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Improvement in social-interpersonal functioning after cognitive therapy for recurrent depression

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Abstract

Background. Cognitive therapy reduces depressive symptoms of major depressive disorder, but little is known about concomitant reduction in social-interpersonal dysfunction.

Method. We evaluated social-interpersonal functioning (self-reported social adjustment, interpersonal problems and dyadic adjustment) and depressive symptoms (two self-report and two clinician scales) in adult outpatients ($n=156$) with recurrent major depressive disorder at several points during a 20-session course of acute phase cognitive therapy. Consenting acute phase responders ($n=84$) entered a 2-year follow-up phase, which included an 8-month experimental trial comparing continuation phase cognitive therapy to assessment-only control.

Results. Social-interpersonal functioning improved after acute phase cognitive therapy (dyadic adjustment $d=0.47$; interpersonal problems $d=0.91$; social adjustment $d=1.19$), but less so than depressive symptoms ($d=1.55$). Improvement in depressive symptoms and social-interpersonal functioning were moderately to highly correlated ($r=0.39-0.72$). Improvement in depressive symptoms was partly independent of social-interpersonal functioning ($r=0.55-0.81$), but improvement in social-interpersonal functioning independent of change in depressive symptoms was not significant ($r=0.01-0.06$). In acute phase responders, continuation phase therapy did not further enhance social-interpersonal functioning, but improvements in social-interpersonal functioning were maintained through the follow-up.

Conclusions. Social-interpersonal functioning is improved after acute phase cognitive therapy and maintained in responders over 2 years. Improvement in social-interpersonal functioning is largely accounted for by decreases in depressive symptoms.

INTRODUCTION

Major depressive disorder often involves significant social-interpersonal dysfunction (e.g. Fredman *et al.* 1988; Gotlib & Lee, 1989; Leader & Klein, 1996; Zlotnick *et al.* 2000). Diagnosis requires an essential depressive symptom (depressed mood or anhedonia), additional depressive symptoms (e.g. neurovegetative signs, negatively focused cognition), and more general ‘functional impairment,’ which may include social-interpersonal dysfunction (APA, 1994). Cognitive therapy (Beck *et al.* 1979), focused on relieving depressive symptoms, has proven quite efficacious in this regard over decades of research (e.g. Jarrett & Rush, 1994; Craighead *et al.* 1998; Strunk & DeRubeis, 2001). In contrast, the extent to which concomitant social-interpersonal dysfunction, which may be a common motivation for seeking treatment (e.g. Meller *et al.* 1989; Wills & DePaulo, 1991), also improves with cognitive

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therapy is not nearly as well assessed or understood. In this report, we evaluate changes in multiple measures of depressive symptoms and self-reported social-interpersonal functioning across acute-phase cognitive therapy and a 2-year follow-up, including a randomized clinical trial comparing 8 months of a continuation cognitive therapy to an assessment-only control. Participants were adult outpatients with DSM-IV (APA, 1994) recurrent major depressive disorder with clear inter-episode recovery.

Although causal connections are not always clear, a large empirical literature strongly links depressive symptoms with social-interpersonal dysfunction. For example, poor social adjustment in various roles and contexts (e.g. as a worker or a parent, with friends or family, in leisure activities) has been linked with several depressive diagnoses (e.g. Fredman *et al.* 1988; Leader & Klein, 1996) and has been shown to improve (although not fully normalize) with remission (Weissman & Paykel, 1974). Moreover, interpersonal problems in depression involving maladaptive behaviors, thoughts and feelings in interpersonal situations (e.g. inappropriate negative self-disclosure, lower assertiveness; Segrin, 2000) predict negative mood (Coyne, 1976), perceptions of low social skill (Lewinsohn *et al.* 1980) and even social rejection (e.g. Joiner *et al.* 1992; Joiner, 1999) from depressed persons' social interaction partners. Finally, discord in marriage and similar dyads correlates moderately with depressive symptoms (O'Leary *et al.* 1994) and major depressive disorder is associated with poor overall marital adjustment (Stravynski *et al.* 1995; Dudek *et al.* 2001), including unpleasant interactions with the spouse or partner (Zlotnick *et al.* 2000).

Unfortunately, there is also considerable evidence that a reduced level of social-interpersonal dysfunction often persists beyond remission of depression. For example, social functioning may improve less than depressive symptoms with treatment and remain impaired relative to control groups at longitudinal follow-up (Gotlib & Lee, 1989). Moreover, persons with major depressive disorder in remission may have poorer social functioning than those without a history of mental illness (Serretti *et al.* 1999) and poorer marital adjustment (as rated by spouses) than persons with bipolar disorder in remission (Horesh & Fennig, 2000).

Social-interpersonal functioning may improve with cognitive therapy for depression. For example, social adjustment improves comparably with cognitive therapy, interpersonal psychotherapy and pharmacotherapy plus clinical management (Imber *et al.* 1990); and partial responders to pharmacotherapy make gains in social adjustment with the addition of cognitive therapy (Scott *et al.* 2000). However, cognitive therapy for depressed wives may not produce significant gains in dyadic adjustment, whereas behavioral marital therapy does produce gains in dyadic adjustment (Beach & O'Leary, 1992). Deeper examination of improvement in social-interpersonal functioning with cognitive therapy would be of value to clinicians and researchers weighing this treatment for depression against alternatives such as interpersonal psychotherapy (Klerman *et al.* 1984), which has a stronger research base supporting its social-interpersonal benefits (e.g. Weissman *et al.* 1974, 1981; Mufson *et al.* 1999; O'Hara *et al.* 2000).

Researchers have just begun to address the question of whether social-interpersonal improvement is accounted for by, or independent of, reduction in depressive symptoms. Hirschfeld *et al.* (2002) compared change in social adjustment (on three self-report scales) with change in depressive symptoms (on one clinician-rated scale) in groups with major depressive disorder receiving pharmacotherapy (nefazodone), a newer form of cognitive-behavioral psychotherapy [Cognitive Behavioral Analysis System of Psychotherapy (CBASP); McCullough, 2000], or both treatments. Consistent with past research, depressive symptoms and social adjustment improved more in the combined treatment group than in the two single treatment groups, which did not differ significantly on these outcomes. In addition, social adjustment improved less than, and partly independently of, depressive symptoms.

The current investigation offers several methodological and conceptual strengths to address these questions. First, Jarrett *et al.* (2001) reported that, after response to acute phase cognitive therapy (A-CT; Beck *et al.* 1979), continuation phase cognitive therapy (C-CT; Jarrett & Kraft, 1997; Jarrett *et al.* 1998) reduced depressive relapse and recurrence compared to the assessment-only control in the current sample. In the current report, we utilize Jarrett *et al.*'s clinical trial dataset to evaluate the effects of C-CT on self-reported social-interpersonal functioning. Second, we present follow-up data 12 and 24 months post-A-CT (4 and 16 months post randomization to C-CT or control) to clarify the duration or maintenance of social-interpersonal improvement. Third, we present results for multiple measures of self-reported social-interpersonal functioning (social adjustment, interpersonal problems, and dyadic adjustment). Finally, we consider the relative magnitude and clinical significance of changes in social-interpersonal functioning and evaluate outcomes relative to normative samples.

We hypothesized that social-interpersonal functioning would improve with A-CT, but not as much as depressive symptoms, which are the primary target of A-CT. Similarly, we hypothesized that C-CT would improve social-interpersonal functioning compared to the assessment-only control group. Finally, we hypothesized that improvement in social-interpersonal functioning would be maintained across the follow-up period.

METHOD

Participants

Participants were adult outpatients presenting with DSM-IV non-psychotic, recurrent, major depressive disorder (APA, 1994). Inclusion criteria included clear inter-depressive episode recovery (≥ 2 months of at least nearly normal functioning) and a score ≥ 16 on the 17-item Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960). Exclusion criteria included concurrent medical disorders potentially accounting for depressive symptoms, organic mental disorders, psychotic disorders, active substance abuse or dependence, primary obsessive compulsive or eating disorders, borderline personality disorder and inability or unwillingness to complete questionnaires or to comply with the treatment protocol. Participants were recruited through media, printed announcements and self- and practitioner referral. They completed telephone screening ($n > 3500$), diagnostic interviews ($n = 608$) and provided informed consent to enter the protocol ($n = 156$). More detail about participants, recruitment, inclusion and exclusion criteria are available in Jarrett *et al.* (2001).

Study phases

Acute phase cognitive therapy—Acute phase cognitive therapy (A-CT; Beck *et al.* 1979) was conducted by five experienced therapists within a 12–14 week protocol, including 20 individual sessions (50–60 min) held twice weekly for the first 8 weeks and once weekly for the last 4 weeks. No pharmacotherapy was provided. A-CT is designed to reduce depressive symptoms by eliciting thoughts associated with negative affect, teaching patients to evaluate the validity of such thoughts through logical and empirical methods, to generate more realistic alternatives when negative thoughts are not supported and to employ problem-solving skills when negative conclusions are warranted.

Experimental phase—A-CT responders who completed the post-A-CT assessment and consented to randomization ($n = 84$) were assigned to either continuation phase cognitive therapy (C-CT; Jarrett & Kraft, 1997; Jarrett *et al.* 1998; $n = 41$) or an assessment-only control condition ($n = 43$). The C-CT protocol consisted of ten 60–90 min sessions of C-CT over 8 months (the first four sessions semi-monthly, and the next six sessions monthly) from the same therapist who had provided A-CT. C-CT is designed to prevent relapse and recurrence of depression through maintenance and generalization of skills learned in A-CT, reduction of

residual depressive symptoms and preparation for current or anticipated vulnerabilities. In C-CT, patients are taught to use emotional distress and symptoms as cues to implement skills learned in A-CT. The patients in the assessment-only control attended evaluation visits scheduled at the same frequency as in C-CT. Evaluators of control patients were prohibited from using psychosocial interventions. Patients who relapsed during the experimental phase were asked to complete all sessions and referred for extra-protocol treatment if not receiving C-CT. Data collected after relapse are utilized in this report to increase the generalizability of findings.

Follow-up phase—All 84 patients entering the experimental phase were eligible for, and 74 entered, the follow-up phase.^{1†} This assessment-only period lasted 16 months beyond the experimental phase (24 months post-A-CT) and consisted of 10 sessions scheduled monthly at months 9–12 post-A-CT and bimonthly at months 14–24 post-A-CT. Patients who experienced relapse or recurrence of depression during follow-up were referred for extra-protocol treatment and followed naturalistically; their data are utilized in this report to increase generalizability.²

Assessment strategy and timing

Two pre-treatment assessments were used to establish eligibility for the study and to render diagnoses. Patients presented at the Department of Psychiatry at The University of Texas Southwestern Medical Center at Dallas and completed the Structured Clinical Interview for DSM-III-R (SCID outpatient version; Spitzer *et al.* 1989), with supplemental interview questions to assess DSM-IV disorders and subtypes, as well as other clinician-rated measures described below. The final assessment was conducted by a doctoral-level diagnostician. Inter-episode recovery and A-CT response definitions were chosen for consistency with the DSM (APA, 1994), consensual scientific definitions (Frank *et al.* 1991), and past research (e.g. Jarrett *et al.* 1998,1999,2001). Specifically, inter-episode recovery was defined as a return to more-or-less normal functioning for 2 or more months between major depressive episodes; and response was defined as not meeting criteria for current DSM major depressive disorder and an HRSD score of 9 or less when exiting or completing the A-CT protocol. Measures used in this report were completed: before A-CT session 1 (or at pre-treatment), at A-CT sessions 9 and 17; post-A-CT/pre-experimental phase (C-CT or assessment-only control); before experimental session 6; post-experimental phase; and 12 and 24 months post-A-CT (4 and 16 months post-experimental phase). As shown in Table 1, the sample size available for analysis varied due to missing data, attrition and the measure (i.e. participants not in committed, cohabitating romantic relationships did not complete the measure of dyadic adjustment described below).

Measures

Hamilton Rating Scale for Depression—The Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) is a widely used, 17-item, clinician rating scale to assess severity of depressive symptoms. Scores range from 0 to 52, and higher values represent greater depressive symptoms. The scale has demonstrated good inter-rater reliability ($r=0.85$; Clark & Watson,

¹Jarrett et al. (2001) reported $n=60$ entering the follow-up phase because the data from 14 patients who met criteria for relapse or recurrence of DSM-IV major depressive disorder during the experimental phase were censored. In the current analyses, all available data, including those collected after relapse or recurrence, were utilized to maximize the generalizability of findings.

[†]The notes will be found on p. 656.

1991), adequate internal consistency (alphas of 0.88 and 0.89 in two large clinic samples; Rush *et al.* 1996), and appropriate convergence with self-report depressive symptom measures ($r=0.70-0.83$; Clark & Watson, 1991). In the current sample, alpha internal consistency was adequate (median=0.85, range=0.73–0.90) with the exception of the pre-A-CT assessment (0.34). However, because the pre-A-CT HRSD correlated highly (0.72) with the clinician version of the Inventory for Depressive Symptomatology (Rush *et al.* 1986,1996; described below), we retained this data point.

Beck Depression Inventory—The Beck Depression Inventory (BDI; Beck *et al.* 1961) is a very widely used, 21-item, self-report measure of depressive symptom severity. Scores range from 0 to 63, and higher values represent greater depressive symptoms. Beck *et al.* (1988) reported an average internal consistency of 0.87, an average short-term (<1 month) retest reliability of 0.60 and considerable convergence with clinical ratings of depressive symptoms, the HRSD and other self-report measures of depressive symptoms. In the current sample, alpha internal consistency was good to excellent (median=0.92, range 0.85–0.95).

Inventory for Depressive Symptomatology—This 28-item scale (Rush *et al.* 1986, 1996) has both self-report (IDSR) and clinician (IDSC) versions to measure the severity of depressive symptoms. Scores range from 0 to 84, and higher values represent greater depressive symptoms. Rush *et al.* (1986) reported internal consistency reliabilities of 0.85 (IDSR) and 0.88 (IDSC), as well as moderate to high convergence with the BDI (IDSR $r=0.78$; IDSC $r=0.61$) and HRSD (IDSR $r=0.67$; IDSC $r=0.92$). In the current sample, alpha internal consistency was moderate to high for both the IDSC (median=0.89, range=0.61–0.94) and the IDSR (median = 0.90, range=0.76–0.93).

Social Adjustment Scale – Self Report—The Social Adjustment Scale – Self Report (SAS-SR; Weissman & Bothwell, 1976) is a 56-item self-report measure of functioning in several important social domains. Participants complete only those sections of the questionnaire reflecting their social roles (e.g. not all participants complete marital or parenting sections). Scores range from 1 to 5 and higher values represent poorer adjustment. In past research, internal consistency for the overall adjustment score was moderate (alpha=0.74) and temporal stability was good ($r=0.80$) across 2-week intervals (Edwards *et al.* 1978). Validity evidence includes appropriate patterns of mean differences, significant correlations with clinical ratings, and sensitivity to change in psychopathology (Weissman & Bothwell, 1976; Weissmann *et al.* 1978). In the current sample, alpha internal consistency was good (median=0.85, range=0.80–0.90).

Inventory of Interpersonal Problems—The Inventory of Interpersonal Problems (IIP; Horowitz *et al.* 1988) is a 127-item self report scale of the extent to which a number of behaviors, thoughts and feelings have been problematic in one's significant relationships. Scores range from 0 to 4 and higher values represent greater interpersonal problems. Horowitz *et al.* (1988) provide evidence of the measure's reliability and validity, including a 10-week retest correlation of 0.98, moderate correlations with measures of psychiatric symptoms, and mean score decreases with psychotherapy. In the current sample, alpha internal consistency for the total score was very high (median=0.98, range=0.97–0.98), due in part to the large number of items. Consequently, we note that the average inter-item correlation also suggested adequate internal consistency (median=0.28, range=0.18–0.32).

²The Longitudinal Interval Follow-up Evaluation structured interview (Keller *et al.* 1987), was administered 4, 8, 12 and 24 months post-ACT, respectively, and indicated that 15.4, 27.8, 38.9 and 44.1% of the assessment-only group, and 10.3, 7.9, 25.0 and 41.9% of the C-CT group, reported receiving extra-protocol treatment (i.e. pharmacotherapy and/or psychotherapy) in the interval since the previous assessment.

Dyadic Adjustment Scale—The Dyadic Adjustment Scale (DYS; Spanier, 1976) is a 32-item, self-report inventory of positive adjustment and satisfaction in marital and similarly committed dyads. Scores range from 0 to 151, and higher values represent better adjustment. Although it is possible to derive subscales, most clinicians use the total score as a reflection of overall relationship quality (Spanier & Thompson, 1978). Spanier (1976) reported an internal consistency of 0.96, as well as evidence for both content and criterion-related validity. In the current sample, alpha internal consistency was also quite high (median =0.96, range=0.95–0.97).

Standardization of scores

To facilitate examination of changes in and among measures of social-interpersonal functioning, and comparisons of these changes with depressive symptoms, individual scales were placed on a common metric. All available cases at the pre-A-CT assessment were used to standardize measures at all assessments into *T*-score units ($M=50$, $S.D.=10$). The formula (e.g. see Minium *et al.* 1993) used to convert a person's raw score x into a *T* score was:

$$x_T = \frac{x_{\text{raw}} - \bar{x}_{\text{pre-treatment}}}{S.D._{\text{pre-treatment}}} \times 10 + 50.$$

This linear transformation does not alter the significance of statistical tests of change within measures (e.g. pre- versus post-treatment) but aids understanding of the magnitude of changes. After scale-level standardization, the four depressive symptom measures (BDI, HRSD, IDSC, IDSR) were averaged to form a single index and again standardized to maintain $S.D.=10$ pre-A-CT. Averaging the four depression symptom reduced the number of statistical analyses and was justified empirically by cross-time factor analyses of the current data set (Vittengl, J. R., *et al.* unpublished observations) which indicated that the scales aggregated strongly by time (e.g. pre-treatment, post-treatment) rather than by method (self- or clinician-report) or measure. Similarly, alpha internal consistency for the 4-item depressive symptom index was high (median=0.95, range=0.89–0.97). Although the primary analyses utilize the standardized measures, Table 1 contains descriptive statistics for the raw scales at each assessment.

Identification of healthy participants

Identification of participants in the 'healthy' range of social-interpersonal functioning was based on a cut-off of 1.28 S.D. from the mean of best available (although not matched) normative samples (i.e. about 10% of the population would be considered unhealthy). This value represents a compromise between the traditional cut-off of 2 S.D. (i.e. about 2% of the population would be considered unhealthy; see e.g. Jacobson & Truax, 1991) and evidence that psychopathology with attendant social-interpersonal dysfunction is more prevalent than 2% in epidemiological samples (e.g. Kessler & Zhao, 1999, reported a 12-month prevalence of about 31% for any disorder; Fredman *et al.* 1988, reported a 2-week prevalence of about 9% for any disorder). For the DYS, Spanier's (1976) norms for married couples were employed. For the IIP, norms from a community sample nominated as mentally healthy by licensed psychologists (and so likely 'super normal') were pooled with an identically sized sample of college students (both datasets from Hansen & Lambert, 1996). Although not ideal demographically, the item mean of this pooled sample did not differ significantly from that of a US census-stratified sample completing an IIP short form (Horowitz *et al.* 2000). Finally, norms from a large community sample (Weissman *et al.* 1978) were available for the SAS-SR. Examination of stricter and more lenient health cut-offs, as well as score distributions, suggested that there were no clear health categories in the current sample, but the 1.28 S.D. cut-off provided heuristically valuable results.

Hypothesis-testing strategy

We based our primary hypothesis tests on ordinary least squares regression and analysis of variance. Linear mixed-effect models using likelihood estimation are becoming increasingly popular for analyzing datasets with similar structures, and offer power and interpretive advantages in some cases (e.g. Nich & Carroll, 1998;Kreft, 2000;Wallace & Green, 2002). In our dataset, however, linear mixed-effect analyses with the social-interpersonal measures yielded substantively equivalent results. Consequently, we present only the more widely understood ordinary least squares analyses here. In addition, due to the use of multiple measures and analyses, we selected a conservative alpha of 0.01, 2-tailed, for significance in all statistical tests and we focus on effect sizes and patterns of results in drawing conclusions. Effect sizes were computed with Cohen's (1988) formulas and included (benchmarks for small, medium and large effects) r for bivariate correlation (0.10, 0.30, 0.50), d for t tests (0.20, 0.50, 0.80), and f for analysis of variance (0.10, 0.25, 0.40).

RESULTS

Sample characteristics

The sample entering A-CT consisted of 155 adult outpatients with DSM-IV recurrent major depressive disorder, including 74.2% females (the intention-to-treat sample of 156 included 1 participant who consented to A-CT but did not begin treatment). The mean age was 41.3 years (S.D.=11.0); the mean level of education was 15.4 years (S.D.=2.8); and 7.1% were African American, 4.5% Hispanic, 1.3% Native American and 87.1% White. The participants' mean age of onset of major depressive disorder was 19.9 years (S.D.=9.6), and participants had experienced a mean of 3.4 major depressive episodes (S.D.=1.3). Prior treatment exposure data for participants' first, most recent two and current major depressive episodes, indicated that 1.9% had been treated previously with electro-convulsive therapy, 56.8% with pharmacotherapy, 59.4% with psychotherapy and 41.3% with at least two of these types of therapy. In addition to the diagnosis of recurrent major depressive disorder, the number of DSMIV Axis I disorders pre-A-CT ranged from 0 to 4 ($M=0.59$; S.D.=0.78). Co-morbid Axis I disorders included social phobia (20.0%),³ specific phobias (12.3%), panic disorder without agoraphobia (8.4%), post-traumatic stress disorder (7.7%), dysthymic disorder (5.2%), obsessive-compulsive disorder (1.3%), panic disorder with agoraphobia (1.3%) and 0.6% each of agoraphobia without a history of panic disorder, attention deficit hyperactivity disorder, bulimia nervosa and hypochondriasis.

Previous outcome analyses with the current dataset

Jarrett *et al.* (2001) present greater detail about treatment outcome in the current dataset. Using the intention-to-treat sample ($n=156$), the response rate to A-CT when exiting A-CT was 62.6% ($n=97$) as rated by the therapist (when the participant attrited; $n=10$) or by an independent clinician (when the participant completed the post-A-CT assessment; $n=87$).⁴ These data are consistent with the research showing that A-CT reduces the symptoms of major depressive disorder in adults (e.g. Rush *et al.* 1977;Hollon *et al.* 1992;Jarrett *et al.* 1999). In addition, C-

³Substantial co-morbidity of anxiety and unipolar mood disorders is common (e.g. Mineka et al. 1998). However, co-morbid social phobia was potentially problematic given our focus on social-interpersonal functioning. The subsample with social phobia had poorer social adjustment, more interpersonal problems, and greater depressive symptoms pre-A-CT ($p<0.01$), as one would expect, but dyadic adjustment did not differ. By 0 months post-A-CT and throughout the experimental and follow-up phases, however, the sub-sample with an initial diagnosis of social phobia did not differ significantly from the subsample not co-morbid for social phobia ($p>0.05$). Consequently, co-morbid social phobia did not interfere with hypothesis tests.

CT reduced relapse and recurrence of DSM-IV major depressive disorder over 8 months post-A-CT compared to the assessment-only control (10% v. 31%) in the current dataset.

Changes in social-interpersonal functioning across A-CT

Fig. 1 depicts changes in the standardized SASSR, IIP, DYS and depressive symptom scores, using all available data at each assessment (see Table 1 for raw M and n). All changes (decreases) in the standardized scores represent improved social-interpersonal functioning relative to the pre-A-CT distributions (i.e. decreases in the SAS-SR represent improved social adjustment; and the DYS scale scores have been reflected such that decreases represent improved dyadic adjustment in contrast to the increasing raw score means shown in Table 1). The difference between the first and the last available A-CT assessments, regardless of therapy completion (i.e. 155 individuals began A-CT, 130 completed the A-CT protocol and 128 also completed the post-A-CT assessment) was used to compute effect sizes for overall change in measures. The DYS improved a small amount [$d=0.47$; $t(90)=4.53$, $p<0.0001$, 2-tailed], the IIP [$d=0.91$; $t(146)=11.01$, $p<0.0001$, 2-tailed] and SAS-SR [$d=1.19$; $t(151)=14.67$, $p<0.0001$, 2-tailed] improved a large amount and depressive symptoms improved by a very large amount [$d=1.55$; $t(154)=19.31$, $p<0.0001$, 2-tailed]. Moreover, the standardized depressive symptom index had decreased more than the three social-interpersonal measures at the session 9, session 17 and 0 months post-A-CT assessments, dependent $t(67-135)>15.11$, $p<0.0001$, 2-tailed, median $d=1.59$ (range 1.41–2.20). However, depressive symptoms improved mostly early in treatment with no significant change between session 17 and 0 months post-A-CT.

Social-interpersonal ‘health’ before and after A-CT

The first and last available A-CT assessments were used to calculate proportions in the estimated ‘healthy’ range of social-interpersonal functioning based on a cutoff of 1.28 S.D. from the mean of available normative samples (i.e. about 10% of the population would be considered unhealthy). Estimated proportions of healthy participants are depicted in Fig. 2. The three social-interpersonal measures yielded quite similar healthy proportions at exit (60–65%), and each increased significantly from pre-A-CT ($p<0.003$, 2-tailed, by McNemar’s test). Although the available normative samples were not matched to one another or to the current sample of depressed patients, results were parallel to continuous measures: The smallest change was in marital discord (DYS) and the greatest was in social role functioning (SAS-SR).

Correlated change in depressive symptoms and social-interpersonal functioning during A-CT

Because social-interpersonal functioning changed less than depressive symptoms, regressions were computed to determine to what degree changes in social-interpersonal functioning could be accounted for by change in depressive symptoms and vice versa. Specifically, change (pre-A-CT minus last A-CT assessment) in each social-interpersonal measure was predicted by change in depressive symptoms. From these regression equations, the t test for nonzero intercept reflects systematic change in social-interpersonal functioning independent of change in depressive symptoms. In each model, change in depressive symptoms was correlated moderately to highly with change in social-interpersonal functioning ($r=0.36$, 0.57 and 0.72 , for the DYS, IIP and SAS-R, respectively, $p<0.0005$, 2-tailed), but there was no significant change in social-interpersonal functioning independent of change in depressive symptoms (converting t to r for effect sizes comparable to the correlations above, $r=0.06$, 0.01 and 0.05 for the DYS, IIP and SAS-SR, respectively, $p>0.51$, 2-tailed). Conversely, in a second set of

⁴Jarrett et al. (2001) reported $n=87$ treatment responders, according to ratings made by an independent evaluator, and consistent with their focus on participants eligible to enter C-CT (i.e. those who completed the A-CT protocol and the post-treatment assessment, in addition to the absence of DSM-IV major depressive disorder and an HRSD score of 9 or less).

regressions in which change in depressive symptoms was predicted by change in the social-interpersonal measures, depressive symptoms changed partly independently of the social-interpersonal measures singly (converting from t , $r=0.81$, 0.69 and 0.55 , for prediction by the DYS, IIP and SAS-SR, respectively, $p<0.0001$, 2-tailed) and collectively (converting from t , $r=0.48$, $p<0.0001$, 2-tailed).

Our finding that social-interpersonal improvement was accounted for by change in depressive symptoms appeared to contradict Hirschfeld *et al.*'s (2002) recent report that social adjustment, as measured by the SAS-SR, improved partly independently of depressive symptoms, as measured by the HRSD. To help understand this difference, an additional series of regressions was run to predict change in the social-interpersonal measures (SAS-SR, IIP, DYS) from change in depressive symptom measures (HRSD, BDI, IDSC, IDSR) individually, instead of our multi-measure/multi-method depressive symptom index. In these regressions, the SAS-SR changed partly independently of the HRSD ($r=0.22$, $p=0.0067$, 2-tailed), in replication of Hirschfeld *et al.* However, all other pairings of social-interpersonal and depressive symptom measures left no significant independent social-interpersonal change ($p>0.05$, 2-tailed).

Differentiation of A-CT responders' and non-responders' social-interpersonal functioning

To understand better the relations between depressive symptoms and social-interpersonal change, we compared A-CT responders' (absence of major depressive disorder and an HRSD score of 9 or less when exiting A-CT; $n=97$) and non-responders' ($n=58$) social-interpersonal functioning across A-CT. Plots of responders' and non-responders' standardized scores are shown in Fig. 3. As these plots suggest, responders and non-responders did not differ significantly at the pre-A-CT assessment on the SAS-SR or IIP ($p>0.10$, 2-tailed), but there was a trend for better pre-A-CT functioning in responders on the DYS, $t(89)=2.35$, $p=0.021$, 2-tailed, $d=0.52$. Consequently, pre-A-CT scores were controlled in analyses of covariance comparing responders and nonresponders at later assessments for each measure. Responders showed better social-interpersonal functioning at the later A-CT assessments on the SAS-SR (session 9, session 17 and post-A-CT) and the IIP (post-A-CT), $F(1, 115-132)>19.87$, $p<0.0001$, median $f=0.66$ (range $0.39-0.92$). For the DYS, responders and non-responders did not differ at A-CT session 9 ($p=0.32$), but responders functioned better at A-CT session 17, $F(1, 72)=7.64$, $p=0.0037$, $f=0.33$ and marginally better post-A-CT, $F(1, 63)=4.51$, $p=0.038$, $f=0.27$.

Changes in social-interpersonal functioning across experimental and follow-up phases

Differences between the C-CT and control groups on the SAS-SR, IIP and DYS were evaluated pre-C-CT (0 months post-A-CT), at C-CT session 6 (4 months post-A-CT), post C-CT (8 months post-A-CT), 4 months post CCT (12 months post-A-CT) and 16 months post C-CT (24 months post-A-CT), but no significant differences were detected ($p>0.05$, 2-tailed, with pairwise deletion of cases with missing data using t tests at each assessment; with listwise deletion of cases with missing data using repeated-measures ANOVAs; and with all cases having at least one non-missing assessment included in linear mixed-effect analyses). However, the C-CT group had numerically (but not statistically significantly) better average functioning than the control group on the SASSR, IIP and DYS at the 4, 6, 8, 12 and 24 month assessments (with the exception of the DAS at the 4 month post-A-CT assessment) suggesting that the lack of statistically significant effects might relate to low statistical power. Conversely, the observed effect sizes for C-CT were typically small for the SAS-SR, IIP and DYS, at the four post-A-CT assessments (median $d=0.34$, range $=x0.11-0.66$), suggesting that clinical significance would be marginal, even if a larger sample had supported statistical significance. Based on these findings, Fig. 4 depicts standardized means after pooling the C-CT and control groups (see Table 1 for raw M). Two conclusions are evident in this sample of A-CT responders.

First, social-interpersonal functioning was relatively stable, with no notable further improvement in functioning across the experimental or follow-up phases of the study. Second, there was a clear ordering of these variables with each maintaining its level of improvement relative to pre-A-CT scores and depressive symptoms.

DISCUSSION

The results of this study supported our first hypothesis that social-interpersonal functioning would improve after A-CT in a sample of adult outpatients with recurrent major depressive disorder. Three major domains of self-reported social-interpersonal functioning – social adjustment, interpersonal problems, and dyadic adjustment – improved significantly across A-CT; however, none improved as much as depressive symptoms relative to the pre-A-CT distribution. Similarly, effect sizes indicated a substantial decrease in depressive symptoms, less substantial but clinically significant improvement in social adjustment and interpersonal problems, and statistically, but likely less clinically, significant improvement in dyadic adjustment.

Regarding potential mechanisms for improvement, social-interpersonal improvement was largely accounted for by change in depressive symptoms across A-CT. In contrast, Hirschfeld *et al.* (2002) found that social adjustment, measured by the SAS-SR, improved partly independently of depressive symptoms, measured by the HRSD. In our data set, too, when only the clinician-rated HRSD was controlled, as opposed to our multi-measure/multi-method depressive symptom index, the SAS-SR changed partly independently. This significant independent change in a social-interpersonal measure was unique to the pairing of the HRSD with the SAS-SR, however; there was no significant independent change in the DYS or IIP when controlling the HRSD and no significant independent change in any of the social-interpersonal measures when controlling the other depressive symptom measures (BDI, IDSC, IDSR) individually, given the current moderate sample size. Consequently, we speculate that the HRSD taps aspects of depressive symptoms less overlapping with social adjustment than other commonly used depressive symptom measures. Further research involving multiple measures of both depressive symptoms and specific social-interpersonal constructs would help clarify this fundamental issue.

Our finding that most social-interpersonal improvement was accounted for by reduction in depressive symptoms is consistent with past research, including equivalent social adjustment outcomes among cognitive therapy, interpersonal therapy, imiprimine plus clinical management, and pill placebo plus clinical management groups in the NIMH Treatment of Depression Collaborative Research Program (Imber *et al.* 1990); and pharmacotherapy alone improving social adjustment (e.g. Kocsis *et al.* 1997). Consequently, we speculate that focusing primarily on depressive symptom reduction in A-CT does not detract from, and may even promote, improvement in social adjustment. Moreover, the current results are consistent with amelioration of depressive symptoms leading to improved social-interpersonal functioning, an idea that corresponds with the complex social-behavioral impairments in depression (e.g. problems in speech content and style, facial expression and gaze, and bodily posture and gestures; Segrin, 2000). Data structures allowing fine-grained time-lagged analyses (e.g. social-interpersonal functioning and depressive symptoms assessed at every therapy session) would be useful in testing potential causal relations among these constructs.

Dyadic adjustment changed relatively little across A-CT, compared to greater change in social adjustment and interpersonal problems. We speculate that dyadic adjustment improved less because it was less strongly associated with depressive symptom severity, which accounted for all significant change in social-interpersonal functioning and because it was less

normatively impaired pre-A-CT, which left less room for improvement. This interpretation is consistent with past research. For example, the current sample's DYS scores were similar to other depressed samples pre-A-CT and to remitted samples post-A-CT (e.g. Dobson, 1987). Moreover, the magnitude of association between dyadic adjustment and depressive symptom severity was consistent with previous reports (e.g. Olin & Fenell, 1989). Our finding of less improvement in dyadic adjustment also may reflect the fact that relationship partners did not participate in the therapy protocol. This is consistent with past research suggesting that behavioral marital (involving both partners), but not cognitive (involving only one partner), therapy for depression improves dyadic adjustment (Jacobson *et al.* 1991; Beach & O'Leary, 1992). We speculate that substantial improvement in dyadic adjustment often requires a treatment targeting relationship satisfaction and involving both partners.

Our second hypothesis that C-CT would further improve social-interpersonal functioning after A-CT was not supported. For those responding to A-CT, who demonstrated larger gains in social-interpersonal functioning than non-responders, C-CT did not significantly enhance social-interpersonal functioning compared to an assessment-only control. In contrast, C-CT does appear helpful in reducing risk of relapse of major depressive disorder over 8 months post-A-CT (Jarrett *et al.* 1998,2001). Because our data suggest that depressive symptoms change partly independently of social-interpersonal functioning, we speculate that C-CT's power to reduce relapse does not generalize to substantial social-interpersonal benefits. Instead, the potential gains in social-interpersonal functioning amenable to cognitive therapy may occur in a 20-session course of A-CT, leaving little room for additional improvement with C-CT. At the same time, we note that approximately 35–40% of the sample entering C-CT needed some yet-to-be-identified intervention to reach the estimated healthy range of social-interpersonal functioning.

Consistent with our third hypothesis, gains in social-interpersonal functioning were maintained across the follow-up period. Both at 12 and 24 months post-A-CT, SAS-SR, IIP and DYS scores were similar to previous assessments at 0, 4 and 8 months post-A-CT. That is, the relatively large improvements in social adjustment and interpersonal problems, and small improvements in dyadic adjustment, were maintained across a 2-year period. These data demonstrate for the first time the long-term maintenance of positive social-interpersonal outcomes of A-CT, as well as add to the large database supporting A-CT's efficacy in reducing depressive symptoms (e.g. Jarrett & Rush, 1994; Craighead *et al.* 1998; Strunk & DeRubeis, 2001).

The current study involves noteworthy limitations. Perhaps most importantly, changes in social-interpersonal functioning with A-CT were not compared with no treatment, a waitlist control, or pharmacotherapy. Consequently, improvement in social-interpersonal functioning cannot be strongly attributed to A-CT relative to a comparison condition. Research demonstrating the efficacy of A-CT in reducing depression in controlled trials (e.g. Jarrett *et al.* 1999) may reduce, but cannot eliminate, this limitation of the current design.

Moreover, social-interpersonal functioning was assessed only by self-report. Consequently, changes in social-interpersonal functioning may represent subjective experiences rather than independently observable behavioral change. Research documenting the correspondence of the current social-interpersonal measures with others' ratings of the same constructs (e.g. Weissman & Bothwell, 1976; Horowitz *et al.* 1988; Dudek *et al.* 2001; Ready & Clark, 2002) lessens but does not eliminate this concern. Future research using additional methods to assess social-interpersonal functioning (e.g. collateral reports; behavioral observations) would make valuable contributions.

Finally, it is possible that a shared negative affectivity component accounts for observed associations among social-interpersonal and depressive symptoms measures (e.g. see Watson & Clark, 1984). Although negative affectivity has been separated from the unique components of depressive symptoms (e.g. Clark & Watson, 1991; Watson *et al.* 1995), comparable work remains for social-interpersonal functioning measures. Discriminant validity in measurement would be an asset in future investigations of concomitant change in depressive symptoms and social-interpersonal functioning. However, elsewhere (Clark *et al.* 2003) we present analyses from the current sample indicating that the overlap among the SAS-SR and IIP and additional psychosocial measures, which related strongly to a measure of trait negative affectivity, is more predictive of depressive symptoms than unique components of the measures. Consequently, efforts to isolate highly discriminant components of social-interpersonal measures may prove challenging.

The maintenance of gains in social-interpersonal functioning across 2 years clarifies the potential benefit of A-CT for individuals with recurrent major depressive disorder. Further, the finding that C-CT did not further improve social-interpersonal functioning suggests that the potential social-interpersonal benefits may be achieved over a typical course of A-CT. Additional research is necessary to conclude definitively that A-CT improves social-interpersonal functioning, and that this improvement is governed by changing depressive symptoms. In future research aimed at clarifying the meaning and causes of social-interpersonal improvements, the results of the current study further highlight the value of multi-measure assessment of the complex constructs of depressive symptoms and social-interpersonal functioning.

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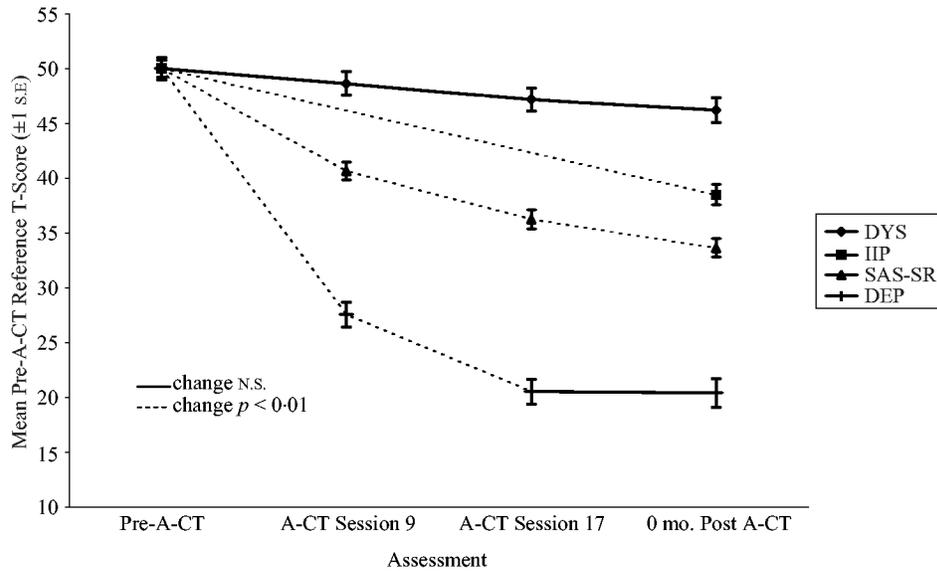


Fig 1. Changes in standardized social-interpersonal and depressive symptoms scales during acute phase cognitive therapy (A-CT) represent improved functioning. DYS, dyadic adjustment scale; IIP, Inventory of Interpersonal Problems; SAS-SR, Social Adjustment Scale – Self Report; DEP, Depressive symptoms index formed from two self-report and two clinician-report scales (see text for details); mo., months.

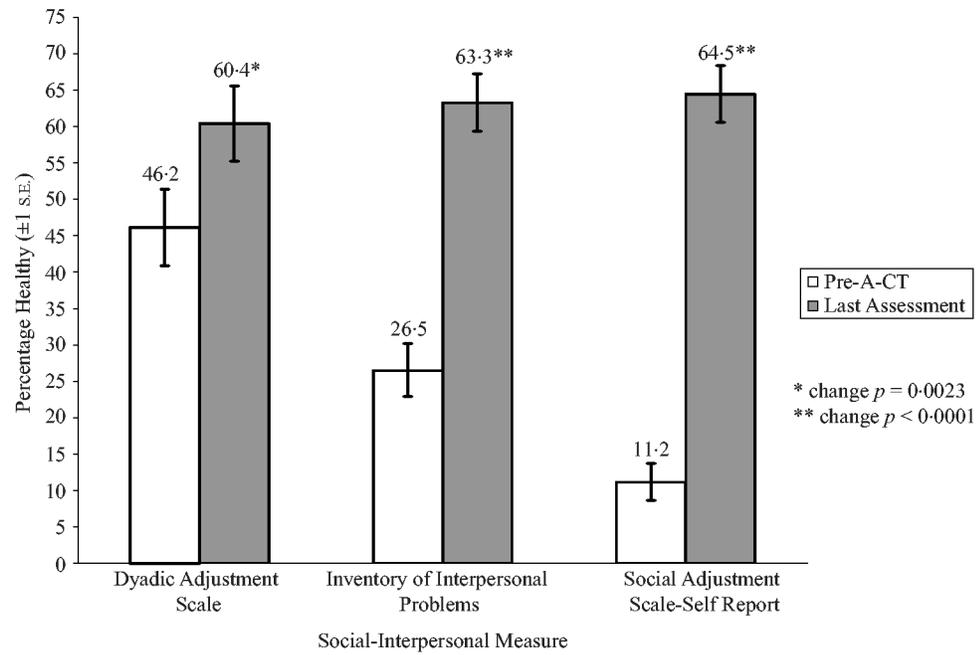


Fig 2. Percentage of social-interpersonally healthy individuals (at or below the 90th percentile of dysfunction in a normative sample) entering and exiting acute phase cognitive therapy (A-CT).

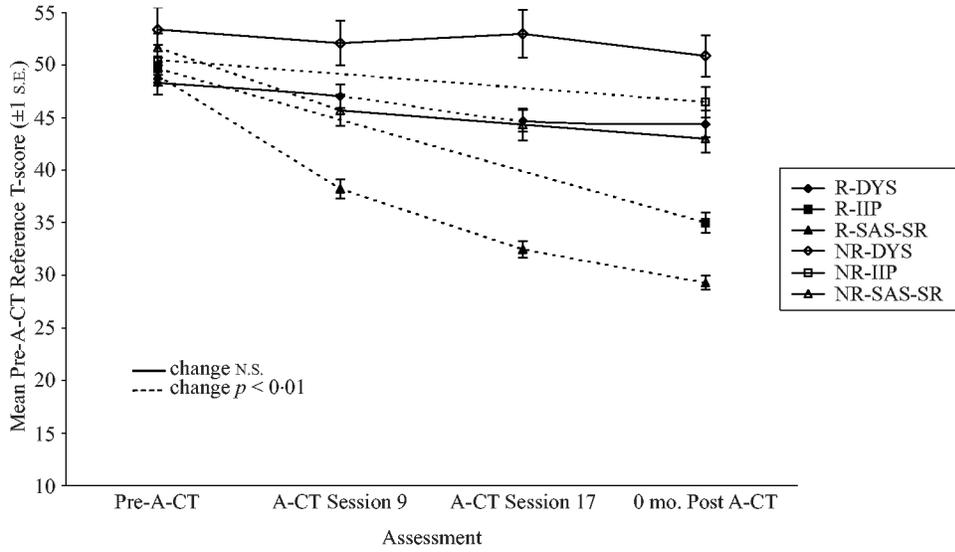


Fig 3. Acute phase cognitive therapy (A-CT) responders' (R) and non-responders' (NR) standardized social-interpersonal functioning and depressive symptoms. DYS, dyadic adjustment scale; IIP, Inventory of Interpersonal Problems; SAS-SR, Social Adjustment Scale – Self Report; DEP, Depressive symptoms index formed from two self-report and two clinician-report scales (see text for details); mo., months.

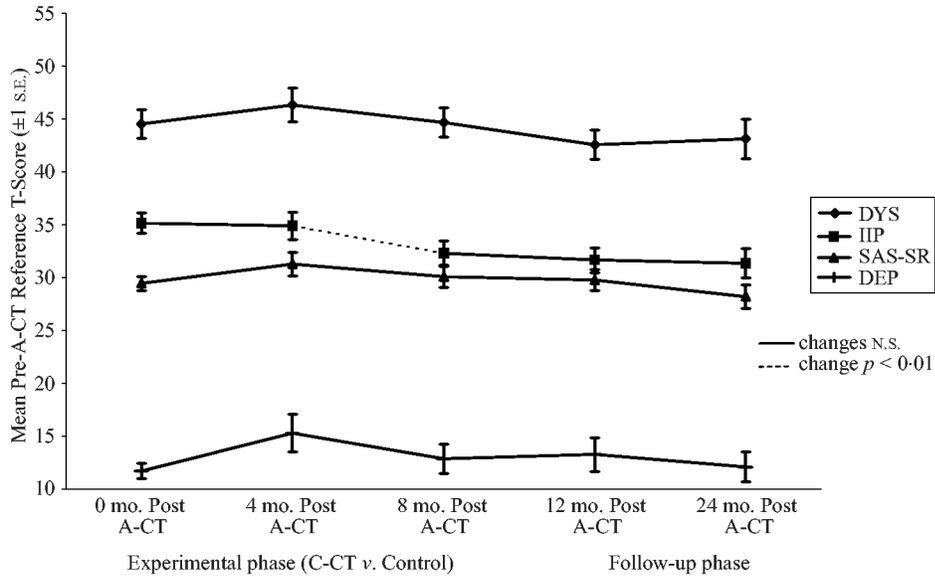


Fig 4. Social-interpersonal functioning after acute phase cognitive therapy (A-CT) for pooled continuation phase cognitive therapy (C-CT) and control groups. *DYS*, dyadic adjustment scale; *IIP*, Inventory of Interpersonal Problems; *SAS-SR*, Social Adjustment Scale – Self Report; *DEP*, Depressive symptoms index formed from two self-report and two clinician-report scales (see text for details); mo., months.

Table 1.
Raw scale score descriptive statistics at each assessment

Scale	Acute Phase Cognitive Therapy (A-CT)					Continuation Phase Cognitive Therapy (C-CT) or control			Post-A-CT follow-up	
	Pre. *	Sess. 9	Sess. 17	Post.	Exit	Pre.	Sess. 6	Post.	12 mo.	24 mo.
BDI										
M	24.78	13.57	9.23	7.57	9.35	3.81	6.30	4.79	5.27	3.81
S.D.	8.04	8.79	7.77	7.37	8.87	4.35	9.17	6.51	6.56	6.41
n	152	137	132	126	154	84	77	71	63	56
HRSD										
M	18.41	8.91	6.64	7.42	8.22	3.62	5.31	4.12	4.63	4.06
S.D.	3.83	4.90	5.05	6.45	6.88	2.85	6.62	5.39	5.67	4.77
n	155	138	135	128	155	84	78	74	71	63
IDSC										
M	33.14	17.38	12.66	13.12	14.60	6.31	8.87	7.21	7.35	6.52
S.D.	7.41	10.01	9.99	11.44	12.41	5.05	11.18	9.06	8.56	7.60
n	155	138	135	128	155	84	78	73	71	63
IDSR										
M	37.47	21.13	14.77	13.02	15.40	7.63	9.45	8.79	8.55	6.46
S.D.	9.23	11.78	10.98	10.62	12.67	6.41	10.73	9.02	7.73	7.05
n	151	137	133	126	154	84	77	71	62	56
SAS-SR										
M	2.52	2.12	1.93	1.82	1.92	1.64	1.72	1.67	1.66	1.59
S.D.	0.43	0.41	0.43	0.40	0.51	0.26	0.43	0.35	0.33	0.34
n	152	136	132	126	152	84	76	69	63	52
IIP										
M	1.62	—	—	1.01	1.15	0.83	0.82	0.68	0.65	0.63
S.D.	0.53	—	—	0.55	0.64	0.48	0.60	0.51	0.50	0.52
n	147	—	—	122	147	83	76	69	62	51
DYS										
M	88.24	91.65	95.27	97.69	94.37	101.96	97.45	101.57	106.82	105.46
S.D.	24.97	24.55	23.57	23.33	27.61	23.02	26.17	21.50	20.20	24.59
n	91	86	77	68	91	47	44	37	34	28

* Pre., pre-treatment; Sess., session; Post., post-treatment; Exit, last available data point used in calculation of effect size and health statistics; Mo., months after completing A-CT; BDI, Beck Depression Inventory; HRSD, Hamilton Rating Scale for Depression; IDSC, Inventory of Depressive Symptomatology (clinician-report); IDSR, Inventory of Depressive Symptomatology (self-report); SAS-SR, Social Adjustment Scale – Self Report; IIP, Inventory of Interpersonal Problems; DYS, Dyadic Adjustment Scale; Follow-ups occurred 12 and 24 months post-A-CT, equivalent to 4 and 16 months post C-CT or assessment-only control.