

Different categories of psychiatric comorbidity may have different effects. For example, in the treatment of adolescent substance abusers (Kaminer et al., 1992), the presence of comorbid mood and adjustment disorders were associated with higher rates of treatment completion, whereas the presence of comorbid conduct disorders was associated with lower rates of treatment completion.
Therefore, in addition to an aggregate measure of any comorbidity (labeled TOTAL), three specific categories of comorbid diagnoses are examined: anxiety disorders (ANX), attention-deficit and disruptive behavior disorders (DIS), and substance use disorders (SUB).

**METHOD**

**Participants and Procedures**

Our sample consisted of 151 adolescent (aged 14–18), who were originally recruited and treated in two controlled clinical trials. In the first clinical trial (Lewinsohn et al., 1990), which occurred between 1986 and 1988, 55 adolescents who met DSM-III criteria for major depressive disorder (MDD) or dysthymia were randomly assigned to treatment condition and completed posttreatment assessment. Participants and their parents completed extensive diagnostic and psychosocial measures at intake, posttreatment, and at 1, 6, 12, and 24 months posttreatment. In the second clinical trial (Clarke et al., 1999), which occurred between 1988 and 1993, 96 adolescents who met DSM-III-R criteria for MDD or dysthymia were randomly assigned to treatment condition and completed the posttreatment assessment. The goals of the second trial were very similar to those of the first clinical trial, with one modification: at the end of treatment, participants were randomly assigned to one of three 24-month follow-up conditions (follow-up condition was unrelated to treatment maintenance and is not discussed further in this paper).

Participants in both studies were recruited at two sites (Eugene and Portland, Oregon) by means of announcements to health and school professionals, media stories, and advertisements. Adolescents were excluded if they: (1) exhibited current mania/hypomania, panic disorder, generalized anxiety disorder, conduct disorder, psychotic abuse/dependence, (2) lifetime organic brain syndrome, mental retardation, or schizophrenia; (3) were currently receiving other treatment for depression and were unwilling to discontinue; or (4) needed immediate acute treatment.

In both studies, eligible participants were randomly assigned to one of three conditions: (1) a 16-session cognitive-behavioral group for adolescents only (condition A, n = 56); (2) an identical group for adolescents supplemented with a separate, 9-session parent group (condition A+P, n = 51); or (3) waitlist control (condition WL, n = 44). A total of 185 adolescents were randomly assigned to treatment condition, of whom 151 (81.6%) completed treatment condition and posttreatment assessment. After the 8-week waitlist assessment, adolescents in the WL condition were offered nonexperimental treatment for depression and were unwilling to discontinue; or (4) needed immediate acute treatment.

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**Assessment**

**Diagnostic Interviews.** Adolescents were interviewed at intake with the Schedule for Affective Disorders and Schizophrenia for School-Aged Children-Epidemiological Version (Orvaschel et al., 1982), which was expanded to obtain DSM diagnoses. At all follow-up assessments, participants were interviewed in regard to psychiatric symptoms and disorders since the previous interview with the Longitudinal Interval Follow-up Evaluation Keller et al., 1987). As part of all diagnostic assessments in the second clinical trial, interviewers rated current level of functioning with the DSM-III-R Global Assessment of Functioning (GAF) scale.

Diagnostic interviewers had bachelor’s or master’s degrees in psychology or social work or related fields, completed extensive training, and were regularly supervised. Diagnostic interviewers demonstrated good-to-excellent reliability (see Clarke et al., 1999; Lewinsohn et al., 1990).

**Adolescent-Report Measures.** Adolescents completed the BDI, a 21-item self-report measure of depressive symptomatology (α = .84), at each assessment.

**In-Session Process Measures.** Group therapists recorded attendance and degree of homework completion for participants at each session. At the third, eighth, and final group sessions, youths completed a group cohesion questionnaire, rating feelings of trust, affiliation, and association with the other group members and with the therapist.

**Treatment Interventions**

Mixed-gender groups of up to 10 adolescents were treated in 16 two-hour sessions over 8 weeks with the CWD-A (Clarke et al., 1990, described in detail by Lewinsohn et al., 1996; available for free download from the Internet at http://www.kpchr.org/acwd/acwd.html). Skills taught included mood monitoring, improving social skills, increasing pleasant activities, decreasing anxiety, reducing depressive cognitions, improving communication, conflict resolution, maintenance and development of a relapse prevention plan. Participants received workbooks that provided brief readings, short quizzes, and homework assignments.

**Parent Group.** A parallel but separate course for the parents of depressed adolescents (Lewinsohn et al., 1991) was used in the A+P condition. Parents reviewed the adolescent course content and learned the same communications and problem-solving skills. Parents met with a separate therapist once a week for eight 2-hour sessions. Two sessions were held with the adolescent group, during which each family practiced conflict reduction skills on salient issues.

**Therapists.** Therapists were either advanced graduate psychology students, social work students, or masters/Ph.D. clinicians. Therapists were provided with 40 hours of specialized training in the delivery of the treatments and attended weekly supervision meetings. Therapist adherence to the protocol was uniformly high (summarized in Clarke, 1998).

**Sample for the Present Study**

Of the 151 adolescents in the present study, 67.5% were female. Mean age of participants at intake was 16.2 years (SD = 1.2); 43.7% came from two-parent families, and 34.4% had one or more parents with a bachelors-level education or higher. Almost all of the participants (96.0%) were white.

All participants met criteria for current depression at intake: 58.9% had MDD, 23.8% had dysthymia or intermittent depression (but not MDD), and 17.2% had MDD superimposed on a more chronic depression (“double depression”). In addition to the depression diagnosis, 39.7% of the participants had one or more lifetime comorbid diagnoses at intake: 21.2% with ANX (most often specific phobia [7.9%], social phobia [6.6%], or obsessive disorder [6.0%]), 19.9% with DIS (9.9% attention-deficit/hyperactivity disorder [ADHD], 8.6% conduct disorder, and 5.3% oppositional defiant disorder), and 11.3% with SUB (8.6% had drug use disorder, 5.3% had alcohol use disorder). Among the 60 participants with lifetime comorbidity, 48 (76.7%) had diagnoses in only one comorbid category, 9 (15.0%) had comorbid diagnoses in two categories, and 5 (8.3%) had diagnoses in all three comorbidity categories. Lifetime comorbidity was unrelated to treatment condition assignment and demographic variables.

**Statistical Analyses**

Differences in depression severity (BDI) and global functioning (GAF) at intake as a function of various lifetime comorbidity groups
were evaluated with t tests. Intake depression diagnosis and attrition analyses as a function of comorbidity were evaluated with contingency table analyses. Treatment process measures were examined with point-biserial correlations.

Response to treatment was evaluated by using both BDI scores and diagnostic recovery at posttreatment. Analysis of covariance (ANCOVA) with intake BDI scores as the covariate was used to replicate the previous comparison of the two active treatments (A and A+P) to the control condition (WL). However, because participants had not been randomly assigned to treatment condition on the basis of lifetime comorbidity status, ANCOVA was not an appropriate test for exploring the moderating effects of comorbidity on treatment response (Keppel and Zedeck, 1989). Instead, BDI difference scores were analyzed with four two-way analysis of variance (ANOVA) models involving TOTAL lifetime comorbidity and the three constituent comorbid diagnoses. In addition to the continuous BDI measure of depression, categorical depression diagnoses were used to assess diagnostic recovery by posttreatment. Two direct logistic regression analyses were run where treatment and lifetime comorbidity status (TOTAL in the first model and ANX, DIS, and SUB in the second model) were entered into the model simultaneously in the first block, followed by the two-way interaction term in the second block.

Recovery and recurrence analyses included the percent who recovered and experienced recurrence posttreatment (evaluated by χ²) and Cox proportional hazards regression to examine (1) time to recovery, and (2) given recovery within 6 months of treatment completion, time to recurrence as a function of lifetime ANX, DIS, SUB, intake MDD, and dysthymia, and sex.

Wherever possible, effect size estimates as per Cohen’s d statistic, odds ratios (ORs) or hazard ratios with 95% confidence intervals (CIs) are provided, in addition to the significance levels. Potential interactions with sex were examined throughout.

RESULTS

H1: Severity at Intake

BDI scores at intake were examined as a function of TOTAL lifetime comorbidity. As shown in Table 1, adolescents with any comorbidity had significantly higher BDI average intake scores compared with adolescents with no history of comorbidity.

BDI intake scores for the three constituent comorbidity diagnoses were also compared. Statistically significant intake BDI differences were found for the participants with ANX versus those with no ANX (BDI mean [SD] = 29.8 [9.7] versus 23.2 [10.0], respectively; \( t_{149} = -3.34, p < .001 \), Cohen’s \( d = 0.66 \)). Differences as a function of the presence of comorbid DIS and comorbid SUB were in the same direction but were statistically nonsignificant. The presence or absence of lifetime comorbidity was not differentially associated with the type of depression diagnosis at intake.

As shown in Table 1, GAF scores at intake likewise indicated significantly poorer functioning for depressed adolescents with comorbidity. (We did not examine specific comorbid conditions because GAF scores were available in only the second data set.)

H2 and H3: Depression Recovery and Differential Treatment Effects

BDI Scores as a Function of Treatment Condition and Comorbidity. With intake BDI scores as a covariate, adjusted group means were analyzed to compare the A and A+P conditions to the WL control group. As expected given our past research, we replicated the significant treatment effects (\( F_{1, 144} = 22.77, p < .001 \), Cohen’s \( d = 0.86 \)), with adjusted mean (SD) BDI posttreatment scores of 10.33 (8.94) in the A condition, 10.57 (8.94) in the A+P condition, and 18.12 (8.93) in the WL condition.

Our focus in the present study was on the main effects of comorbidity on treatment response (i.e., does the presence of comorbidity result in less recovery?) and the interactions between treatment condition and comorbidity, which would indicate that differences between the three treatment conditions varied as a function of comorbidity. We addressed these issues by calculating two-way ANOVAs using BDI difference scores. As shown in Table 1, contrary to H2, TOTAL lifetime comorbidity had a significant main effect, with greater BDI change from intake to posttreatment among participants with a history of comorbidity versus those with “pure” (noncomorbid) depression. The interaction of treatment condition (A versus A+P) × TOTAL lifetime comorbidity (H3) was nonsignificant.

Pre-post BDI change scores were evaluated for the specific comorbid diagnoses. The main effect for ANX was significant (\( F_{1, 142} = 4.64, p < .05 \)), with significantly greater pre-post change on BDI for adolescents with a history of ANX (mean [SD] change = 14.9 [9.9] points) compared with adolescents with no history of ANX (mean [SD] change = 10.5 [12.5] points; Cohen’s \( d = 0.35 \)). Main effects for DIS and SUB in predicting BDI difference scores were nonsignificant, as were all treatment condition × specific comorbidity interactions.

Diagnostic Recovery by Posttreatment. In addition to the continuous BDI measure of depression, categorical DSM depression diagnoses were used to assess diagnostic recovery by posttreatment. Two hierarchical logistic regression analyses were computed, with treatment (active versus waitlist; A versus A+P) and lifetime comorbidity status (TOTAL in the first model and ANX, DIS, and SUB in the second model) entered into the model simultaneously, followed by the two-way interaction term. The main effect for active treatment versus waitlist was significant in terms
of recovery ($\chi^2_1 = 9.14, p < .01$), which resulted in an adjusted OR = 3.07 (95% CI = 1.46–6.44); support for $H_1$ (A versus AP) was nonsignificant. In both models, the main effect for comorbidity and the group $\times$ comorbidity interactions were nonsignificant. The percentage of patients who recovered in the various treatment conditions is shown in Table 1.

**Percentage Recovered During the Follow-up Period.** In addition to recovering by the end of treatment, participants were compared on whether they achieved recovery from their intake depression disorder within the 24-month follow-up period. Differences in percentage recovered as a function of TOTAL lifetime comorbidity were nonsignificant, as were differences in recovery as a function of each specific comorbidity diagnoses.

**Time to Recovery.** In a Cox proportional hazards regression containing the three specific lifetime comorbid diagnoses and sex, SUB was associated with significantly longer time to depression recovery (Wald test $[1] = 6.04, p < .05$); magnitude of the effect is indicated by a hazard ratio = 2.53 (95% CI = 1.18–5.43). Median time to recovery from intake for participants with SUB was 72.2 weeks, compared with a median time to recovery of 10.0 weeks for participants with no lifetime history of SUB. Other variables examined in the Cox proportional hazards model were unrelated to time to recovery.

### TABLE 1

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Lifetime Comorbidity at Intake</th>
<th>Test Statistic</th>
<th>$p$</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes (SD)</td>
<td>No (SD)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression severity at intake</td>
<td>26.8 (10.3)</td>
<td>23.0 (10.0)</td>
<td>$t_{48} = -2.27$</td>
<td>.025</td>
</tr>
<tr>
<td>BDI score, mean (SD)</td>
<td>$X^2(N = 151) = 2.92$</td>
<td>.232</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Depression diagnosis</td>
<td>% MDD only</td>
<td>63.3</td>
<td>56.0</td>
<td></td>
</tr>
<tr>
<td>% Dysthymia only</td>
<td>16.7</td>
<td>28.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% Double depression</td>
<td>20.0</td>
<td>15.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GAF score, mean (SD)</td>
<td>54.4 (7.4)</td>
<td>60.0 (7.4)</td>
<td>$t_{67} = 3.66$</td>
<td>.000</td>
</tr>
<tr>
<td>Response to treatment at post assessment</td>
<td>13.7 (10.9)</td>
<td>10.4 (10.2)</td>
<td>$F_{1,44} = 3.97$</td>
<td>.048</td>
</tr>
<tr>
<td>BDI change from intake to post, mean (SD)</td>
<td>16.1 (10.9)</td>
<td>13.0 (11.0)</td>
<td>$F_{2,142} = 0.13$</td>
<td>.885</td>
</tr>
<tr>
<td>As a function of treatment condition$^a$</td>
<td>16.4 (11.4)</td>
<td>11.9 (9.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WL</td>
<td>7.7 (8.3)</td>
<td>5.3 (8.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent recovered at posttreatment</td>
<td>51.7</td>
<td>50.5</td>
<td>$Wald (1) = .002$</td>
<td>.963</td>
</tr>
<tr>
<td>% of all subjects</td>
<td>50.0</td>
<td>61.1</td>
<td>$Wald (2) = 2.06$</td>
<td>.358</td>
</tr>
<tr>
<td>% in A</td>
<td>69.6</td>
<td>53.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% in WL</td>
<td>29.4</td>
<td>33.3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percent recovered during FU</td>
<td>83.3</td>
<td>93.2</td>
<td>$X^2(N = 95) = 2.32$</td>
<td>.128</td>
</tr>
<tr>
<td>Participation at various FU assessments</td>
<td>83.3</td>
<td>80.5</td>
<td>$X^2(N = 185) = 0.23$</td>
<td>.631</td>
</tr>
<tr>
<td>% at post</td>
<td>62.5</td>
<td>54.0</td>
<td>$X^2(N = 185) = 1.30$</td>
<td>.253</td>
</tr>
<tr>
<td>% at 12-month</td>
<td>52.8</td>
<td>53.1</td>
<td>$X^2(N = 185) = 0.01$</td>
<td>.966</td>
</tr>
<tr>
<td>% at 24-month</td>
<td>25.0</td>
<td>12.5</td>
<td>$X^2(N = 64) = 1.65$</td>
<td>.199</td>
</tr>
<tr>
<td>Depression recurrence</td>
<td>20.6</td>
<td>12.5</td>
<td>$X^2(N = 64) = 1.65$</td>
<td>.199</td>
</tr>
<tr>
<td>Global functioning, mean (SD)</td>
<td>67.7 (12.8)</td>
<td>69.6 (13.3)</td>
<td>$t_{55} = 0.72$</td>
<td>.475</td>
</tr>
<tr>
<td>GAF at posttreatment</td>
<td>75.5 (9.5)</td>
<td>77.2 (10.9)</td>
<td>$t_{68} = 0.70$</td>
<td>.484</td>
</tr>
<tr>
<td>GAF at 12-month FU</td>
<td>74.7 (11.4)</td>
<td>80.9 (8.6)</td>
<td>$t_{66} = 2.56$</td>
<td>.013</td>
</tr>
</tbody>
</table>

**Note:** BDI = Beck Depression Inventory; MDD = major depressive disorder; GAF = Global Assessment of Functioning; OR = odds ratio (including 95% confidence interval); FU = follow-up; NA = not applicable.

$^a$ A = 16-session cognitive-behavioral group for adolescents only; A+P = an identical group for adolescents supplemented with a separate, 9-session parent group; WL = waitlist control.
**H4: Participation**

Although not an indicator of treatment response, it was important to examine whether participants with comorbidity were less likely to continue participation in the study. Significantly greater attrition for adolescents with comorbidity could be indicative of participation problems, in addition to potentially biasing other results. As shown in Table 1, differences in completing the various follow-up assessments as a function of TOTAL lifetime comorbidity were nonsignificant at all three assessment points. Attrition analyses as a function of ANX, SUB, and DIS were similarly nonsignificant.

All correlations between comorbidity (TOTAL, ANX, DIS, SUB) and the in-session process indicators (i.e., number of sessions attended, average homework completed, group cohesion scores at sessions 3, 8, and 16) were nonsignificant. The only correlation that was significant at the level of a trend suggested that lifetime SUB was associated with lower ratings of group cohesiveness early in treatment (i.e., session 3) ($r = -0.19$, $p = .10$).

**H5: Recurrence**

*Percentage Experiencing Depression Recurrence.* Recurrence analyses were restricted to participants who recovered within 6 months of treatment completion and had follow-up data at 24 months ($n = 64$). As shown in Table 1, although they were in the predicted direction (i.e., more depression recurrence associated with comorbidity), differences in depression recurrence for participants with TOTAL lifetime comorbidity versus those with no previous comorbidity were nonsignificant. Associations of depression recurrence with ANX and SUB were also nonsignificant. A trend was present for lifetime DIS at intake to predict higher likelihood of depression recurrence in the follow-up period (36.4% of participants with DIS at intake experienced depression recurrence versus 13.2% of participants with no history of DIS; $\chi^2_1 [N = 64] = 3.43$, $p = .064$; OR $= 3.76$ [95% CI = 0.87–16.22]).

*Time to Depression Recurrence.* Lifetime history of DIS at intake was predictive of shorter time to depression recurrence posttreatment (Wald test $[1] = 5.44$, $p < .01$; hazard ratio $= 4.63$ [95% CI = 1.28–16.74]). Median time to depression recurrence could not be reliably calculated because too few of the adolescents experienced recurrence. Results for ANX and SUB were nonsignificant.

**H6: Impact of Comorbidity on Indices Other Than Depression**

* Differences in Global Functioning.* GAF scores at posttreatment and at 12 months posttreatment for those with versus those without comorbidity were nonsignificant. However, as shown in Table 1, by 24 months posttreatment, depressed adolescent with psychiatric comorbidity at intake were scoring significantly poorer on the measure of global functioning.

**Psychopathology as a Function of Depression Recovery.** In the last analysis, we examined whether comorbid depressed adolescents who recovered from their depression were less likely to experience continued or new nonaffective disorders, compared with comorbid depressed adolescents who do not recover from their depression. Analyses were restricted to the 37 patients with current comorbid disorders at intake (and 24-month follow-up data). Twenty-four of these adolescents recovered by posttreatment; 12 (50.0%) had some form of nonaffective psychopathology in the follow-up period. The remaining 13 adolescents had not recovered from their depression by posttreatment; 9 (69.2%) had nonaffective psychopathology in the follow-up period. Although it was in the predicted direction, the difference was nonsignificant ($\chi^2_1 [N = 37] = 1.27$, $p = .26$; OR $= 2.25$ [95% CI = 0.54–9.34]).

**DISCUSSION**

Which treatments work, for which patients, with which characteristics? This often-posed question is relevant to the present investigation. Given the prevalence of psychiatric comorbidity among adolescents with mood disorders, teasing apart the relative benefits of depression-specific treatments for youths with and without comorbidity is particularly important. Although the general issues of psychiatric comorbidity has received a great deal of research attention, its specific impacts on clinical interventions has been much less frequently examined. To our knowledge, the present study is the most detailed examination of the impact of comorbidity on adolescent depression treatment to date.

Our primary question in this study was whether our structured group cognitive-behavioral intervention was less effective for depressed youths with comorbid conditions. Although some outcomes were worse for some comorbid conditions, the reassuring overall answer to these questions seems to be negative. Depressed adolescents with comorbidity (especially anxiety disorders) enter treatment more severely depressed and with more functional impairment. However, by the end of treatment, recovery rates were comparable for all adolescents. If anything, comorbid anxiety disorder was associated with...
better outcome, as measured by reductions in depression scores during treatment. This finding is probably due to the anxious depressed patients reporting more depression symptoms at intake. In regard to therapy process, there was no indication of greater attrition or less positive participation in treatment as a function of comorbidity. Within the limits of our experimental design, the presence of psychiatric comorbidity was not a contraindication for use of our group cognitive-behavioral therapy program, as evaluated by its effectiveness during the course of treatment.

Recovery continues after treatment ends. Broadening our examination to the 24-month follow-up period, one category of comorbid disorders—substance abuse and dependence—was associated with slower time to recovery. One positive aspect of the findings concerning recovery is that depressed adolescents with comorbid substance abuse/dependence (and other psychopathologies) eventually achieved comparable rates of improvement; recovery just took longer.

Among the adolescents who recovered within 6 months of treatment, patients with a lifetime history of comorbidity were not statistically more likely to experience recurrence. It should be noted, however, that the percentage of comorbid patients who experienced recurrence was double the rate for noncomorbid patients; given a larger sample, this effect may well have been significant. Among the specific comorbidity categories, participants with conduct disorder, oppositional defiant disorder, and ADHD were more likely to relapse within the 24-month follow-up period. Again, these findings need to be independently replicated. However, given the often chronic nature of attention-deficit and disruptive behavior disorders, these conditions might have been triggering factors in the depression recurrence.

Our third hypothesis was that the presence of comorbidity would magnify treatment group differences. The percentage of patients recovered as a function of group and comorbidity was in the predicted direction (i.e., larger differences between A versus A+P active conditions given comorbidity), but the treatment condition × comorbidity interaction did not attain statistical significance. Although we still believe that parental involvement has beneficial therapeutic effects in specific cases, the findings suggest that both treatments are comparably superior to waitlist for both the comorbid and noncomorbid depressed adolescent.

The second major question is, were the comorbid patients seen as needing help with their other conditions even after the depressed had remitted? We had predicted that recovery from depression would be associated with less comorbidity posttreatment; results were in the predicted direction but were statistically nonsignificant. GAF scores by the end of follow-up, however, indicated that depressed patients with initial comorbidity were functioning more poorly overall compared with their noncomorbid depressed peers. If the comorbid conditions are unaffected by a specific treatment, it suggests that supplemental treatment may be warranted.

In addition to the aggregate category of comorbidity, three specific diagnostic categories of comorbidity were examined. The presence of comorbid anxiety disorders had its impact prior to and during acute treatment, resulting in greater depression severity at intake. Anxiety disorders had no subsequent impact on course of recovery or recurrence. Conversely, the negative impact of substance abuse/dependence and attention-deficit/disruptive behavior disorders did not emerge until treatment was completed. Given that these comorbidity categories were unrelated to in-session process variables, it appear that their effects on the course of depression was not specifically related to treatment.

Limitations

Probably the most important limitation of the present study is that certain current psychiatric comorbid conditions were exclusion criteria for the research protocol. The exclusion of depressed adolescents with severe current comorbid disorders may have decreased our ability to detect the moderating effects of comorbidity. Thus, the present study is a conservative test of the role of comorbidity on treatment, and we are unable to estimate the complete impact of current comorbidity on treatment response. Furthermore, significant moderator effects are difficult to detect statistically, and this difficulty is compounded in studies that do not adequately represent all levels of the moderator (McClelland and Judd, 1993). We may not have had the range of comorbid severities to generate results that generalize to general clinical practice. The present results are probably most relevant to clinics that represent themselves as providing focused treatment for depression, as we did.

A second limitation is the fact that our participants were actively recruited and could not be receiving con-
current treatment for depression. These factors may have skewed our sample to a less severely depressed population, compared with a representative clinical sample. Third, the available in-session process measures were not fine-grained, and we may have been unable to detect ways in which comorbidity negatively influenced the treatment process. Lastly, the majority of results were nonsignificant, and, given the exploratory nature of this report, we did not control for experiment-wide type I error. In addition, when comorbidity had a significant effect, the magnitude of the effect was generally small to medium in size. Because of limitations, results should be regarded as tentative pending cross validation.

Clinical Implications

Findings in the present study lead to several assessment and treatment recommendations. First, clinicians need to routinely assess both current and past comorbidity with the depressed adolescent patient. The anxious-depressed adolescent may enter treatment more severely depressed. Although we did not examine this issue, it seems reasonable to suspect that suicidal ideation may be quite high in this group, resulting in a need for frequent monitoring.

In regard to response to treatment, the lifetime presence of nonaffective psychopathology appears to affect treatment response in two ways. First, depressed youths with a history of substance use disorders may recover more slowly. Second, treatment gains of the depressed externalizing adolescent may be more tenuous. Both of these findings suggest that maintenance sessions or follow-up monitoring coupled with booster sessions may be beneficial for these subsets of depressed adolescents.

Given the frequency of comorbidity, clinicians have long treated comorbid depressed adolescents by relying on clinical judgment rather than decisions that are empirically derived. Psychotherapies need to be tested to understand which treatment interventions address which disorders in comorbid adolescents. This line of research is necessary because treatments found to be efficacious for specific disorders need to be evaluated, and perhaps modified, for use when comorbid disorders are present. The present study provides additional evidence for the treatment efficacy of a group cognitive-behavioral depression treatment and suggests that it may be beneficial to a broad range of depressed youths, including those with some forms of psychiatric comorbidity.

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