A Randomized Controlled Trial of Cognitive Therapy for Bipolar Disorder: Focus on Long-Term Change

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Background: This study reports the outcome of a randomized controlled trial of cognitive therapy (CT) for bipolar disorder. The treatment protocol differed from other published forms of CT for bipolar disorder through the addition of emotive techniques.

Method: Fifty-two patients with DSM-IV bipolar I or II disorder were randomly allocated to a 6-month trial of either CT or treatment as usual, with both treatment groups also receiving mood stabilizers. Outcome measures included relapse rates, dysfunctional attitudes, psychosocial functioning, hopelessness, self-control, and medication adherence. Patients were assessed during treatment by independent raters blind to the patients' group status.

Results: At posttreatment, patients allocated to CT had experienced less severe depression scores (Beck Depression Inventory and Montgomery-Asberg Depression Rating Scale) and less dysfunctional attitudes. After controlling for the presence of major depressive episode at baseline, there was a statistical trend toward a greater time to depressive relapse ($p = .06$) for the CT group. At 12-month follow-up, the CT group showed a trend toward lower Young Mania Rating Scale scores and improved behavioral self-control. The Clinical Global Impressions-Improvement scale, comparing the 18 months prior to treatment to the severity of illness status at follow-up, showed a substantial difference between groups in favor of CT.

Conclusion: Our findings corroborate previous bipolar disorder research in demonstrating the value of CT, particularly immediately posttreatment, and indicate some continuation (albeit diminishing) of benefits in the succeeding 12 months. These findings suggest that psychological booster sessions may be crucial for maintaining the beneficial effects of cognitive therapy.

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The main challenges for psychological treatments for bipolar disorder are preventing relapse, modifying dysfunctional attitudes, and improving psychosocial functioning. Despite considerable advances in pharmacotherapy,[1] about 40% of patients with bipolar disorder are reported to relapse within 1 year, 60% over 2 years, and 73% over 5 years.[2] Repeated hospitalizations and recurrent episodes are highly disruptive to the patients' functioning in everyday life, with such poor psychosocial capacity itself becoming a vulnerability factor for more frequent relapses.[2] Issues associated with loss of relationships, career, status, and identity are well documented,[3,4] with 30% to 60% of individuals with bipolar disorder failing to regain full functioning in the occupational and social domains.[5] Such poor functional recovery underlies the ranking of bipolar disorder as the sixth most disabling condition globally.[6]

Several decades ago, cognitive therapy (CT) emerged as a useful tool for helping patients with unipolar depression develop skills for moderating their subjective responses to real and perceived stresses.[7] Extending this ap-

proach to bipolar disorder, a number of randomized controlled trials evaluating individual CT have been published.[3,8–13] The clearest evidence is for the impact of individual CT on symptoms, social functioning, and risk of relapse during active treatment,[14] although long-term maintenance of gains has been found to be less robust.[13] Cognitive vulnerabilities among patients with bipolar disorder have shown similarities to those seen in patients with recurrent unipolar depression.[15] Dysfunctional attitudes associated with sociotrophy and autonomy[15] and extreme goal striving[16] are particularly prevalent, leading to the proposal that the interaction between these beliefs and the illness itself predisposes patients with bipolar disorder to a more severe course of illness.[16] In published CT trials to date, long-term modification of dysfunctional attitudes has proven difficult.[13]

An earlier article by the present authors[17] outlined a biopsychosocial model of chronic illness behavior highlighting the importance of cognitive styles and attitudinal change in treating patients with bipolar disorder. Clinical research associated with other chronic and relapsing illnesses such as cancer[18] has examined the interaction of illness and non-illness beliefs in determining how well patients adapt socially and psychologically. This cognitive model of dysfunctional illness behavior incorporates self-schema about competence, vulnerability, and the meaning individuals assign to their particular illness. Newman et al.[19] were among the first group of researchers to postulate the role of maladaptive schemas in relation to the functioning of patients with bipolar disorder. They proposed that maladaptive schemas that originate in childhood may be reactivated in response to the bipolar illness or the condition itself may encourage maladaptive schema, especially during adolescence.

Recent developments in clinical research have highlighted the importance of emotive techniques in accessing schemas and maladaptive beliefs and constructing new and more adaptive emotional and behavioral responses.[20] Many of the techniques are drawn from Gestalt therapy and involve accessing emotion and triggering dysfunctional attitudes during the session in order to effect change. Traditional CT has produced limited success with more complicated presentations, particularly where rigid belief systems, self-defeating behaviors, and avoidant coping styles are prominent.[21] This observation has led to emotive techniques becoming an integral part of CT, providing a valuable addition to the therapeutic arsenal.[22,23]

Emotive techniques are associated with experiential learning that occurs in therapy when experiences are felt in vivo or in memory. Such techniques have been used effectively in the treatment of unipolar depression[20] and in combination with CT for personality disorders.[21–23] Specifically, such techniques include imagery, narratives, and reliving earlier experiences.[21] Basically, the process involves 3 key elements: (1) acknowledging and validating the patient's initial experience; (2) activating the emotional memories and any associated dysfunctional beliefs by arousing, for example, fear and shame in reaction to an imagined scene, in the safety of the therapeutic situation; and (3) activating healthy emotional resources in the patient, such as anger at violation and sadness at loss or self-soothing as alternative responses to replace the person's maladaptive emotional, social, or behavioral responses.[24] As these new experiences are repeatedly processed, the traumatic memory or sense of loss is considered to fade over time. Toward the end of the process, the therapist facilitates the development of a new meaning structure that incorporates modified emotions and attitudes. This new belief system is then considered to help the patient engage in more adaptive psychosocial behaviors.[24] Since emotive techniques are potentially arousing of affect, it is considered critical that their use be restricted to times of stable mood.

The present study evaluated a CT program modified for patients with bipolar disorder by the addition of emotive techniques. This new treatment approach was specifically designed to address the cognitive, social, and behavioral adjustments considered necessary for managing a chronic mental illness and facilitating long-term benefits of cognitive therapy. The theoretical assumption of our treatment was that relevant maladaptive schemas may result from either childhood traumatic experiences or the damaging impact of the bipolar disorder itself.

The primary hypotheses were that at posttreatment and follow-up (1) the CT group would experience fewer depressive and manic/hypomanic relapses and symptoms, fewer days unwell, and fewer hospitalizations than the treatment as usual (TAU) group; (2) the CT group would show less dysfunctional attitudes; and (3) the CT group would demonstrate more improved psychosocial functioning. The secondary hypotheses were that, compared with TAU, (1) the CT group would have improved self-control, (2) the CT group would show less hopelessness, and (3) the CT group would show better medication adherence.

METHOD

Sample

Patients were recruited through referrals from general practitioners, psychiatrists, and self-help organizations, and all provided written informed consent. After screening, patients were independently assessed by experienced psychiatrists and, after giving written informed consent, were included in the study if they had a lifetime DSM-IV diagnosis of bipolar I or II disorder; were aged over 18 years (there was no specified upper age limit); had experienced at least 1 episode of hypomania, mania, or depression over the prior 18 months; and were able to be maintained on their usual mood stabilizing medication for the duration of treatment. Ethical approval was

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granted by the University of New South Wales Human Research Ethics Committee. Patients were eligible for inclusion if they were euthymic, mildly depressed, or hypomanic at the time of initial assessment. Subjects were excluded if they had a Beck Depression Inventory (BDI)[25] score ≥ 30, a 17-item Hamilton Rating Scale for Depression (HAM-D-17)[26] score ≥ 15, or a Young Mania Rating Scale (YMRS)[27] score ≥ 20. Other exclusion criteria were significant suicidal ideation, schizophrenia, schizoaffective disorder, antisocial or borderline personality disorder, bipolar disorder secondary to an organic disorder, or a significant current medical condition. Patients were not permitted to be in concurrent psychotherapy.

Interventions

Patients were randomly allocated to either CT or TAU and commenced treatment within a maximum of 3 weeks from the baseline assessment. Independent randomization took place through the use of computer-generated numbers. A treatment duration period of 6 months for both groups was based upon consideration of clinical implications, in particular, how translatable the therapy regimen would be to clinical practice in public sector mental health settings. This phenomena is especially highlighted in the current health services environment in which consideration of cost-effectiveness and minimal treatment duration requirements are considered paramount.

Treatment as usual. The TAU group received sessions as required by the patient's regular general practitioner or psychiatrist. These clinicians were provided with an educational package on bipolar disorder with detailed instructions for monitoring mood.

Cognitive therapy. The CT program was manualized and involved 20 1-hour weekly sessions administered by an experienced clinical psychologist (A.S.) with significant affective disorders expertise. She received intensive training in the use of CT with bipolar disorder and was supervised on a weekly basis by the senior author (J.R.B.). In order to evaluate the therapist's adherence to the fundamental principles of CT and this treatment program in particular, 1 in 5 sessions was randomly selected for rating according to Young and Beck's coding system.[28] Each tape was then assessed by 2 external clinical psychologists with postgraduate training in CT who were independent to the therapy component of the study. The system rated (1) the therapeutic relationship (genuineness, warmth, empathy, and rapport), (2) compliance to the fundamental principles of CT (collaboration, establishing an agenda, setting homework), and (3) adherence to 1 or more modules and interventions specified in the CT treatment manual. The CT comprised the following modules:

1. Assessment (approximately 1 session). Therapy began by examining the patient's understanding of bipolar disorder and involved a review of previous episodes, precipitants, coping strategies, and treatment history.
2. Psychoeducation (approximately 1 session). These sessions focused on psychoeducation about bipolar disorder within a cognitive-behavioral framework, with self-monitoring and self-regulation strategies being introduced early in the treatment process. Feelings of hopelessness and suicidality were addressed, with crisis management plans for acute episodes established.
3. Identifying early warning signs (approximately 1–2 sessions). Patients were taught to identify prodromal symptom patterns in the 2 to 4 weeks before a full manic or depressive relapse.
4. Establishing stable routines (approximately 1–2 sessions). Activity scheduling was introduced to help patients gradually increase pleasurable and achievement-oriented activities when depressed and to prioritize and reduce current levels of activity during hypomanic and manic states.
5. Identifying and modifying cognitions (approximately 6 sessions). Cognitive restructuring techniques were used to modify dysfunctional automatic thoughts, enhance problem solving, identify personal strengths, accept limitations, and construct a positive view of the future. Sessions were directed toward changing maladaptive behavioral patterns with tasks set to reinforce more adaptive behaviors. When hypomanic or manic, patients were encouraged to make realistic appraisals of the costs and benefits of pursuing grandiose goals. Reality testing (when delusions were present) and shifting attention were used to reduce impulsivity, improve decision making, and minimize the adverse consequences of inappropriate behavior.
6. Identifying and modifying schemas (approximately 8 sessions). Patients were given the opportunity to access and express emotions such as loss, fear, frustration, and anger through emotive techniques including role plays, writings, imagery, and discussions of past and current experiences. The individual traumas, losses, and adjustments associated with the patient's unique experience of bipolar disorder were addressed in detail. The Schema Questionnaire,[21] a commonly used clinical tool, was used in an attempt to identify and make conscious core beliefs. Patients were encouraged to acknowledge and label emotions and indicate when earlier experiences triggered similar emotional and somatic responses. More adaptive behavioral responses were encouraged. As these emotive techniques are arousing of affect, they were only used if appropriate for the needs of that particular patient and if his or her mood state was stable.

Assessment

At baseline, patients were assessed using the Structured Clinical Interview for DSM-IV[29] and a modified version of the Diagnostic Interview for Genetic Studies.[30] Relapse was defined as any bipolar episode that fulfilled DSM-IV criteria for major depressive disorder, mania, hypomania, or mixed episode. Throughout treatment, recordings were made of number of hospitalizations, number of episodes, and total days unwell. Such information was obtained from the patients' self-monitoring records of their moods on a daily basis, using a modified version of the structured system of Denicoff et al.[31] Medication adherence was monitored by serum mood stabilizer concentrations and a self-report measure. Relapse status was assessed using these monitoring records when patients were reviewed during the acute treatment phase at 5 weeks, 10 weeks, and 6 months and also at 12 months posttreatment follow-up by psychiatrists blind to the allocated treatment.

At baseline and the intervals specified above, psychosocial functioning was assessed according to 2 clinician-rated scales, namely, the Social Performance Scale (SPS)[32] and the Global Assessment of Functioning Scale (GAF),[33] and a self-report measure, the Social Adjustment Scale-Modified Version (SAS).[34]

Symptomatic severity was assessed using scores on 3 clinician rating scales: the HAM-D-17, the Montgomery-Asberg Depression Rating Scale (MADRS),[35] and the YMRS. Regular meetings were held to ensure reliability in administration of the measures. Several self-report measures were also administered, namely the BDI, the Beck Hopelessness Scale (BHS),[36] and the Internal State Scale.[37] In addition, patients also completed the Automatic Thoughts Questionnaire (ATQ),[38,39] the Dysfunctional Attitude Scale (DAS),[40] and the Self-Control Behavior Schedule (SCBS).[41]

The Clinical Global Impressions scale (CGI; as modified for bipolar disorder by Spearing et al.)[42] was administered at the 12-month follow-up point by the assessing psychiatrists who were blind to the patients' group allocation. Patients were rated on their improvement in the period preceding the assessment compared to the 18 months prior to the study (CGI-Ia) and improvement over the entire 18-month study period compared to the 18 months before entering the study (CGI-Ib).

The study coordinator maintained ongoing contact with psychiatrists and general practitioners involved in the patients' usual care.

Statistical Analyses

An intention-to-treat analysis was undertaken with the sample consisting of all patients who completed the baseline assessment. For continuous measures, the last-observation-carried-forward method was used to impute missing values. Student t tests were used to test for differences at baseline and posttreatment between the 2 groups. The Mann-Whitney U test was used for nonparametric data. Change within groups over time on continuous outcome variables was analyzed using repeated-measures analysis of variance. Chi-square analyses were used for dichotomous data, and the Fisher exact test was used when expected frequencies were lower than 5 in 1 or more cells. Analysis of covariance and logistic regression were used when significant differences on baseline measures between the 2 groups were presented. Survival analyses examining time to first episode were conducted using Cox regression. For those patients who were depressed at baseline, survival time was calculated from the time they entered the study rather than when they recovered from their episode. Additionally, the baseline episode was not counted as an event in the analysis. Therefore, those patients with a baseline major depressive episode were only censored in the analysis if they dropped out or went on to relapse. Relapse was defined as the occurrence of DSM-IV manic, hypomanic, depressive, or mixed episodes after at least 2 months' recovery, that is, 2 months of symptomatic remission (in line with DSM-IV bipolar disorder diagnostic guidelines).

RESULTS

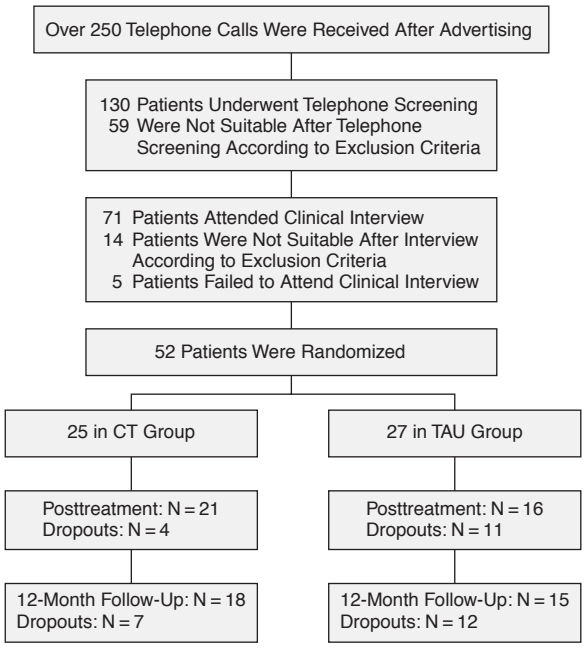
Patient Characteristics

A total of 130 individuals underwent a telephone-screening interview for inclusion into the study. Of these individuals, 71 were identified as appropriate and invited to attend a face-to-face screening interview. Five patients did not attend, and after such assessment, 14 patients were excluded as per study inclusion and exclusion criteria. A total of 52 patients were randomly assigned to receive either CT or TAU. Figure 1 depicts the recruitment and randomization process plus details of dropouts at each stage. No demographic differences were found between those patients who entered into the study and those who were referred but refused or did not attend for interview.

Table 1 presents the demographic and clinical characteristics of the intention-to-treat sample by group. There were no significant differences between groups on cross-sectional severity measures for depression or mania. However, scores on the BDI ($t = 1.97$, $p = .055$) and HAM-D-17 ($t = 1.90$, $p = .064$) approached significance, with the TAU group having higher levels of depressive symptomatology. There was a significant difference between the 2 groups in the proportion of patients who met DSM-IV criteria for a major depressive episode at baseline (8/27 vs. 0/25; $\chi^2 = 8.75$, df = 1, Fisher exact test, $p = .003$), with those in the CT group being less likely to enter treatment with a current depressive episode. Both groups demonstrated multiple prior manic and depressive episodes.

Forty-one percent (N = 11) of the TAU group and 20% (N = 5) of the CT group dropped out during the

Figure 1. Recruitment Flow and Attrition of Study Participants Randomly Assigned to Cognitive Therapy (CT) or Treatment as Usual (TAU)



active treatment phase after week 5. Reasons for not completing treatment included the following: 25% (N = 4) "did not feel they were benefiting from participation," 19% (N = 3) were too mentally unwell, 6% (N = 1) wanted to pursue additional cognitive therapy, 6% (N = 1) died of natural causes (the patient was aged 75 years), 19% (N = 3) continued to participate in the study but did not attend the posttreatment review, and for 25% (N = 4) reasons for dropping out were unknown. The difference in dropout rates between the CT and TAU groups demonstrated a trend but was not formally significant ($\chi^2 = 2.91$, df = 1, p = .09).

Therapist Adherence

Interrater agreement was high for all 3 factors rated. Clear agreement between raters was present in 98.3% of the ratings for both the nature of the therapeutic relationship and adherence to the components of CT. Similarly, both raters agreed 94.3% of the time that the fundamentals of CT were clearly present.

Medication Compliance

Medication adherence was assessed by both serum mood stabilizer concentrations and self-report. However, while blood tests for mood stabilizer concentrations were routinely ordered as part of the study protocol, few patients complied. Accordingly, as numbers were so few, these data are not presented here. Therefore, medication adherence was assessed solely by the self-report questionnaire. At posttreatment, 40.7% (11/27) of the TAU group and 60.0% (15/25) of the CT group report adequate compliance with medication (defined as reporting missing medications no more than twice at 2 of the 3 assessment occasions). At 12-month follow-up, 66.7% (18/27) of the TAU group and 60.0% (15/25) of the CT group reported adequate medication compliance (defined as reporting missing medication no more than twice at 6 of the 7 assessment occasions). There were no significant differences between the groups on self-reported medication compliance at either posttreatment or 12-month follow-up.

Episodes

At posttreatment, a relapse was experienced by 26.9% of the sample (N = 14), with relapse defined as experiencing either a DSM-IV manic, hypomanic, mixed, or depressive episode. Relapse rates were 20.0% (5/25) for the CT group and 33.3% (9/27) for the TAU group. No significant differences were observed between the 2 groups for the number of overall bipolar episodes or singular episode types.

When time to depressive relapse was examined, Cox regression showed that those in the TAU group had a shorter time to relapse compared to the CT group (hazard ratio = 0.11, 95% CI = 0.01 to 0.91; p = .04). After controlling for the presence of a depressive episode at baseline, this result approached significance (hazard ratio = 0.12, 95% CI = 0.13 to 1.08; p = .06). The actuarial cumulative relapse rates for depressive episodes for the CT and TAU groups, respectively, were 4.0% (1/25) and 25.9% (7/27). This trend was not observed for overall bipolar episodes or manic/hypomanic episodes.

Over the entire 18 months, a bipolar relapse was experienced by 55.8% (N = 29) of the sample. The relapse rates for the CT and TAU groups, respectively, were 52.0% (13/25) and 59.3% (16/27) at 12-month follow-up. No significant differences between the 2 groups were found. Figure 2 depicts the survival analysis of the 2 groups, with the number of weeks preceding the first bipolar disorder episode as the dependent variable and a major depressive episode at baseline as a covariate. The hazard ratio for relapse in the CT group relative to the TAU group was nonsignificant (hazard ratio = 0.66, 95% CI = 0.29 to 1.51; p = .33).

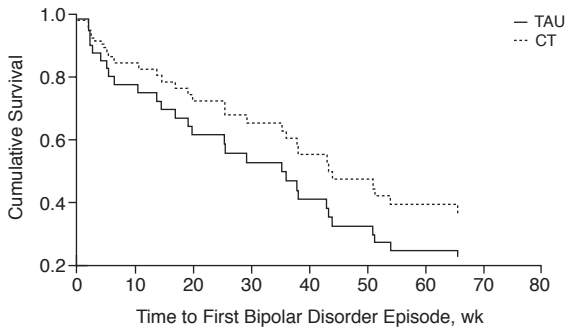
At 12-month follow-up, Cox regression showed group differences for depressive episodes that approached significance (hazard ratio = 0.38, 95% CI = 0.14 to 1.03; p = .057), with a trend for those in the CT group to have a longer time to relapse compared to the TAU group. However, after controlling for the presence of a depressive episode at baseline, this group difference was no longer significant (hazard ratio = 0.57, 95% CI = 0.17 to 1.88; p = .36; Figure 3). The actuarial

Table 1. Baseline Characteristics of the Intention-to-Treat Sample By Group

Variable	Cognitive Therapy Group (N = 25)	Treatment as Usual Group (N = 27)
Age, mean (SD), y	41.56 (14.61)	42.52 (14.49)
Age, range, y	26–77	23–76
Female, N	14	16
Beck Depression Inventory score, mean (SD)	12.88 (7.98)	18.96 (13.23)*
17-item Hamilton Rating Scale for Depression score, mean (SD)	4.64 (6.67)	6.26 (5.65)*
Young Mania Rating Scale score, mean (SD)	1.92 (4.24)	1.22 (2.83)
Depressive episode at baseline, N (%)	0	8 (29.6)**
Hypomanic episode at baseline, N (%)	2 (8.0)	3 (11.1)
No. of depressive episodes 18 months prior to study, mean (SD)	1.20 (0.71)	1.37 (1.28)
No. of manic episodes 18 months prior to study, mean (SD)	0.56 (0.82)	0.67 (0.73)
No. of hypomanic episodes 18 months prior to study, mean (SD)	0.92 (0.95)	0.89 (1.25)
No. of mixed episodes 18 months prior to study, mean (SD)	0.00	0.04 (0.19)
No. of hospitalizations 18 months prior to study, mean (SD)	0.52 (0.77)	0.78 (1.22)
No. of hospitalizations 18 months prior to study, range	0–3	0–6
No. of lifetime depressive episodes, mean (SD)	6.65 (6.09)	9.08 (10.49)
No. of lifetime manic/hypomanic episodes, mean (SD)	4.60 (3.25)	7.12 (5.99)
No. of lifetime mixed episodes, mean (SD)	0.36 (1.11)	0.23 (0.71)
Patients taking mood stabilizers, N (%)		
1 mood stabilizer	23 (92.0)	22 (81.5)
2 mood stabilizers	2 (8.0)	2 (7.4)
Patients taking antidepressants, N (%)	13 (52.0)	14 (51.9)
Patients taking major tranquilizers, N (%)	5 (20.0)	6 (22.2)

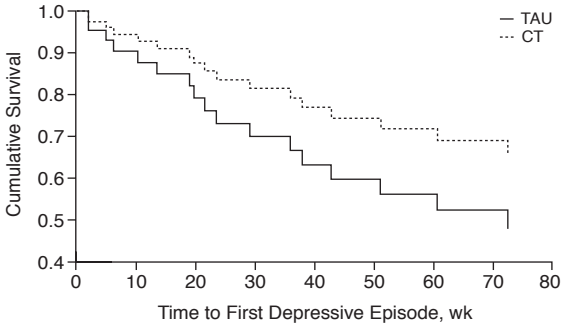
*p < .10.
**p < .05.

Figure 2. Time to Bipolar Disorder Relapse (hypomanic, manic, depressive, or mixed) for Patients Randomly Assigned to Treatment With Cognitive Therapy (CT) or Treatment as Usual (TAU)[a]



[a]The figure depicts the survival analysis of the 2 groups, with the number of weeks preceding the first bipolar disorder episode as the dependent variable and a major depressive episode at baseline as a covariate.

Figure 3. Time to Depressive Episode Relapse for Patients With Bipolar Disorder Randomly Assigned to Treatment With Cognitive Therapy (CT) or Treatment as Usual (TAU)



cumulative depressive relapse rates for the CT and TAU groups, respectively, were 24.0% (6/25) and 40.7% (11/27) at 12-month follow-up. Identical analyses conducted for manic/hypomanic episodes were nonsignificant. It should be noted that all 8 subjects with a baseline major depressive episode recovered from this index episode at some point during the study.

At posttreatment, the CT group and the TAU group had experienced a similar number of mean (SD) days in a bipolar episode, 21.20 (19.89) and 20.58 (25.71) days, respectively. By 12-month follow-up, days in a bipolar episode had increased to a mean (SD) of 74.96 (53.52) and 75.95 (86.64) days, respectively. No significant differences were observed between the 2 groups at either time point. A similar pattern of results was observed for number of days with depressive episodes, with the CT group reporting a mean (SD) of 12.83 (15.15) days and the TAU group reporting 18.61 (25.56) days. By 12-month follow-up, number of days with a depressive episode had increased to a mean (SD) of 46.65 (36.99) days for the CT group and 57.07 (65.61) days for the TAU group. Days with manic/hypomanic symptoms were less common in the sample, with the CT group experiencing a mean (SD) of 7.21 (13.76) days and the TAU group a mean (SD) of 1.75 (2.74) days. At 12-month follow-up, days with manic/hypomanic symptoms had increased to a mean

Table 2. Scores of the Measures Administered Over the 24-Week Study Period for the Intention-to-Treat Sample: Cognitive Therapy (N = 25) and Treatment as Usual (N = 27)[a]

	Baseline		Posttreatment		3-Month Follow-Up		6-Month Follow-Up		9-Month Follow-Up		12-Month Follow-Up	
Scale	CT	TAU	CT	TAU	CT	TAU	CT	TAU	CT	TAU	CT	TAU
Depression												
BDI (range, 0–63)	12.88 (7.97)	18.96[d] (13.23)	6.60 (6.76)	13.32[b,c] (10.39)	10.32 (9.97)	13.64 (12.51)	8.60 (8.08)	12.84 (12.92)	9.72 (7.61)	11.72 (11.93)	10.12 (10.75)	10.92[b] (10.39)
BHS (range, 0–20)	7.72 (4.80)	8.60 (5.56)	5.24 (4.26)	6.56[b] (5.82)	5.92 (5.28)	6.64 (6.25)	6.60 (5.12)	6.56 (6.22)	6.24 (4.91)	6.12 (5.85)	6.56 (5.58)	6.40[b] (6.23)
HAM-D-17 (range, 0–52)	3.16 (3.42)	5.48[d] (5.00)	3.32 (5.29)	5.87[d] (6.84)	4.48 (6.04)	6.15 (7.25)	4.92 (6.73)	6.52 (8.29)	4.76 (6.34)	6.52 (7.95)	3.96 (6.62)	5.33 (6.70)
MADRS (range, 0–60)	4.36 (5.63)	8.37[c] (7.85)	3.96 (5.88)	8.33[c] (9.10)	5.84 (8.70)	7.44 (8.71)	5.44 (7.75)	7.93 (11.53)	6.60 (8.68)	8.15 (11.13)	5.76 (10.16)	6.52 (9.20)
Mania												
YMRS (range, 0–60)	1.92 (4.24)	1.22 (2.83)	0.84 (3.41)	0.89 (2.22)	1.60 (3.89)	0.78 (2.14)	0.32 (0.99)	0.78 (2.15)	0.32 (0.90)	0.78 (2.15)	0.00	0.85[b,d] (2.36)
SRMI (range, 0–46)[43]	6.76 (7.29)	7.20 (6.96)	4.80 (7.09)	6.44 (6.63)	5.16 (7.24)	7.00 (7.05)	4.76 (6.67)	7.36 (6.47)	5.16 (6.78)	6.24 (6.08)	3.92 (5.33)	6.04 (6.34)
Psychosocial functioning												
GAF (range, 0–100)	80.68 (11.17)	74.70 (14.30)	81.56 (9.25)	75.67 (16.60)	75.72 (13.49)	75.85 (17.43)	79.08 (14.03)	75.37 (18.84)	76.96 (13.77)	74.89 (19.79)	81.28 (11.68)	77.85 (15.98)
SAS (range, 0–225)	15.73 (3.25)	17.02 (4.88)	13.47 (2.61)	15.28[b,d] (4.60)	14.25 (3.74)	14.54 (4.28)	13.60 (3.46)	14.54 (4.87)	13.89 (3.22)	14.44 (4.78)	14.11 (3.83)	14.88[b] (5.07)
SPS (range, 0–100)	12.78 (15.09)	20.13 (17.92)	8.09 (11.51)	15.11[b,d] (14.56)	12.08 (16.05)	15.93 (14.53)	8.34 (12.75)	15.72[d] (16.62)	8.59 (12.43)	15.73[d] (16.82)	11.72 (19.01)	12.15 (13.50)
Cognitive functioning												
ATQ-N (range, 30–150)	60.88 (18.65)	70.57 (30.10)	56.36 (21.17)	65.13 (26.21)	57.16 (26.22)	60.65 (32.39)	53.80 (18.23)	65.95 (35.15)	56.44 (20.52)	59.74 (27.23)	62.60 (26.00)	56.65 (27.27)
SCBS (range, –108 to 108)	4.83 (26.98)	3.00 (28.64)	13.00 (23.75)	3.33 (29.18)	13.21 (28.61)	6.21 (33.19)	17.04 (29.81)	7.63 (37.64)	15.21 (30.50)	11.75 (35.01)	29.71 (36.42)	11.04[b,d] (34.97)
DAS (range, 40–280)	133.40 (34.03)	139.25 (32.57)	117.52 (29.42)	136.33[b,c] (35.03)	116.76 (33.94)	135.00 (41.39)	116.08 (35.87)	133.67 (43.01)	118.88 (36.29)	128.04 (33.11)	115.08 (28.88)	128.00[b] (35.18)

[a]Values are presented as mean (SD).
[b]Significant main effect of time, $p < .05$.
[c]Significant difference between groups, $p < .05$.
[d]Difference between groups approached significance, $p < .10$.
Abbreviations: ATQ-N = Automatic Thoughts Questionnaire-Negative subscale, BDI = Beck Depression Inventory, BHS = Beck Hopelessness Scale, CT = cognitive therapy, DAS = Dysfunctional Attitude Scale, GAF = Global Assessment of Functioning, HAM-D-17 = 17-item Hamilton Rating Scale for Depression, MADRS = Montgomery-Asberg Depression Rating Scale, SAS = Social Adjustment Scale, SCBS = Self-Control Behavior Schedule, SPS = Social Performance Scale, SRMI = Self-Report Manic Inventory, TAU = treatment as usual, YMRS = Young Mania Rating Scale.

(SD) of 26.39 (32.88) and 17.95 (34.74) days for the CT and TAU groups, respectively.

Continuous Outcome Measures

As detailed in Table 2, at posttreatment, the CT group had significantly greater improvement on the BDI ($t = 2.71$, $p = .009$), the DAS ($t = 2.04$, $p = .047$), and the MADRS ($t = 2.98$, $p = .04$). A trend in the same direction was observed for the HAM-D-17 ($t = 1.90$, $p = .06$), SAS ($t = 1.71$, $p = .094$), and SPS ($t = 1.92$, $p = .06$). The SPS also approached significance at 6-month ($t = 1.79$, $p = .080$) and 9-month follow-up ($t = 1.73$, $p = .09$), with those in the CT group rated as experiencing lower levels of disability due to bipolar disorder compared to those in the TAU group. However, this trend was not maintained or strengthened at 12-month follow-up. At 12-month follow-up, a trend for the CT group to have improved scores relative to the TAU group on the SCBS ($t = –1.81$, $p = .08$) and the YMRS ($z = –1.80$, $p = .08$) was observed.

Table 2 also shows the mean scores of the continuous measures administered to the patients over the overall 18-month period. There was a significant time effect for the BDI ($F = 5.14$, $df = 7,336$; $p < .00$), BHS ($F = 3.48$, $df = 7,336$; $p = .001$), YMRS ($F = 2.10$, $df = 7,350$; $p = .04$), ATQ-Positive subscale ($F = 3.72$, $df = 5,41$; $p = .007$), DAS ($F = 3.43$, $df = 5,235$; $p = .005$), SCBS ($F = 5.21$, $df = 5,230$; $p < .00$), and SAS ($F = 4.85$, $df = 7,336$; $p < .00$), indicating that, on average, both groups improved over the 18-month study period. The time effect for the SPS ($F = 1.90$, $df = 5,250$; $p = .095$) approached significance. There were no significant time × group effects over this 18-month interval.

Clinical Global Impression

At 12-month follow-up, those in the CT group were rated as experiencing greater improvement in the severity of their illness since the last follow-up assessment compared to the 18 months preceding the study (CGI-Ia). The CT group had greater improvements in the severity of their depressive symptoms (17/25 vs. 9/27; $\chi^2 = 6.24$, $df = 1$, $p = .01$) and overall bipolar symptoms (19/25 vs. 13/27; $\chi^2 = 4.25$, $df = 1$, $p = .04$) compared to the TAU group. This difference only approached significance for mania symptoms (17/25 vs. 12/27; $\chi^2 = 2.92$, $df = 1$, $p = .09$). When improvement in illness severity over the 18-month period of the

study was compared to their illness severity in the 18-month period preceding the study, there were no significant differences between the groups (CGI-Ib). As this scale was only administered once, at the end of the 12-month follow-up period, data were only available for the completer sample.

DISCUSSION

This is the first randomized controlled trial in bipolar disorder to evaluate the efficacy of CT modeled to include emotive techniques. The CT group showed significantly lower BDI and MADRS scores posttreatment compared to the TAU group. After controlling for the presence of a (mild) major depressive episode at baseline, a trend toward longer time to depressive relapse remained for the CT group. These findings are similar to previous randomized controlled trials evaluating CT for bipolar disorder,[10–13] which have also demonstrated reduction in depressive symptoms and prolonged time to relapse at posttreatment. Since the CT group did not differ from the TAU group in medication adherence, this outcome could not simply be attributed to compliance effects. At 12-month follow-up, these between-group differences in depressive symptomatology and relapse were no longer significant.

Cognitive therapy demonstrated significantly greater benefits in dysfunctional attitudes at posttreatment than TAU, with similar trends also for social adjustment and social performance. The relative benefits among CT participants in these measures were strongest during active treatment, with the significance gradually diminishing as the effect of therapy became more distant. Scores on the SPS continued to approach significance at 6- and 9-month follow-up, but had weakened by follow-up at 12 months. Dysfunctional Attitude Scale scores in the CT group remained in the normal range at both posttreatment and follow-up, with no loss of treatment benefits occurring over this period ($F = 0.29$, $df = 1,24$; $p = .88$), although there were no significant differences or trends compared to TAU after the immediate posttreatment assessment. At follow-up, the TAU group was not in the normal range according to the DAS norms, suggesting that the DAS scores only normalized in the CT group. A formal statistical difference was not demonstrated, possibly due to lack of statistical power. The robustness of the CT group to remain in the normal range at posttreatment and follow-up suggests the sustainability of attitudinal change once active treatment was withdrawn, although it must be acknowledged that DAS scores for the CT group were no longer superior to the TAU group.

At 12-month follow-up, the CT group showed trends (although nonsignificant) toward more effective self-control behaviors and a lower YMRS score than the TAU group. This improvement in manic symptoms at follow-up is in accordance with the findings of Lam et al.[16] and Perry et al.[9] in which the latter study employed cognitive-behavioral techniques to help patients detect early warning signs. Given the small sample size of the current study, these trends are considered noteworthy. At posttreatment, the CT group scored in the "recovery range" for self-control behaviors and gradually improved until eventually reaching the normal range at 12-month follow-up. Mania has recently been conceptualized in terms of dysregulation of the behavioral activation system responsible for regulating a broad band of goal-seeking behavior.[16] Modification of these behaviors is considered to influence the course of the bipolar illness. In our study, cognitive changes demonstrated on the DAS appeared to precede self-regulatory behavioral change. While several previous trials have included follow-up data,[9,11,13] this is the first study to demonstrate sustained benefits in dysfunctional attitudes 12 months after therapy had been withdrawn. These findings offer partial support for our primary hypotheses.

The strongest finding at 12-month follow-up was found on the CGI-I, with the CT group being rated as demonstrating significantly greater improvement in the severity of their illness compared to the 18 months preceding the study. The CGI is widely used as a primary outcome measure in clinical trials and has also been used to gauge the active phase of treatment response and overall prophylactic benefits in bipolar disorder studies.[44,45] In modifying the CGI for use with patients with bipolar disorder, the scale has been standardized, increasing its reliability, validity, and utility of ratings.[42] The CT group was rated as showing the greatest improvement in the severity of depressive relapse and symptoms and overall bipolar disorder features and approached significance for hypomanic/manic features. This discrepancy on depressive symptoms when compared to other measures of depression at follow-up may partially be explained by the CGI being conducted on treatment completers only, whereas the other measures were analyzed on an intention-to-treat basis. It is also possible that the clinicians were detecting some change in the patients' depressive features of which the patients themselves were unaware.

The difference between attrition rates during the active treatment phase among the CT group (20%) and the TAU group (41%) approached significance ($p < .09$), with most patients dropping out in the first weeks of treatment. These percentage rates are historically consistent with most clinical trials for bipolar disorder, be they focused on medication or psychotherapy. Compared to the majority of comparative trials of CT in bipolar disorder, with 1 exception,[12,13] these rates appear relatively favorable.

The efficacy of CT in eliciting certain benefits at posttreatment and to a lesser degree at follow-up may, in part, be attributed to the addition of emotive techniques in our CT protocol. Emotive techniques are known to be power-

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ful in accessing maladaptive beliefs and constructing more adaptive cognitive, emotional, and behavioral responses. It has been proposed that cognitive change depends on a certain level of affective experience,[22] and such emotive techniques are sometimes necessary "to open up the sluices of new learning or unlearning."[23(p80)] Imagery techniques have long been considered powerful means of reconstructing experiences and generating alternative attitudes and emotions.[46] New emotions are elicited, not by changing feelings with reason, but through imagination and priming.[24] Furthermore, the importance of narratives in helping patients relive their experiences and access feelings is also widely used as a means of altering affect and dysfunctional attitudes.[47] Emotive techniques may be effecting emotional and cognitive changes leading to deeper and enduring changes in the patients' deeper schematic structures and subsequent behaviors.[46] To understand more fully the contribution of these techniques to enhancing cognitive-behavioral techniques, it would be necessary to directly compare CT with and without emotive techniques.

Both groups demonstrated significant improvements over the course of treatment on numerous measures at posttreatment. Given the recurrent nature of bipolar disorder and the requirement that patients in the study had experience of at least 1 bipolar episode in the 18 months prior to participating in treatment, it is unlikely that these effects could be explained purely in terms of the course of the illness. It appears that the TAU intervention may have also been effective to some extent in the modification of mood, attitudes, and social functioning over the treatment and follow-up periods, but that CT provided stronger benefits in some areas. In our study, TAU was designed as the ideal routine care model for general practitioners and psychiatrists to follow in managing patients with bipolar disorder. Treatment as usual involved medication review, ongoing mood monitoring, and brief psychoeducation. This model of TAU was chosen not only to minimize attrition rates, but also to ensure accurate assessment of mood status over the treatment period. Previous randomized controlled trials for bipolar disorder have often been criticized for their reliance on retrospective patient recall or analysis of case notes in order to identify relapses and admissions.

A further strength of our study was the use of a manualized treatment design that increased internal validity and offered a clearer distinction between treatment conditions. The rating of treatment fidelity enabled us to demonstrate high therapist adherence to this particular treatment intervention.

However, our study had several limitations. The small sample size and relatively large number of dependent variables restricted the power to demonstrate differences between the groups and increased the risk of type I and type II errors. A review of comparative studies on CT for bipolar disorder (Cochran,[8] N = 28; Zaretsky et al.,[10] N = 22; Scott et al.,[11] N = 42; Lam et al.,[3] N = 103) indicated that, to detect a moderate effect size with a probability level of 0.05 and 80% power, a total sample size of 102 would be required. Due to recruitment issues, our sample size of 52 was below the requirement for adequate statistical power, though it still represents one of the larger CT studies for this condition.

Another limitation stems from a single therapist (A.S.) delivering this modified form of CT to all patients, which inevitably limits the extent to which the results of the study may be generalized. Furthermore, no control was made for the attentional effects or nonspecific effects of treatment. At the time of the study onset, there were no valid measures with demonstrated reliability and validity for assessing emotive techniques. The Schema Questionnaire[21] has been developed as a clinical instrument for assessing core beliefs but is highly mood dependent and lacks valid normative data. Another issue that must be acknowledged is that of low compliance rates with blood testing. The poor compliance of patients attending for blood draws was probably due to the unavailability of a convenient venipuncture facility. The limitations of this unavailability are acknowledged.

CONCLUSIONS

Our findings support other recent studies suggesting that CT is effective in prolongation of time to depressive relapse, reduction of depressive symptoms, modification of dysfunctional attitudes, and, to a lesser extent, improved social adjustment and performance. This is one of the first CT trials (in accordance with Lam et al.[13]) in which patients with bipolar disorder have shown a suggestion of maintaining significant improvements in dysfunctional attitudes for 12 months after active CT has been withdrawn. The study of Lam et al.[13] using traditional CT reported very similar results to the present study. In order to clarify more clearly whether the addition of emotive techniques can add to the efficacy of CT treatment, it would be necessary for future research to directly compare CT with and without emotive techniques. At follow-up, independent clinicians' ratings suggested that patients in the CT group improved more than the TAU group compared to the 18 months prior to treatment; furthermore, a trend was noted for improved self-control behavior and fewer hypomanic/manic symptoms.

However, many benefits gradually diminished once psychological treatment was withdrawn, suggesting that mood stabilizers alone do not appear to be sufficient in sustaining the positive effects once CT has ceased. This study has implications for future CT trials with patients with bipolar disorder in highlighting the importance of maintenance psychological therapy or booster sessions once acute treatment has ceased.

Disclosure of off-label usage: The authors have determined that, to the best of their knowledge, no investigational information about pharmaceutical agents that is outside U.S. Food and Drug Administration–approved labeling has been presented in this article.

REFERENCES

1. Mitchell PB, Malhi GS. The expanding pharmacopoeia for bipolar disorder. Annu Rev Med 2002;53:173–188
2. Gitlin MJ, Swendsen J, Heller TL, et al. Relapse and impairment in bipolar disorder. Am J Psychiatry 1995;152:1635–1640
3. Lam DH, Bright J, Jones S, et al. Cognitive therapy for bipolar illness: a pilot study of relapse prevention. Cognit Ther Res 2000;24:303–320
4. Jamison KR. Touched With Fire: Manic-Depressive Illness and the Artistic Temperament. New York, NY: Free Press; 1998
5. MacQueen GM, Young LT, Joffe RT. A review of psychosocial outcome in patients with bipolar disorder. Acta Psychiatr Scand 2001;103: 163–170
6. Murray CH, Lopez AD, eds. The Global Burden of Disease. Geneva, Switzerland: World Health Organization; 1996
7. Beck AT, Rush AJ, Shaw BF, et al. Cognitive Therapy of Depression. Elmsford, NY: Pergamon Press; 1979
8. Cochran SD. Preventing medical noncompliance in the outpatient treatment of bipolar affective disorders. J Consult Clin Psychol 1984;52: 873–878
9. Perry A, Tarrier N, Morriss R, et al. Randomized controlled trial of efficacy of teaching patients with bipolar disorder to identify early symptoms of relapse and obtain treatment. BMJ 1999;318:49–53
10. Zaretsky AE, Segal ZV, Gemar M. Cognitive therapy for bipolar depression: a pilot study. Can J Psychiatry 1999;44:491–494
11. Scott J, Garland A, Moorhead S. A pilot study of cognitive therapy in bipolar disorders. Psychol Med 2001;31:459–467
12. Lam DH, Watkins ER, Haywood P. A randomized controlled study of cognitive therapy for relapse prevention for bipolar affective disorder: outcome of the first year. Arch Gen Psychiatry 2003;60:145–152
13. Lam DH, Haywood P, Watkins ER, et al. Relapse prevention in patients with bipolar disorder: cognitive therapy outcome after 2 years. Am J Psychiatry 2005;162:324–329
14. Vieta E, Colom F. Psychological interventions in bipolar disorder: from wishful thinking to an evidence-based approach. Acta Psychiatr Scand Suppl 2004;422:34–38
15. Scott J, Pope M. Cognitive styles in individuals with bipolar disorder. Psychol Med 2003;33:1081–1088
16. Lam D, Wright K, Smith N. Dysfunctional assumptions in bipolar disorder. J Affect Disord 2004;79:193–199
17. Ball J, Mitchell P, Malhi G, et al. Schema-focused cognitive therapy for bipolar disorder: reducing vulnerability to relapse through attitudinal change. Aust N Z J Psychiatry 2003;37:41–48
18. Williams C. A cognitive model of dysfunctional illness behavior. Br J Health Psychol 1997;2:153–165
19. Newman CF, Leahy RL, Beck AT, et al. Bipolar Disorder: A Cognitive Therapy Approach. Washington, DC: American Psychological Association; 2002
20. Greenberg LS, Watson J. Experiential therapy of depression: differential effects of client-centered relationship conditions and active experiential interventions. Psychother Res 1998;8:210–224
21. Young JE. Cognitive Therapy for Personality Disorders: A Schema-Focused Approach. Sarasota, Fla: Professional Resource Press; 1994
22. Beck AT. Cognitive therapy. In: Zeig JK, ed. The Evolution of Psychotherapy. New York, NY: Brunner/Mazel; 1987
23. Beck AT, Freeman A. Cognitive Therapy of Personality Disorder. New York, NY: The Guilford Press; 1990
24. Greenberg LS. Emotion-Focused Therapy. Washington, DC: American Psychological Association; 2002
25. Beck AT, Steer RA, Brown GK. Beck Depression Inventory. 2nd ed. San Antonio, Tex: The Psychological Corporation; 1996
26. Hamilton M. A rating scale for depression. J Neurol Neurosurg Psychiatry 1960;23:56–62
27. Young RC, Biggs JT, Ziegler VE, et al. A rating scale for mania: reliability, validity and sensitivity. Br J Psychiatry 1978;133:429–435
28. Young J, Beck AT. Cognitive Therapy Scale: Rating Manual. Philadelphia, Pa: Beck Institute for Cognitive Therapy and Research; 1980
29. First MB, Spitzer RL, Gibbon M, et al. Structured Clinical Interview for DSM-IV Axis I, Clinical Version (SCID-CV). Washington, DC: American Psychiatric Press; 1997
30. Nurnberger JI Jr, Blehar MC, Kaufmann CA, et al. Diagnostic Interview for Genetic Studies: rationale, unique features, and training. NIMH Genetics Initiative. Arch Gen Psychiatry 1994;51:849–859
31. Denicoff KD, Smith-Jackson EE, Disney ER, et al. Preliminary evidence of the reliability and validity of the prospective life-chart methodology (LCM-p). J Psychiatr Res 1997;31:593–603
32. Hurry J, Sturt R, Bebbington P, et al. Sociodemographic associations with social disablement in a community sample. Soc Psychiatry 1983; 18:113–121
33. Endicott J, Spitzer RL, Fleiss JL, et al. The Global Assessment Scale: a procedure for measuring overall severity of psychiatric disturbance. Arch Gen Psychiatry 1976;33:766–771
34. Cooper P, Osborn M, Gath D, et al. Evaluation of a modified self-report measure of social adjustment. Br J Psychiatry 1982;141:68–75
35. Montgomery SA, Asberg M. A new depression scale designed to be sensitive to change. Br J Psychiatry 1979;134:382–389
36. Beck AT, Weissman A, Lester D, et al. The measurement of pessimism: the Hopelessness Scale. J Consult Clin Psychol 1974;42:861–865
37. Bauer MS, Crits-Cristoph P, Ball WA, et al. Independent assessment of manic and depressive symptoms by self-rating: scale characteristics and implications for the study of mania. Arch Gen Psychiatry 1991;48:807–812
38. Hollon SD, Kendall PC. Cognitive self-statements in depression: development of an Automatic Thoughts Questionnaire. Cognit Ther Res 1980;4:383–395
39. Ingram RE, Wisnicki KS. Assessment of positive automatic cognition. J Consult Clin Psychol 1988;56:898–902
40. Weissman AN. The Dysfunctional Attitude Scale: a validation scale. Dissertation Abstract Int 1979;40:1389B–1390B
41. Rosenbaum M. A schedule for assessing self-control behaviors: preliminary findings. Behav Ther 1980;11:109–121
42. Spearing MK, Post RM, Leverich GS, et al. Modification of the Clinical Global Impressions (CGI) scale for use in bipolar disorder (BP): the CGI-BP. Psychiatry Res 1997;73:159–171
43. Shugar G, Schertzer S, Toner B, et al. Development, use and factor analysis of the self-report inventory for mania. Comp Psychiatry 1992;33:325–331
44. Okuma T. Effects of carbamazepine and lithium on affective disorder. Neuropsychobiology 1993;27:138–145
45. Calabrese JR, Woyshville MJ, Kimmel SE, et al. Predictors of valproate response in bipolar rapid cycling. J Clin Psychopharmacol 1993;13: 280–283
46. Layden MA, Newman CF, Freeman A, et al. Cognitive Therapy of Borderline Personality Disorder. Boston, Mass: Allyn and Bacon; 1993
47. Little M, Jordens C, Paul K, et al. Surviving Survival: Life After Cancer. Sydney, Australia: Choice; 2001

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