Behaviour Research and Therapy 49 (2011) 875–884

Contents lists available at SciVerse ScienceDirect

Behaviour Research and Therapy

journal homepage: www.elsevier.com/locate/brat

Comorbid social anxiety disorder in clients with depressive disorders: Predicting changes in depressive symptoms, therapeutic relationships, and focus of attention in group treatment

Todd B. Kashdan [a,*], John E. Roberts [b]

[a] Department of Psychology, MS 3F5, George Mason University, Fairfax, VA 22030, United States
[b] University at Buffalo, State University of New York, Buffalo, NY, United States

ARTICLE INFO

Article history:
Received 7 February 2011
Received in revised form
25 September 2011
Accepted 4 October 2011

Keywords:
Depression
Social phobia
Comorbidity
Treatment
Self-focus
Therapeutic alliance
Attachment

ABSTRACT

The current study examined whether depressed outpatients with comorbid SAD respond differently to a cognitive-behavioral group intervention and if so, how and why. Using growth curve modeling, we found evidence that depressed clients with comorbid SAD had rapid improvement in depressive symptoms over the course of treatment and generally did not differ from those without comorbidity in developing close therapeutic relationships and modifying the direction of attentional focus away from the self. Non-linear effects demonstrated that rates of change in depressive symptoms, relationship variables, and focus of attention, were most rapid early in treatment. In contrast to hypotheses, trajectories of change in therapeutic relationships and attentional focus did not mediate the effect of SAD on treatment improvement in depressive symptoms. These findings suggest that comorbid SAD does not have a detrimental effect on the course of depression treatment and group-based treatments can be as beneficial for depressed individuals with comorbid SAD. It may be that group-based treatments for depression provide explicit opportunity for emotional processing in social situations (i.e., exposure) and hence mimic efficacious therapies for SAD.

Although cognitive-behavioral therapy (CBT) is an efficacious intervention for depression, data suggest that between 20 and 64% of clients treated for depression fail to recover (e.g., Brown & Lewinsohn, 1984; Peterson & Halstead, 1998). Given these findings, it seems important to explore individual difference factors and change process variables that enable or thwart treatment impact. Some of the most replicated predictors of client changes in CBT for depression include comorbid anxiety disorders (e.g., Brown, Schulberg, Madonia, Shear, & Houck, 1996; Sherbourne & Wells, 1997), therapist–client interpersonal processes (e.g., Feeley, DeRubeis, & Gelfand, 1999; Persons & Burns, 1985), client perceptions of group cohesion (Hoberman, Lewinsohn, & Tilson, 1988), and interpersonal impairment (Sotsky et al., 1991). The foregoing constructs possess theoretical and empirical ties to social anxiety disorder (SAD). We explored how SAD is relevant to depressive symptom change across the course of group cognitive-behavioral therapy for depression.

Comparing social anxiety and depression

For people meeting criteria for SAD during their lifetime, rates of comorbid major depressive disorder range from 20 to 37% (Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996; Merikangas & Angst, 1995). In an examination of over 1000 clients seeking treatment in anxiety disorder clinics (Brown, Campbell, Lehman, Grisham, & Mancill, 2001), 48% with principal diagnoses of SAD met criteria for comorbid major depressive disorder; 56% with a principal diagnosis of major depressive disorder met criteria for comorbid SAD. While these two disorders often co-occur, their convergence is often neglected and undertreated.

Both depression and SAD are characterized by chronic, excessive self-focused attention to negative stimuli (Clark & Wells, 1995; Ingram, 1990), an affective profile of intensified negative emotions and attenuated positive emotions (Kashdan, 2007; Kashdan, Weeks, & Savostyanova, 2011), and inhibited behaviors such as avoidance and unassertiveness (e.g., Eng, Heimberg, Hart, Schneier, & Liebowitz, 2001; Gotlib & Meltzer, 1987). Despite similarities, there are clinical features specific to SAD. According to cognitive models (Rapee & Heimberg, 1997), people with SAD disproportionately allocate attentional resources to negative self-

* Corresponding author. Tel.: +1 703 993 9486; fax: +1 703 993 1359.
E-mail address: tkashdan@gmu.edu (T.B. Kashdan).

0005-7967/$ – see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.brat.2011.10.002

appraisals and somatic symptoms as well as environmental social threat cues. Biased attention to negative stimuli such as memories of prior social failures, and negative attributions following ambiguous situations (such as when a conversation partner yawns), perpetuate initial fears and somatic symptoms.

People with SAD are also plagued by impression management concerns. Besides doubting their ability to form a good impression, people with SAD assume other people share these unflattering assessments. Subsequently, people with SAD tend to avoid social situations or be minimally engaged. The social impairments and hedonic deficits interfere with the ability of individuals with SAD to form and sustain healthy relationships.

Comorbidity implications

People with comorbid depression and SAD have been shown to exhibit greater distress, and occupational and social impairment than people meeting diagnostic criteria for only one of these conditions (Darlymple, & Zimmerman, 2007). In a one-year prospective study of adult outpatients, the presence of comorbid SAD increased the severity, chronicity, and disability associated with major depressive disorder (Gaynes et al., 1999). In a four-year prospective study of adolescents, the presence of comorbid SAD increased the likelihood that adolescents with major depressive disorder met criteria for substance abuse disorders, attempted suicide, and experienced chronic depression at the follow-up (Stein et al., 2001). Available evidence suggests that the presence of comorbid SAD amplifies the presenting problems of people suffering from major depressive disorder.

There is a surprising paucity of research on how anxiety symptoms influence the efficacy of treatments for depression on depressive symptoms. Published findings are mixed on the effects of comorbid anxiety symptoms on the treatment of depression. In two separate trials of CBT, clients with greater anxiety symptoms fared no worse than clients with "pure" depression (Fournier et al., 2009; Gibbons & DeRubeis, 2008). Two other studies found that clients treated for depression fared worse when diagnosed with comorbid SAD. Mulder et al. (2006) found that when diagnosed with comorbid SAD, clients treated for depression with fluoxetine or nortriptyline were 2.5 times more likely to achieve no remission or recovery (poor response) compared with full recovery up to six months. Brent et al. (1998) found that adolescents diagnosed with depression and comorbid SAD had a lower rate of depression remission compared with adolescents diagnosed with only depression. In a comparison of treatments for depression, people suffering from extreme, pervasive, chronic social anxiety difficulties (i.e., comorbid avoidant personality disorder) experienced poorer responses to interpersonal therapy, however, these symptoms did not influence responses to cognitive-behavioral therapy (Joyce et al., 2007). In sum, few published studies exist on the impact of SAD on depressive symptom outcomes following treatment for depression. Each of these studies used individual treatment approaches. Different results might emerge for comorbid SAD when examining cognitive-behavioral group treatment approaches to depression.

To our knowledge, only three studies of the efficacy of CBT for depression with comorbid SAD used intermittent assessments of depressive symptom s to study rate of improvement. Smits, Minhajuddin, and Jarrett (2009) found that clients with and without comorbid SAD did not differ in the amount or speed of improvement in depressive symptoms. Surprisingly, in two studies (each including 57 separate outpatients, respectively) researchers found that greater anxiety symptoms at baseline predicted a faster rate of depressive symptom improvement, which happened to occur in early sessions (i.e., rapid gains) (Forand, Gunthert, Cohen, Butler, & Beck, 2011). Following prior theory and research, two competing hypotheses emerged. First, depressed clients with comorbid SAD might exhibit poorer outcomes following group treatment for depression. This is because the excessive self-directed attention of people with SAD might interfere with learning information in social-evaluative situations, such as the skills being taught in a group treatment focusing on depression. Social anxiety difficulties in depressed clients might also lead to perceptions of poor relationships with therapists and other group members that in turn exacerbate social fears, inhibit motivation, and interfere with the benefits of healthy therapeutic alliances. Second, clients with comorbid SAD might fare better than clients with "pure" depression in treatment. After all, group-based interventions provide exposure to "safe" situations where clients can experiment with assertiveness, relaxation, and cognitive retraining skills being taught by clinicians (Heimberg & Becker, 2002). Cognitive-behavioral treatments have been shown to effectively treat the maladaptive emotions, thoughts, and behaviors that are common to anxiety and depressive conditions (Hollon, Stewart, & Strunk, 2006).

Potential mechanisms of action

The current study is the first to examine the impact of comorbid SAD on the processes and outcomes of group psychotherapy—depressive symptom change, the direction of attention (self vs. other) during treatment sessions, and closeness and attachment orientation to the therapy group. In terms of direction of attention, individuals with SAD have elevated levels of self-focused attention (e.g., Hofmann, 2000), which may interfere with their ability to fully focus and attend to the material covered during treatment sessions. Rather than attending to the particular skills being taught, these individuals might perceive themselves as social objects, and become inundated with negative self-focused thoughts including beliefs of social incompetence and concerns that symptoms are noticeable by others (Clark & Wells, 1995; Rapee & Heimberg, 1997). As a result, clients with comorbid SAD may derive less benefit from group treatment for depression. In addition, excessively anxious clients are more likely to have difficulty forming positive attachments with their therapists (Mallinckrodt, Coble, & Gantt, 1995), and rate themselves as less trusting and more fearful of being rejected by therapists (Mallinckrodt, King, & Coble, 1998). More generally, clients with SAD are prone to feeling abandoned and rejected by significant others (anxious dimension), and maintain beliefs that others cannot be trusted to provide support or be privy to personal vulnerabilities (avoidance dimension) (e.g., Eng et al., 2001). Given that attachment styles between clients and therapists are strong predictors of self-disclosure, social competence, emotion regulation, willingness to use intervention techniques, and treatment outcome (e.g., Lopez & Brennan, 2000; Satterfield & Lyddon, 1998) and that the quality of relationships between therapists and clients facilitates treatment engagement and symptom amelioration (e.g., Horvath & Symonds, 1991), individuals with SAD may be less likely to benefit from group treatment for depression; they would be predisposed to experiencing poor relationships with therapists and other group members.

On the other hand, it is plausible that each of the above mechanisms might accelerate recovery from depressive symptoms among depressed clients with comorbid SAD (cf. Forand et al., 2011). For instance, depressed individuals with SAD might have greater room for improvement in attentional focus than those with pure depression, and the group intervention might be particularly well suited for facilitating shifts in attentional focus from the self to positive attributes of the external environment. Consequently, the presence of SAD might lead to more rapid declines in depressive

symptoms as a function of declining self-focused attention within and between sessions. In a similar manner, the group format might provide a powerful vehicle for clients with comorbid SAD to develop positive therapeutic relationships either with other group members and/or therapists. Consistent with this notion, a recent clinical study found that people with SAD experience a therapeutic alliance that strengthens over the course of group therapy (Woody & Adessky, 2002). Although clients with comorbid SAD might initially experience worse relationships with therapists and other group members compared with "pure" depression clients, these relationships might strengthen more rapidly over time and contribute to greater reductions in depressive symptoms.

The present study

We examined the impact of comorbid SAD during group psychotherapy for depression with repeated assessments of depressive symptoms and process-oriented variables. We tested two primary questions. Does comorbid SAD influence the trajectory depressive symptom improvement during treatment? Are differential treatment responses mediated by excessive self-focused attention and/or relationship difficulties with therapists and other group members? Competing hypotheses emerged. Social anxiety disorder might interfere with depressive symptom improvement as a function of self-focused attention and relationship perceptions. Alternatively, clients with comorbid SAD might demonstrate rapid improvement in depressive symptoms because of the exposure inherent in group treatment.

Method

Participants

The present sample represents the subset of patients in an outpatient treatment program for depression who had been administered measures to evaluate relationships with therapists and other group members (see below) and who met diagnostic criteria for a depressive disorder (based on structured clinical interview) and a Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) score of 15 or greater. Exclusionary criteria included Bipolar Disorder or any psychotic disorder. A total of 76 people (59 women) met these criteria. The sample was primarily Caucasian (89.5%) and single (57.9% single, 23.7% married, 2.6% separated, and 15.8% divorced). The mean age was 37.8 (SD = 10.4). Forty clients (52.6%) met criteria for current Major Depressive Disorder (MDD), 31 (40.8%) met criteria for MDD with some degree of remission, and 5 (6.6%) had MDD-NOS Minor Depression. Twenty-one clients (27.6%) met criteria for comorbid SAD. Forty-one clients (53.9%) reported ongoing psychopharmacological treatment.

Clinical intervention

Treatment involved a 10-session psychoeducational group for depression based on the "Coping with Depression" course (CWDC; see Lewinsohn, Antonuccio, Steinmetz, & Teri, 1984). Two sessions were conducted in an individual format and eight sessions had a group format. Subsequent to the initial diagnostic evaluation, a single individual session focused on psychoeducation about depression, the process of self-change, and the nature of treatment. The following eight sessions had a group format with two clinicians leading weekly group meetings with a maximum of eight clients. Each session was 90 min in length. The final termination session had an individual format, focusing on consolidating gains. Clinicians were advanced clinical graduate students. Skills taught to alleviate emotional distress included cognitive restructuring, increasing pleasant activities, relaxation training, and enhancing social skills. Two sessions were spent on each skill. Clients were asked to purchase an adjunctive self-help book, *Control Your Depression* (Lewinsohn, Munoz, Youngren, & Zeiss, 1986). Weekly homework assignments were derived from the book, including reading relevant chapters, self-monitoring daily mood and factors that affect and are affected by mood, and practicing skills. In a meta-analysis, the CWDC was shown to exhibit a mean effect size of 0.65, indicative of a large between-subjects effect (Cuijpers, 1998). A mean within-subjects effect size of 1.21 was found for pre- to post-treatment improvement in depressive symptoms.

Measures

Clients completed self-report questionnaires and participated in structured interviews focusing on psychiatric diagnoses. This included basic demographic questions addressing gender, age, ethnicity, socio-economic status, and religious orientation. Only measures relevant to the current study are reported.

Diagnostic assessment

The Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1997) was administered by advanced graduate students. Interviewers assessed current and lifetime incidence of mood and anxiety disorders. Diagnoses of SAD were qualified with the Generalized or Nongeneralized subtype. Inter-rater reliability was examined with19 audiotaped interviews evaluated by an advanced graduate student. Kappa coefficients were .69 for MDD, .64 for Dysthymic Disorder, and 1.0 for SAD.

Depression severity

Clients completed the BDI-II before each treatment session. The BDI-II consists of 21 questions on a 0–3 point scale with larger numbers indicative of greater severity.

Social anxiety severity

At baseline, clients completed the 19-item *Social Interaction Anxiety Scale* (SIAS; Mattick & Clarke, 1998). Participants rated various statements assessing anxiety of social situations along a 5-point Likert scale (0 = "Not at all" to 4 = "Extremely"). The SIAS has been shown to have excellent psychometric properties, be highly sensitive to clinical interventions, and adequately distinguish individuals with and without SAD diagnoses (e.g., Brown et al., 1997; Mattick & Clarke, 1998).

Therapy process measures

Direction of attention

To assess focus of attention during treatment, clients completed the Focus of Attention Questionnaire (FAQ; Woody, Chambless, & Glass, 1997) after each session. Self-focus items reflected attention to personal feelings, cognitions, and past social failures, whereas external-focus items reflected attention to the task and environment. Prior work indicates that the FAQ-self has excellent psychometric properties, construct validity, and sensitivity to treatment (e.g., Woody, 1996; Woody et al., 1997). Clients responded to items on a 7-point Likert Scale (1–7). When collapsed across time, the FAQ-self and FAQ-external each demonstrated adequate internal consistency ($\alpha = .79$ and .75, respectively).

Quality of relationships with therapists and group members

To assess the quality of relationships with therapists and treatment group members, clients completed three measures immediately after sessions. First, they completed a modified version of the 36-item Experiences in Close Relationships Inventory (ECR;

Brennan, Clark, & Shaver, 1998). The ECR is comprised of two 18-item scales assessing the higher-order attachment dimensions of Anxiety and Avoidance with responses on a 7-point Likert Scale (1–7). The wording of the ECR was modified to reflect immediate perceptions of relationships with therapists and group members following group treatment sessions. Since not all scale items were translatable into "state" items, we created 8-item Anxiety and Avoidance scales based on the highest-factor loadings reported in Brennan et al. (1998). The Anxiety scale measured concerns about the ability to create and sustain a reciprocally close relationship (e.g., "I worried a fair amount about not having a relationship with them"). The Avoidance scale measured concerns about being able to disclose one's innermost feelings and thoughts (e.g., "I felt comfortable sharing my private thoughts and feelings with them."). When collapsed across time, the ECR-Anxiety and ECR-Avoidance scales demonstrated good to excellent internal consistency ($\alpha = .94$ and .84, respectively).

Second, clients were administered the one-item Inclusion of Other in the Self Scale (IOS; Aron, Aron, & Smollan, 1992) to assess perceived closeness to therapists and other group members. The IOS has a series of seven pairs of overlapping circles that gradually escalate in their degree of overlap. Circles in each pair are labeled "self" and "other" to represent a relationship. Third, clients were given a variant of the IOS where clients rated perceived closeness compared to existing relationships in their life—an ecological reference point (Berscheid, Snyder, & Omoto, 1989). Research finds the IOS to have similar psychometric properties to longer, complex relationship measures. Thus, clients completed four items reflecting closeness felt during a session to: (1) therapists, (2) therapists compared to existing relationships, (3) group members, and (4) group members compared to existing relationships. Collapsed across time, the four items were highly correlated (*r*s between .70 and .87) and we therefore used a composite to assess closeness to therapists and group members during treatment sessions ($\alpha = .94$).

Assessment procedure

Prior to treatment, clients were administered the SCID, and completed questionnaires including the BDI-II. Prior to each session, clients completed an additional BDI-II. To examine process variables (direction of attention, relationship with therapists and group members), immediately after each group treatment session, clients were led to the clinic waiting room where an independent research assistant handed them questionnaires; therapists did not have access. At the final group treatment session, clients were provided questionnaires identical to the pre-treatment packet. Two weeks later, clients were administered the HRSD. Treatment completers reflected clients attending at least four of eight group sessions.

Data analytic plan

We examined longitudinal growth curve trajectories of BDI-II scores and therapy process variables during the course of treatment using multilevel modeling (Speer & Greenbaum, 1995). Data were hierarchical with repeated weekly assessments nested within clients. Thus, each participant's rate of change coefficient (or growth curve) was estimated using all available data. To represent the rate of treatment change, the variable Time reflected session number. Preliminary analyses tested whether there was significant variance in the slope of Time by conducting deviance tests comparing models with and without a slope specified as random. Social Anxiety Disorder served as the primary between-person independent variable and we tested the cross-level interaction between Time (Level-1) and SAD (Level-2) to determine if SAD influenced treatment change.

The variable Time was centered at the intake assessment such that the intercept reflected the predicted score at the start of treatment. Social Anxiety Disorder was a binary variable scored 0 when absent and 1 when present. A large portion of clients in treatment for depression show substantial symptom reductions early in treatment followed by a slower rate of sustained symptom relief (Lueger et al., 2001; Tang & DeRubeis, 1999a, 1999b). Consequently, we included a quadratic term to model non-linear change trajectories. Multilevel growth models were estimated using the nlme program (Pinheiro, Bates, DebRoy, Sarkar, & the R Core Team, 2009) in R 2.11 (R Development Core Team, 2010). Mediation was tested using bootstrap parametrics (MacKinnon, Lockwood, & Williams, 2004) in which individual growth curve intercepts and slopes were estimated (based on nlme) and these parameters were then used as variables in linear regression models. Unless otherwise noted, analyses were based on the intent-to-treat sample. Results were substantively identical with the treatment completer sample (defined as attending at least 4 of the 8 group sessions ($n = 62$)).

Results

Preliminary analyses

Participants attended 8.0 (SD = 2.0) out of the 11 sessions on average. SAD was not significantly associated with number of sessions attended, $t = 0.12$, $p = .23$, treatment completion status, $\chi^2 = 0.06$, $p = .81$, or medication status, $\chi^2 = 0.00$, $p = .99$. Further, SAD was not associated with gender, $\chi^2 = 0.02$, $p = .90$, ethnicity (Caucasian vs. non-Caucasian), $\chi^2 = 0.06$, $p = .81$, or age, $t = 0.25$, $p = .81$. Although initial BDI-II scores did not differ as a function of SAD, $t = 1.41$, $p = .17$, participants with SAD ($M = 77.3$) reported elevated symptoms of social anxiety compared to those without SAD ($M = 49.8$), $t = 4.25$, $p < .001$.

Among the 62 treatment completers, 69.4% were responders (defined as $a \geq 50\%$ reduction in depression scores between baseline and treatment termination), while 56.5% achieved remission (defined as BDI-II < 10 at termination). SAD was not associated with either responder, $\chi^2 = 0.84$, $p = .85$, or remission, $\chi^2 = 0.46$, $p = .49$, status. As shown in Table 1, at the first session of group treatment, each of the putative mediators, except for closeness, was associated with greater depressive symptoms (*r*s between .25 and .35).

Is there significant variability in growth curves?

Using the full sample, we found support for both linear, $b = -1.48$, $t = 11.06$, $p < .001$, and quadratic, $b = 0.17$, $t = 6.78$,

Table 1
Correlation matrix of baseline depressive symptoms and putative mediating variables.

	1	2	3	4	5	6
1. BDI-II	–	.29	.26	.38	.44	−.14
2. FAQ-Self	–	–	.66	.43	.51	−.02
3. FAQ-External	–	–	–	.31	.46	−.21
4. Attach-Anxiety	–	–	–	–	.66	.02
5. Attach-Avoid	–	–	–	–	–	−.10
6. IOS	–	–	–	–	–	–

Notes. BDI-II = Beck Depression Inventory-II. FAQ-Self = Focus of Attention Questionnaire, Self-Focus Subscale. FAQ-External = Focus of Attention Questionnaire, External-Focus Subscale. Attach-Anxiety = Attachment Orientation to Therapists and Group Members, Anxiety Scale. Attach-Avoid = Attachment Orientation to Therapists and Group Members, Avoidance Scale. IOS = Inclusion of Other in the Self Scale. All of these variables were from the first group session. The BDI-II was completed before the first group treatment session and the other variables were completed after the session was over.

$p < .001$, trajectories of change in BDI-II scores. The negative slope of the linear term indicates that on average, the sample experienced reductions in BDI-II scores over the course of treatment. In terms of the quadratic effect, simple slope analyses (see Aiken & West, 1991) found that change occurred more rapidly early in treatment compared to later in treatment. For example, when conditioned at the first group treatment session, the simple effect was $b = -2.44$, $t = 12.53$, $p < .001$, while conditioned at the final group session, it was $b = -0.08$, $t = 0.33$, $p = .74$. Based on a model with the linear effect, the estimated initial BDI-II score was 26.3 and estimated termination score was 11.6. Despite an overall trend of improvement, there was significant variability in the linear rate of treatment change, Likelihood Ratio = 73.40, $p < .001$. In other words, rate of change was a random effect varying from person to person. These data provide support for examining individual difference predictors of rate of change in subsequent models. These subsequent models include the quadratic trajectory as a covariate.

Does SAD predict rates of depressive symptom improvement?

Time variables (i.e., Linear and Quadratic Time predictors) were centered at the intake assessment such that the intercept reflected predicted baseline scores. For the full sample, the main effect of SAD, $b = 4.08$, SE = 2.70, $t = 1.51$, $p = .14$, was not statistically significant. This indicates that clients with comorbid SAD did not differ from those without SAD on baseline depressive symptoms. In contrast, the main effect of Time, $b = -2.93$, SE = 0.29, $t = 10.10$, $p < .001$, the quadratic term, $b = 0.17$, SE = 0.03, $t = 6.02$, $p < .001$, and of most relevance the Time × SAD interaction, $b = -0.61$, $t = 3.58$, $p < .001$, were each statistically significant. The main effect of time indicates that on average, the sample reported decreases in depressive symptoms with each subsequent session and the quadratic effect indicates that these rates of change were most rapid early in treatment. In terms of the interaction, simple effect analyses demonstrated that the linear rate of improvement was more rapid when clients presented with SAD, $b = -3.54$, SE = 0.31, $t = 11.37$, $p < .001$, than when SAD was absent, $b = -2.93$, SE = 0.29, $t = 10.10$, $p < .001$. See Fig. 1 for a graphical presentation.

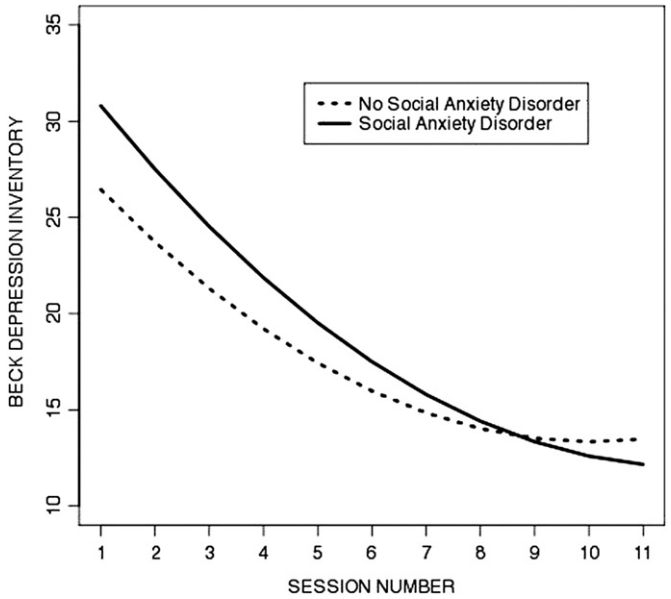

Fig. 1. Changes in depressive symptoms as a function of treatment session and social anxiety disorder.

Are there demographic or treatment related third variables that account for SAD effects?

As possible third-variable confounds, we examined whether gender, age, ethnicity (Caucasian vs. non-Caucasian), number of treatment sessions attended (Sessions) or pharmacotherapy moderated the growth curve. In the intent-to-treat sample, there was a significant Time × Sessions interaction, $b = 0.12$, SE = 0.06, $t = 2.05$, $p < .05$, but the Time × SAD interaction remained significant, $b = -0.61$, SE = 0.17, $t = 3.58$, $p < .001$, even when controlling for this effect. Likewise, there was a significant Time × Gender interaction, $b = 0.36$, SE = 0.18, $t = 1.99$, $p < .05$, but the Time × SAD interaction remained significant, $b = -0.61$, SE = 0.17, $t = 3.61$, $p < .001$, even controlling for this effect. None of the other variables approached significance as moderators of the growth curve (all $ts < 1.29$, all $ps > .19$). Despite redundancy, we examined pre-treatment BDI-II scores as a covariate in the model. Although baseline depression scores were associated with elevated depression over the course of treatment, $b = 0.73$, SE = 0.09, $t = 7.88$, $p < .001$, the inclusion of this covariate had no effect on the Time × SAD interaction, $b = -0.66$, SE = 0.19, $t = 3.54$, $p < .001$. Finally, given that group size could fluctuate from session-to-session, we included this variable as a covariate. Again the Time - × SAD interaction remained statistically significant, $b = -0.64$, SE = 0.19, $t = 3.46$, $p < .001$.[1]

Is the effect of SAD unique or do other anxiety disorders show similar effects?

To answer this question we created a new factor with three categories: (1) pure depression ($n = 28$), (2) depression with comorbid anxiety disorders other than SAD ($n = 27$), and (3) depression with comorbid SAD ($n = 21$). Although these three groups did not differ in terms of baseline BDI scores, $F(2, 73) = 1.01$, $p = .37$, they differed on social anxiety, $F(2,73) = 7.54$, $p < .005$. Pairwise comparisons indicated that the comorbid SAD group had higher symptoms than both the pure depressed, $p < .0001$, and the other anxiety disorder groups, $p < .0005$, whereas the pure depressed and other anxiety disorders groups did not differ, $p = .75$. The three groups did not differ in their likelihood of either treatment response or remission, $ps > .50$.

Consistent with the results presented above, growth curve modeling indicated that there was a statistically significant Group × Time interaction, $F(2,584) = 7.03$, $p < .005$. Consistent with the notion of specificity, the pure depression vs. depression with SAD contrast yielded a significant Group × Time interaction, $b = -0.72$, SE = 0.20, $t = 3.67$, $p < .001$, whereas the pure depression vs. depression with other anxiety disorders contrast yielded a non-significant Group × Time interaction, $b = -0.21$, SE = 0.19, $t = 1.11$, $p = .26$. These results suggest that SAD has a unique effect on depression growth curves that is not shared with other anxiety disorders.

Do treatment process variables mediate the effects of SAD on rates of improvement?

We tested five treatment process variables that were hypothesized to mediate the effects of SAD on treatment response, including self-focused attention, other focused attention, anxious attachment orientation to the therapy group, avoidant attachment orientation to the therapy group, and overall closeness to the

[1] Results were relatively identical when order of treatment modules was included as a covariate.

therapy group. To capitalize on the repeated measurements of these variables at each of the eight group therapy sessions, we conducted analyses similar to those reported above. We tested whether clients with SAD would differ from those without SAD in either their estimated baseline score (intercept) or rate of change (slope) on each putative mediator. For those variables where this was the case, models with both random intercepts (centered to the first group session) and slopes were estimated, and these random effects were extracted for use in subsequent mediation analyses. In other words, we generated new values reflecting estimated initial scores of the hypothetical mediator (random intercepts) and its rate of change over the course of treatment (random slopes) and used these variables as potential mediators of the Time × SAD interaction in predicting treatment response.

Focus of attention

For external-focused attention in the full sample, the main effect of SAD, $b = -0.43$, SE = 1.15, $t = 0.37$, $p = .71$, was not significant, indicating that clients with comorbid SAD did not differ from those without SAD on baseline external-focused attention. In contrast, the main effect of Time, $b = -0.84$, SE = 0.22, $t = 3.76$, $p < .001$, and the quadratic term, $b = 0.08$, SE = 0.03, $t = 2.61$, $p < .01$, were each statistically significant. The main effect indicates that with each subsequent session, clients reported less external focus of attention, while the quadratic effect indicates that rates of change were most rapid early in treatment. Of most relevance, the Time × SAD interaction, $b = 0.31$, SE = 0.14, $t = 2.30$, $p < .05$, was statistically significant (see Fig. 2). Simple slope analyses indicated that decreases in external focus were more rapid for clients without SAD, $b = -0.84$, SE = 0.22, $t = 3.76$, $p < .001$, than clients with SAD, $b = -0.53$, SE = 0.24, $t = 2.19$, $p < .05$.

Because rates of change in external-focused attention varied as a function of SAD it could have served as a mediator. As a preliminary step in testing mediation, we examined the correlation between the growth curve of depression (estimated slopes of BDI scores over time across individuals) and the growth curve of external-focused attention (estimated slopes of external focus over time across individuals). Inconsistent with mediation, this correlation was not statistically significant, $r = -.13$, $p = .28$, in the intent-to-treat sample. Likewise, the indirect effect was not statistically significant (95% confidence intervals between −0.14 and 0.05) when its standard error was estimated through bootstrapping with 5000 simulated samples (see MacKinnon et al., 2004) using mediation: R Package for Causal Mediation Analysis (Keele, Tingley, Yamamoto, & Imai, 2009). In addition, there was no evidence of mediation in the completer sample (95% confidence intervals between −0.17 and 0.08).

For self-focused attention in the full sample, the SAD main effect, $b = 1.41$, SE = 1.34, $t = 1.05$, $p = .30$, and Time × SAD interaction, $b = -0.08$, SE = 0.15, $t = 0.51$, $p = .61$, were not significant. This indicates that clients with comorbid SAD did not differ from those without SAD on baseline self-focused attention or in rates of change over the course of treatment. In contrast, the main effect of Time, $b = -0.89$, SE = 0.24, $t = 3.66$, $p < .005$, and the quadratic term, $b = 0.09$, SE = 0.03, $t = 2.53$, $p < .05$, were each significant. The main effect indicates that with each subsequent session, clients reported less self-focused attention, while the quadratic effect indicates that rates of change were most rapid early in treatment (see Fig. 2). These results indicate that self-focused attention was not a viable mediator.

Closeness in therapeutic relationships

For closeness in therapeutic relationships in the full sample, the main effect of SAD, $b = -1.74$, SE = 1.55, $t = 1.13$, $p = .26$, was not statistically significant, indicating that clients with comorbid SAD did not differ from those without SAD on baseline therapeutic relationships. In addition, the main effect of Time, $b = 0.14$, SE = 0.24, $t = 0.61$, $p = .54$, and the quadratic term, $b = 0.01$, SE = 0.03, $t = 0.31$, $p = .76$, were not statistically significant. In contrast, the Time × SAD interaction, $b = 0.26$, SE = 0.14, $t = 1.87$, $p = .06$, was marginally significant. Simple slope analyses indicated that rates of change were more rapid among clients with SAD, $b = 0.41$, SE = 0.254, $t = 1.60$, $p = .11$, than clients without SAD, $b = -0.14$, SE = 0.24, $t = 0.61$, $p = .54$; see Fig. 3. Given that the Time × SAD interaction was marginally significant and difficult to interpret, closeness was deemed to be non-viable as a possible mediator.

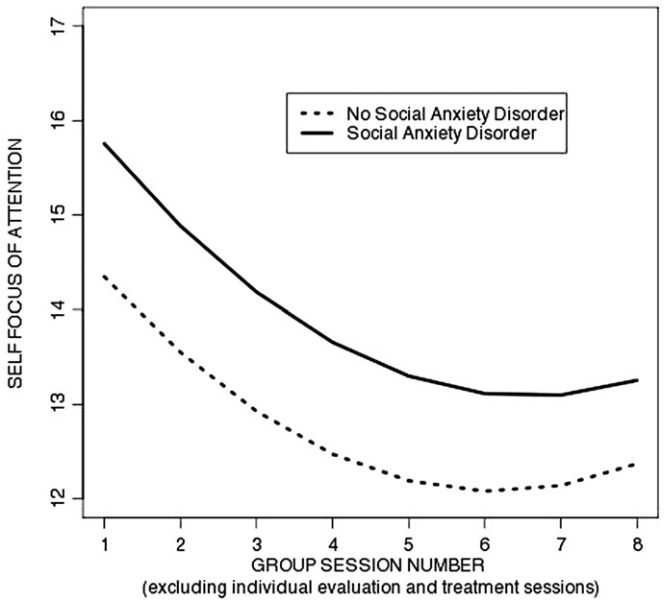

Fig. 2. Changes in focus of attention as a function of treatment session and social anxiety disorder.

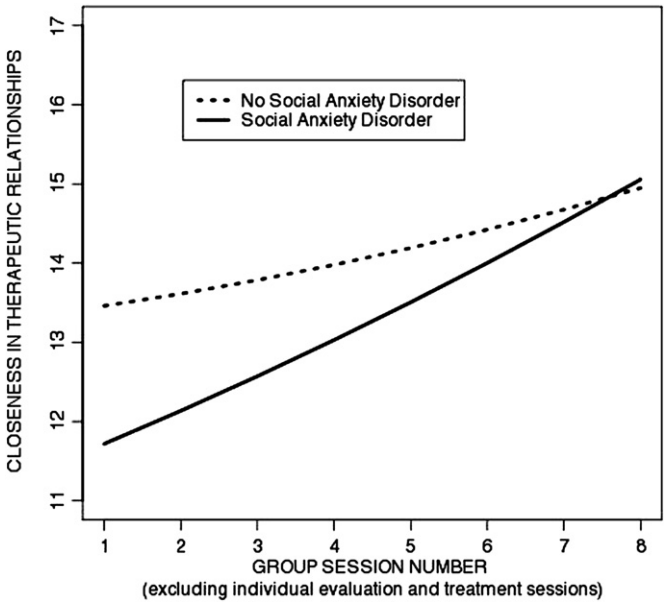

Fig. 3. Changes in closeness in therapeutic relationships as a function of treatment session and social anxiety disorder.

Avoidant and anxious attachment in therapeutic relationships

For anxious attachment in the full sample, the SAD main effect, $b = -0.94$, SE = 2.03, $t = 0.46$, $p = .65$, and Time × SAD interaction, $b = -0.002$, SE = 0.175, $t = 0.01$, $p = .99$, were not significant. Thus, clients with comorbid SAD did not differ from those without SAD on baseline anxious attachment or rates of change over the course of treatment. In contrast, the main effect of Time, $b = -1.03$, SE = 0.29, $t = 3.53$, $p < .001$, and the quadratic term, $b = 0.12$, SE = 0.04, $t = 2.96$, $p < .005$, were each significant. The main effect indicates that with each subsequent session, clients reported reductions in anxious attachment, while the quadratic effect indicates that rates of change were most rapid early in treatment (see Fig. 4).

For avoidant attachment in the full sample, the main effect of SAD, $b = 0.57$, SE = 1.44, $t = 0.40$, $p = .69$, and the Time × SAD interaction, $b = -0.203$, SE = 0.175, $t = 1.16$, $p = .25$, were not statistically significant. This indicates that clients with comorbid SAD did not differ from those without SAD on baseline avoidant attachment or in rates of change over the course of treatment. In contrast, the main effect of Time, $b = -0.73$, SE = 0.29, $t = 2.51$, $p < .05$, was statistically significant, whereas the quadratic term was not, $b = 0.062$, SE = 0.041, $t = 1.51$, $p = .25$. The main effect indicates that with each subsequent session, clients reported decreased avoidant attachment (see Fig. 4). Together these results suggest that neither anxious nor avoidant attachment was a viable mediator of the Time × SAD interaction in predicting treatment response.

Discussion

This study investigated the impact of SAD on the efficacy of group-based cognitive-behavioral therapy for depression. Although this intervention focally targeted depression, clients with comorbid SAD exhibited a significantly faster rate of symptom reduction over the course of treatment compared to those without SAD—primarily accounted for by rapid gains in early sessions. Findings remained after statistically controlling for initial symptom severity and the number of treatment sessions attended (ruling out alternative explanations such as regression to the mean) and were similar regardless of whether examinations focused on the full sample (intent-to-treat) or completers. Furthermore, these effects were unique to SAD and were not found with other comorbid anxiety disorders. In contrast to SAD effects on better treatment response, SAD had no significant association with baseline depressive symptoms, number of sessions attended, or attrition. Based on dominant theoretical models of SAD (Clark & Wells, 1995; Rapee & Heimberg, 1997), we tested multiple plausible mediating mechanisms that might account for how SAD impacts the treatment of depression: focus of attention and perceived relationships with therapists and other group members. Both of these mechanisms failed to account for the effects of SAD on treatment response. In fact, clients with comorbid SAD tended to exhibit similar rates of change in the development of therapeutic relationships and in focus of attention during the course of treatment compared to those without this form of comorbidity.

Improvement and growth trajectory of depressive symptoms during treatment

Previous examinations of the "Coping with Depression Course" (Lewinsohn et al., 1984, 1986) have found substantial improvement from pre- to post-treatment (Cuijpers, 1998; Roberts, Shapiro, & Gamble, 1999). Our work extended these findings with a more sophisticated longitudinal growth curve modeling approach. Capitalizing on strategic, repeated, BDI-II assessments throughout treatment, we applied a longitudinal growth curve modeling approach to change. Researchers have recently found non-linear trajectories of change over the course of psychological treatments for depression (e.g., Gunthert, Cohen, Butler, & Beck, 2005). With separate analyses for completer and intent-to-treat groups, we found support for a curvilinear trend (see Fig. 1). For the average client, depressive symptoms decreased at a rapid rate during the beginning of treatment followed by a slow, stable rate of change for the remainder of treatment. These results fit with work suggesting that for a large number of clients, the vast percentage of symptom improvement occurs in the first few weeks of therapy (Ilardi & Craighead, 1994; Tang & DeRubeis, 1999a, 1999b). Longitudinal growth curve modeling can elucidate "how," "when," and "why" clients change or fail to change over the course of treatment.

SAD and treatment response

The use of longitudinal growth curve modeling allowed us to understand whether the change process during treatment had the same shape for clients with and without comorbid SAD diagnoses. We found evidence that SAD moderated individual rates of change

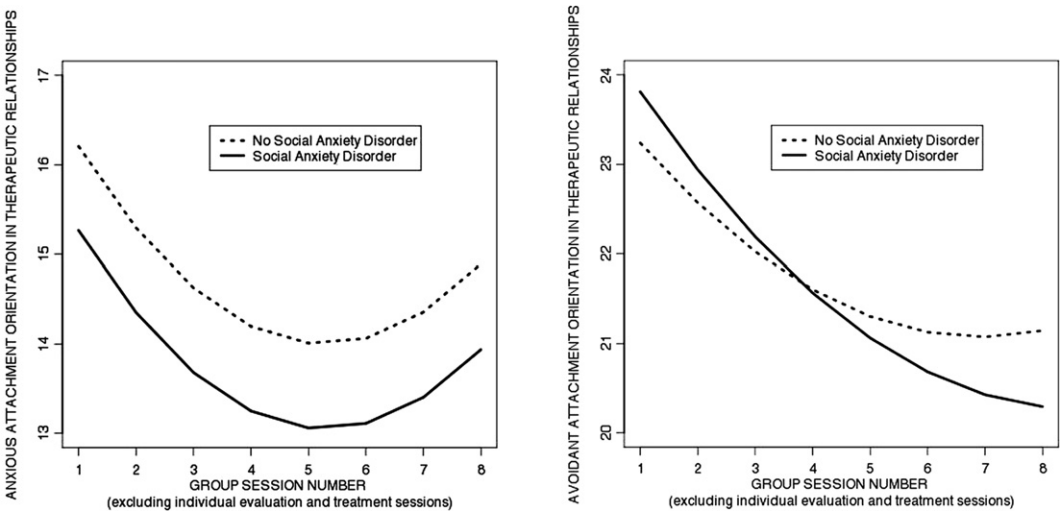

Fig. 4. Changes in attachment in therapeutic relationships as a function of treatment session and social anxiety disorder.

(Figs. 1 and 2). Clients with SAD recovered from depression at a faster rate than clients without SAD, particularly in the first few group sessions. These results attest to consistently rapid change trajectories for clients and the need to understand early gains in treatment. Future research will need to extend individual change trajectories beyond symptom reduction to understand functional impairment and the consolidation or deterioration of gains following treatment termination (McKnight & Kashdan, 2009). Web-based data collection and ecological momentary assessment techniques are useful tools for investigating "how" and "why" depressed clients with and without SAD experience differential change trajectories. With technological advancements, assessments can occur in clients' natural environment, minimizing response biases in assessing symptoms in the same location where treatment occurs. Information processing and behavioral tasks (including application of skills taught in treatment) can serve as supplements to traditional self-report approaches.

Temporal examination of therapeutic mechanisms

Based on relevant theory, we examined specific therapeutic processes that might mediate the effect of SAD on depressive treatment outcomes. As a result of repeated measurements at each treatment session, we addressed how temporal changes in self-focused attention and the quality of therapeutic relationships early in treatment predicted symptom changes across treatment. Despite the sophistication of this approach, we failed to find support for any of our proposed mediating mechanisms. However, we did discover how these therapeutic mechanisms unfold over the course of treatment. With each subsequent session, clients reported reductions in self-focused attention and anxious and avoidant attachment orientations with therapists and other group members during sessions. For each of these mechanisms, the rate of change occurred most rapidly early in treatment before leveling off in remaining sessions. Surprisingly, clients with and without SAD did not differ in self-focused attention or relationships with therapists and group members within initial group therapy sessions. At a trend level, compared to clients without SAD, clients with SAD reported more rapid increases in feelings of closeness to therapists and other group members over the course of treatment. Although replication is required, this latter finding suggests that the impact of SAD on the treatment of depression might be relatively benign in terms of perceived intimacy with other people in the therapeutic environment. People with SAD tend to have impoverished social networks including fewer and less satisfying friendships, a low probability of being sexually active, and a tendency to feel dependent on the people in their lives (Kashdan, Adams, et al., 2011; Rodebaugh, 2009; Schneier et al., 1994). Thus, our results might reflect a low threshold for closeness in clients with SAD or excessive attachments to anyone providing signs of affection and care. It will be useful to test competing hypotheses as for what these stronger relationships with therapists and group members signify and how to best cultivate healthy alliances and group cohesion in treatment settings.

Our findings also suggest that excessive self-focused attention does not play a role in understanding the effects of SAD on depressive symptom changes during group treatment for depression. Data suggest that self-focused attention can be reduced over the course of treatment for SAD (Hofmann, 2000; Woody et al., 1997), and new therapies have been designed to specifically target excessive self-focused attention (Hofmann, Moscovitch, Kim, & Taylor, 2004). The average client in the current study, irrespective of SAD status, improved their attentional focus to be less self-directed and avoidant over the course of treatment. Thus, group-based cognitive-behavioral treatment for depression, without explicit modules targeting self-focused attention, was effective in addressing core cognitive features of SAD.

Caveats and limitations

Our treatment was a standard, manualized group treatment program with decades of research supporting its efficacy (Cuijpers, 1998). Yet, our use of rolling admissions led to continual changes in group dynamics. Although this approach reduces time spent on the waiting list for clients, the instability of the treatment group may account for our inability to find many effects of SAD on therapeutic relationship variables. It may have been difficult for group cohesiveness to develop, if the group members were continually changing. Nonetheless, results indicated that in the sample as a whole, clients tended to develop attachment security over the course of treatment. Other potential confounds related to our methodology, including fluctuations in group size, did not have significant relationships with primary variables. Despite our ability to rule out regression to the mean for depressive symptoms as an alternative explanation for our findings (by controlling for initial severity levels), we were unable to rule out the possibility that clients with SAD were more functionally impaired than clients without SAD. We relied on self-report instruments to assess qualities of the therapeutic relationship and focus of attention. There would be value in cross-validating these constructs with independent observations of videotaped sessions in future research. Finally, approximately half of our sample received psychotropic medication from a health professional. Although medication status had no association with predictors or outcomes, medication that is efficacious in treating SAD might have altered the effects of SAD on treatment response.

Implications and conclusions

We found SAD to predict a better response to group treatment for depression in a naturalistic setting. These results were derived from individual growth curve trajectories from session-to-session during the course of treatment. Pending replications, there are several clinical and research implications. The discussions, disclosure, and emotional processing inherent to cognitive-behavioral group treatment for depression (Lewinsohn et al., 1984) can serve as exposure therapy for clients with SAD. When processing the fears associated with expressing and experiencing painful experiences in a group social context, depressed clients with SAD might derive benefits that are not found in individual treatment. This includes the natural remission of fear via habituation within two-hour group sessions, and amelioration of self-focused attention and the development of close, secure attachments over the course of repeated therapeutic socialization (Heimberg & Becker, 2002; Turner, Beidel, & Cooley, 1994). Given the high rate of comorbid SAD in people suffering from depression, and the large portion of depressed clients who fail to recover at the end of treatment, our findings illuminate alternative perspectives on what influences treatment response. Although comorbidity may be the norm rather than the exception, comorbidity does not automatically imply greater problems in treatment settings (Lilienfeld, 2003).

Acknowledgments

Appreciation is extended to William E. Pelham, Jr., R. Lorraine Collins, Craig Colder, Paul A. Luce, and Martin M. Antony for their invaluable suggestions during the first author's dissertation defense. We would like to thank the individuals who served as therapists: Kathryn Bottonari, Christine Calmes, Jeffrey Ciesla, David Direnfeld, Stephanie Gamble, Morgen Kelly, and James

Prisciandaro. This study was supported by National Institute of Mental Health Grant MH63565 and MH073937, and dissertation grants from the Society for the Study of Clinical Psychology and the College of Arts and Sciences at the State University of New York at Buffalo awarded to the first author, and a NARSAD Young Investigator Award and a National Institute of Mental Health Grant R03MH060325-01 awarded to the second author.

References

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. London: Sage.

Aron, A., Aron, E. N., & Smollan, D. (1992). Inclusion of other in the self scale and the structure of interpersonal closeness. *Journal of Personality and Social Psychology, 63*, 596–612.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck depression inventory manual* (2nd ed.). San Antonio, TX: Psychological Corporation.

Berscheid, E., Snyder, M., & Omoto, A. M. (1989). The relationship closeness inventory: assessing the closeness of interpersonal relationships. *Journal of Personality and Social Psychology, 57*, 792–807.

Brennan, K. A., Clark, C. L., & Shaver, P. R. (1998). Self-report measurement of adult attachment: an integrative overview. In J. A. Simpson, & W. S. Rholes (Eds.), *Attachment theory and close relationships* (pp. 46–76). New York: Guilford Press.

Brent, D. A., Kolko, D. J., Birmaher, B., Baugher, M., Bridge, J., Roth, C., et al. (1998). Predictors of treatment efficacy in a clinical trial of three psychosocial treatments for adolescent depression. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*, 906–914.

Brown, C., Schulberg, H. C., Madonia, M. J., Shear, M. K., & Houck, P. R. (1996). Treatment outcomes for primary care patients with major depression and lifetime anxiety disorders. *American Journal of Psychiatry, 153*, 1293–1300.

Brown, E. J., Turovsky, J., Heimberg, R. G., Juster, H. R., Brown, T. A., & Barlow, D. H. (1997). Validation of the social interaction anxiety scale and the social phobia scale across the anxiety disorders. *Psychological Assessment, 9*, 21–27.

Brown, R. A., & Lewinsohn, P. M. (1984). A psychoeducational approach to the treatment of depression: comparison of group, individual, and minimal contact procedures. *Journal of Consulting and Clinical Psychology, 52*, 774–783.

Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the DSM-IV anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology, 110*, 585–599.

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York: Guilford Press.

Cuijpers, P. (1998). A psychoeducational approach to the treatment of depression: a meta- analysis of Lewinsohn's "coping with depression" course. *Behavior Therapy, 29*, 521–533.

Dalrymple, K. L., & Zimmerman, M. (2007). Does comorbid social anxiety disorder impact the clinical presentation of principal major depressive disorder? *Journal of Affective Disorders, 100*, 241–247.

Eng, W., Heimberg, R. G., Hart, T. A., Schneier, F. R., & Liebowitz, M. R. (2001). Attachment in individuals with social anxiety disorder: the relationship among adult attachment styles, social anxiety, and depression. *Emotion, 1*, 365–381.

Feeley, M., DeRubeis, R. J., & Gelfand, L. A. (1999). The temporal relation of adherence and alliance to symptom change in cognitive therapy for depression. *Journal of Consulting and Clinical Psychology, 67*, 578–582.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1997). *Structured clinical interview for DSM-IV axis I disorders – Patient edition (SCID)*. New York: Biometrics Research Department.

Forand, N., Gunthert, K., Cohen, L., Butler, A., & Beck, J. (2011). Preliminary evidence that anxiety is associated with accelerated response in cognitive therapy for depression. *Cognitive Therapy and Research, 35*, 151–160.

Fournier, J. C., DeRubeis, R. J., Shelton, R. C., Hollon, S. D., Amsterdam, J. D., & Gallop, R. (2009). Prediction of the response to medication and cognitive therapy in the treatment of moderate to severe depression. *Journal of Consulting and Clinical Psychology, 77*, 775–787.

Gaynes, B., Magruder, K. M., Burns, B. J., Wagner, H. R., Yarnall, K. S. H., & Broadhead, W. E. (1999). Does a coexisting anxiety disorder predict persistence of depressive illness in primary care patients with major depression? *General Hospital Psychiatry, 21*, 158–167.

Gibbons, C. J., & DeRubeis, R. J. (2008). Anxiety symptoms focus in sessions of cognitive therapy for depression. *Behavior Therapy, 39*, 117–125.

Gotlib, I. H., & Meltzer, S. J. (1987). Depression and the perception of social skill in dyadic interaction. *Cognitive Therapy and Research, 11*, 41–53.

Gunthert, K. C., Cohen, L. H., Butler, A. C., & Beck, J. S. (2005). Predictive role of daily coping and affective reactivity in cognitive therapy outcome: application of a daily process design to psychotherapy research. *Behavior Therapy, 36*, 77–88.

Heimberg, R. G., & Becker, R. E. (2002). *Cognitive-behavioral group treatment for social phobia: Basic mechanisms and clinical applications*. New York: Guilford Press.

Hoberman, H. M., Lewinsohn, P. M., & Tilson, M. (1988). Group treatment of depression: individual predictors of outcome. *Journal of Consulting and Clinical Psychology, 56*, 393–398.

Hofmann, S. G. (2000). Self-focused attention before and after treatment of social phobia. *Behaviour Research and Therapy, 38*, 717–725.

Hofmann, S. G., Moscovitch, D. A., Kim, H., & Taylor, A. N. (2004). Changes in self-perception during treatment of social phobia. *Journal of Consulting and Clinical Psychology, 72*, 588–596.

Hollon, S. D., Stewart, M. O., & Strunk, D. (2006). Cognitive behavior therapy has enduring effects in the treatment of depression and anxiety. *Annual Review of Psychology, 57*, 285–315.

Horvath, A. O., & Symonds, B. D. (1991). Relations between working alliance and outcome in psychotherapy: a meta-analysis. *Journal of Counseling Psychology, 38*, 139–149.

Ilardi, S. S., & Craighead, W. E. (1994). The role of nonspecific factors in cognitive-behavior therapy for depression. *Clinical Psychology: Science and Practice, 1*, 138–156.

Ingram, R. E. (1990). Self-focused attention in clinical disorders: review and a conceptual model. *Psychological Bulletin, 107*, 156–176.

Joyce, P. R., McKenzie, J. M., Carter, J. D., Rae, A. M., Luty, S. E., Frampton, C. M. A., et al. (2007). Temperament, character and personality disorders as predictors of response to interpersonal psychotherapy and cognitive-behavioural therapy for depression. *British Journal of Psychiatry, 190*, 503–508.

Kashdan, T. B. (2007). Social anxiety spectrum and diminished positive experiences: theoretical synthesis and meta-analysis. *Clinical Psychology Review, 27*, 348–365.

Kashdan, T. B., Adams, L., Savostyanova, A. A., Ferssizidis, P., McKnight, P. E., & Nezlek, J. B. (2011). Effects of social anxiety and depressive symptoms on the frequency and quality of sexual activity: a daily process approach. *Behaviour Research and Therapy, 49*, 352–360.

Kashdan, T. B., Weeks, J. W., & Savostyanova, A. A. (2011). Whether, how, and when social anxiety shapes positive experiences and events: a self-regulatory framework and treatment implications. *Clinical Psychology Review, 31*, 786–799.

Keele, L., Tingley, D., Yamamoto, T., & Imai, K. (2009). *Mediation: R package for causal mediation analysis*. R package version 2.1. http://CRAN.R-project.org/package=mediation.

Lewinsohn, P. M., Antonuccio, D. O., Steinmetz, J. L., & Teri, L. (1984). *The coping with depression course*. Eugene, Oregon: Castalia Publishing Company.

Lewinsohn, P. M., Munoz, R. F., Youngren, M. A., & Zeiss, A. M. (1986). *Control your depression*. New York: Prentice Hall.

Lilienfeld, S. O. (2003). Comorbidity between and within childhood externalizing and internalizing disorders: reflections and directions. *Journal of Abnormal Child Psychology, 31*, 285–291.

Lopez, F. G., & Brennan, K. A. (2000). Dynamic processes underlying adult attachment organization: toward an attachment-theoretical perspective on the healthy and effective self. *Journal of Counseling Psychology, 47*, 283–300.

Lueger, R. J., Howard, K. I., Martinovich, Z., Lutz, W., Anderson, E. E., & Grissom, G. (2001). Assessing treatment progress of individual patients using expected treatment response models. *Journal of Consulting and Clinical Psychology, 69*, 150–158.

MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: distribution of the product and resampling methods. *Multivariate Behavioral Research, 39*(1), 99–128.

Magee, W. J., Eaton, W. W., Wittchen, H.-U., McGonagle, K. A., & Kessler, R. C. (1996). Agoraphobia, simple phobia, and social phobia in the national comorbidity survey. *Archives of General Psychiatry, 53*, 159–168.

Mallinckrodt, B., Coble, H. M., & Gantt, D. L. (1995). Working alliance, attachment memories, and social competencies of women in brief therapy. *Journal of Counseling Psychology, 42*, 79–84.

Mallinckrodt, B., King, J. L., & Coble, H. M. (1998). Family dysfunction, alexithymia, and client attachment to therapist. *Journal of Counseling Psychology, 45*, 497–504.

Mattick, R. P., & Clarke, J. C. (1998). Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. *Behaviour Research and Therapy, 36*, 455–470.

McKnight, P. E., & Kashdan, T. B. (2009). The importance of functional impairment to mental health outcomes: a case for reassessing our goals in depression treatment research. *Clinical Psychology Review, 29*, 243–259.

Merikangas, K. R., & Angst, J. (1995). Comorbidity and social phobia: evidence from clinical, epidemiologic, and genetic studies. *European Archives of Psychiatry and Clinical Neuroscience, 244*, 297–303.

Mulder, R. T., Joyce, P. R., Frampton, C. M. A., et al. (2006). Six months of treatment for depression: outcome and predictors of the course of illness. *American Journal of Psychiatry, 163*, 95–100.

Persons, J. B., & Burns, D. D. (1985). Mechanisms of action of cognitive therapy: the relative contributions of technical and interpersonal interventions. *Cognitive Therapy and Research, 9*, 539–551.

Peterson, A. L., & Halstead, T. S. (1998). Group cognitive behavior therapy for depression in a community setting: a clinical replication series. *Behavior Therapy, 29*, 3–18.

Pinheiro, P., Bates, D., DebRoy, S., Sarkar, D., & , the R Core Team. (2009). *nlme: Linear and nonlinear mixed effects models*. R package version 3.1-96.

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*, 741–756.

R Development Core Team. (2010). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing, ISBN 3-900051-07-0. http://www.R-project.org URL.

Roberts, J. E., Shapiro, A. M., & Gamble, S. (1999). Level and perceived stability of self-esteem prospectively predict depressive symptoms during psychoeducational group treatment. *British Journal of Clinical Psychology, 38*, 425–429.

Rodebaugh, T. L. (2009). Social phobia and perceived friendship quality. *Journal of Anxiety Disorders, 23*, 872–878.

Satterfield, W. A., & Lyddon, W. J. (1998). Client attachment and the working alliance. *Counseling Psychology Quarterly, 11*, 407–416.

Schneier, F. R., Heckelman, L. R., Garfinkel, R., Campeas, R., Fallon, B. A., Gitow, A., et al. (1994). Functional impairment in social phobia. *Journal of Clinical Psychiatry, 55*, 322–331.

Sherbourne, C. D., & Wells, K. B. (1997). Course of depression in patients with comorbid anxiety disorders. *Journal of Affective Disorders, 43*, 245–250.

Smits, J. A. J., Minhajuddin, A., & Jarrett, R. B. (2009). Cognitive therapy for depressed adults with comorbid social phobia. *Journal of Affective Disorders, 114*, 271–278.

Sotsky, S. M., Glass, D. R., Shea, M. T., Pilkonis, P. A., Collins, J. F., Elkin, I., et al. (1991). Patient predictors of response to psychotherapy and pharmacotherapy: findings in the NIMH treatment of depression collaborative research program. *American Journal of Psychiatry, 148*, 997–1008.

Speer, D. C., & Greenbaum, P. E. (1995). Five methods for computing significant individual client change and improvement rates: support for an individual growth curve approach. *Journal of Consulting and Clinical Psychology, 63*, 1044–1048.

Stein, M. B., Fuetsch, M., Muller, N., Hofler, M., Lieb, R., & Wittchen, H. U. (2001). Social anxiety disorder and the risk of depression: a prospective community study of adolescents and young adults. *Archives of General Psychiatry, 58*, 251–256.

Tang, T. Z., & DeRubeis, R. J. (1999a). Sudden gains and critical sessions in cognitive-behavioral therapy for depression. *Journal of Consulting and Clinical Psychology, 67*, 894–904.

Tang, T. Z., & DeRubeis, R. J. (1999b). Reconsidering rapid early response in cognitive-behavioral therapy for depression. *Clinical Psychology: Science and Practice, 6*, 283–288.

Turner, S. M., Beidel, D. C., & Cooley, M. R. (1994). *Social effectiveness therapy: A program for overcoming social anxiety and social phobia*. Mt. Pleasant, SC: Turndel.

Woody, S. R. (1996). Effects of focus of attention on social phobics' anxiety and social performance. *Journal of Abnormal Psychology, 105*, 61–69.

Woody, S. R., & Adessky, R. S. (2002). Therapeutic alliance, group cohesion, and homework compliance during cognitive-behavioral group treatment of social phobia. *Behavior Therapy, 33*, 5–27.

Woody, S. R., Chambless, D. L., & Glass, C. R. (1997). Self-focused attention in the treatment of social phobia. *Behaviour Research and Therapy, 35*, 117–129.