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Mediation of Changes in Anxiety and Depression During Treatment of Social Phobia

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To investigate the interactive process of changes in social anxiety and depression during treatment, the authors assessed weekly symptoms in 66 adult outpatients with social phobia (social anxiety disorder) who participated in cognitive–behavioral group therapy. Multilevel mediational analyses revealed that improvements in social anxiety mediated 91% of the improvements in depression over time. Conversely, decreases in depression only accounted for 6% of the decreases in social anxiety over time. Changes in social anxiety fully mediated changes in depression during the course of treatment. The theoretical and clinical implications of these findings for the relationship between anxiety and depression are discussed.

Keywords: Social anxiety, social phobia, depression, cognitive–behavior therapy, treatment mechanisms, mediator of change

The central question facing those currently engaged in the research of psychotherapeutic interventions is, "Which treatments work, for whom, and under what conditions?" (Paul, 1967). Answering this question requires an understanding of the active ingredients or mechanisms of action underlying empirically supported interventions, and an essential step in the pursuit of these mechanisms is the identification of variables that mediate treatment outcomes.

Social phobia (social anxiety disorder), the third most prevalent psychiatric disorder (Kessler et al., 1994), is characterized by extreme fear of negative evaluation in social and performance situations (American Psychiatric Association, 1994). The most efficacious psychological interventions for social phobia are exposure-based cognitive–behavioral treatments (Hofmann & Barlow, 2002). Several controlled studies have established the efficacy of cognitive–behavioral therapy (CBT) for social phobia (e.g., Gould, Buckminster, Pollack, Otto, & Yap, 1997). However, the treatment mechanisms underlying the efficacy of CBT for social phobia are still not well understood. Moreover, a proportion of socially phobic individuals do not seem to benefit from CBT and fail to show marked symptom reduction by the end of treatment (e.g., Heimberg et al., 1998). As a result, there has been increased interest in the examination of variables that may mediate and moderate treatment outcomes in social phobia (e.g., Hofmann, 2000).

Within this context, researchers have investigated the role of comorbid depression in social phobia. Chambless, Tran, and Glass (1997) examined the effects of several variables on treatment outcome in 62 patients with social phobia who participated in 12 sessions of cognitive–behavioral group therapy (CBGT; Heimberg, 1991). Patients were assessed at pre- and posttreatment, and at 6-month follow-up on a number of measures, including self-report questionnaires and behavioral tests. Results indicated that of all the variables examined, depression symptom severity was the most powerful predictor of treatment success: Patients who reported greater pretreatment scores on the Beck Depression Inventory were less likely to benefit from treatment.

Erwin, Heimberg, Juster, and Mindlin (2002) compared CBGT response in three groups of social phobia patients: those with a primary diagnosis of social phobia and no comorbid diagnoses, those with a primary diagnosis of social phobia and an additional anxiety disorder diagnosis, and those with a primary diagnosis of social phobia and an additional mood disorder diagnosis. They found that social phobia patients with comorbid mood disorders, but not comorbid anxiety disorders, were more severely impaired than those with no comorbid diagnosis both before and after 12 weeks of CBGT. However, the rate of improvement in therapy, when measured pre- and posttreatment, was the same in both groups.

Despite these inconsistent findings, it remains important to understand the relationship between social phobia and depression, and specifically, whether and how symptoms of depression impact therapeutic processes and outcomes in patients with social phobia. Understanding this relationship is significant because individuals with social phobia are frequently also depressed. This finding has emerged consistently in the literature, both in large-scale epidemiological studies (Kessler et al., 1994; Regier, Rae, & Narrow, 1998; Robins & Regier, 1991) and in numerous investigations of diagnostic comorbidity among clinical outpatients (Alpert et al.,

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1999; Brown, Campbell, Lehman, Grisham, & Mancill, 2001; van Ameringen, Mancini, Styan, & Donison, 1991). Findings from the National Comorbidity Survey revealed that 34.2% of individuals with a lifetime diagnosis of social phobia had a history of mood disorders in comparison with only 14.5% of people who had never been socially phobic (Kessler, Stang, Wittchen, Stein, & Walters, 1999). Brown, Campbell, et al. (2001) in interviews of 1,127 outpatients found that current and lifetime diagnoses of social phobia were associated with a significantly elevated risk of major depressive disorder (MDD) and dysthymia. Of the 449 patients in their sample with a lifetime diagnosis of social phobia, those who met diagnostic criteria for MDD and dysthymia at some point in their lives numbered 269 (60%) and 90 (20%), respectively (Brown, Campbell, et al., 2001). The onset of social phobia typically occurs before that of depression (e.g., Brown, Campbell, et al., 2001; Ingram, Ramel, Chavira, & Scher, 2001; van Ameringen et al., 1991), and there is evidence to suggest that a diagnosis of social phobia increases the risk of subsequent depression (Alpert et al., 1999; Stein et al., 2001).

Despite the frequent co-occurrence of social phobia and depression in clinical outpatients, little is known about the interactive process of symptom changes in social anxiety and depression during therapy. A recent study by Persons, Roberts, and Zalecki (2003) examined session-by-session symptom changes in anxiety and depression among 58 outpatients who received individual CBT for a variety of anxiety and mood disorders, although not specifically social phobia. The authors demonstrated that self-reported symptoms of anxiety and depression were highly predictive of one another and more strongly correlated when measured in the same session than when measured at different session-by-session time points. On the basis of these findings, they argued that anxiety and depression change together during the course of treatment and that these nosologically distinct diagnoses may actually represent variants of a unitary, underlying disorder. However, Persons and colleagues (2003) did not differentiate between patients with principal diagnoses of anxiety disorders and those with principal mood disorders, or between patients who received particular treatment components designed to target mood disturbances and those who participated in therapies intended specifically to ameliorate symptoms of anxiety.

In the present study, we used multilevel mediational analyses to investigate session-by-session changes in symptoms of social anxiety and depression in patients with a principal diagnosis of social phobia who participated in weekly sessions of cognitive–behavioral group therapy. The results reported here are from a large clinical trial that examined the role of cognitions in the treatment of social phobia. Thirty of the 66 participants from the present study were also used for other analyses reported in two previous studies. Results of the analyses examining changes from pretest to posttest in self-perception through the use of open-ended thought listings are published in Hofmann, Moscovitch, Kim, and Taylor (2004). Results of the analyses examining short-term (from pre- to post) changes in cognitions on longer term (from pre- to follow-up) changes in social anxiety are reported in Hofmann (2004).

By examining the session-by-session changes in social anxiety and depression during CBT, the present study attempts to extend the body of research on social phobia and comorbid depression beyond the questions of whether depression predicts poor treatment outcome in patients with social phobia (e.g., Chambless et al., 1997; Erwin et al., 2002) and whether general measures of anxiety and depression are highly correlated at session-by-session time points (e.g., Persons et al., 2003). We sought to investigate the interactional process and sequence of symptom changes in social anxiety and depression during treatment for social phobia. When controlling for diagnosis and treatment type, do anxiety and depression change simultaneously during CBT, or do initial changes in social anxiety mediate subsequent improvements in depression? This question, which is the focus of our study, can only be addressed by measuring symptoms of social anxiety and depression at frequent intervals during the course of therapy rather than in the context of a pre–post design.

We tested two multilevel mediational models, seeking to distinguish between the following four possible outcomes that might characterize the interactional process of symptom changes in social anxiety and depression during treatment: (a) changes in social anxiety mediate changes in depression but not vice versa, (b) changes in depression mediate changes in social anxiety but not vice versa, (c) changes in both social anxiety and depression mediate changes in the other, and (d) changes in social anxiety are unrelated to changes in depression.

Method

Participants

Sixty-six adult outpatients who met *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV*; American Psychiatric Association, 1994) criteria for a principal diagnosis of social phobia participated in this study. All participants were assessed by trained doctoral students with the Anxiety Disorders Interview Schedule for *DSM–IV*—Lifetime version (ADIS–IV–L; DiNardo, Brown, & Barlow, 1994). Eligible participants were selected from a data pool of patients who participated in a large treatment outcome study at the Center for Anxiety and Related Disorders at Boston University. Exclusion criteria for the original study consisted of the following (a) prior nonresponse to an adequately delivered treatment at our clinic, (b) current diagnosis of psychoactive substance abuse or dependence, (c) current active suicidal potential, (d) current diagnosis of bipolar disorder, (e) current diagnosis of schizophrenia, and (f) current diagnosis of other psychotic disorders. Following the criteria used by Persons et al. (2003), we included participants in the present study who completed at least four sessions of self-reported measures of anxiety and depression during their participation in therapy. These measures were completed prior to each treatment session. All participants received the same type of treatment, which is described in the *Treatment* section.

The majority of participants were male ($n = 37$), unmarried ($n = 46$), and Caucasian ($n = 60$). Non-Caucasian participants identified themselves as Asian ($n = 2$), African American ($n = 1$), Hispanic ($n = 1$), and Other ($n = 2$). At intake, participants were, on average, 32.06 ($SD = 9.26$) years old and had completed 16.19 ($SD = 2.37$) years of education. Participants' mean annual income was \$47,970 ($SD = \$52,485$), with a fairly balanced distribution across different income brackets (\$0–\$20,000 [$n = 15$]; \$22,000–\$48,000 [$n = 21$]; \$50,000–\$96,000 [$n = 10$]; \$100,000 and above [$n = 8$]; missing data [$n = 12$]).

Sixty-seven percent of the sample ($n = 44$) met criteria for the generalized subtype of social phobia. Forty-two percent of participants ($n = 28$) received an additional clinical diagnosis (ADIS–IV–L clinical severity rating [CSR] ≥ 4 on a 0–8 scale) secondary to social phobia, with 18% ($n = 12$) receiving more than one additional diagnosis. There were 65 total comorbid diagnoses in the sample. The most common comorbid diagnoses were MDD ($n = 12$), generalized anxiety disorder ($n = 7$), dysthymia ($n =$

4), obsessive–compulsive disorder ($n = 4$), and panic disorder with agoraphobia ($n = 3$).

Treatment

CBGT for social phobia is a comprehensive treatment package based on the cognitive model of anxiety (e.g., Beck, Emery, & Greenberg, 1985). Therapy includes psychoeducation, in-session exposure simulations, cognitive restructuring, and homework assignments. CBGT has been shown to be efficacious for treating social phobia (e.g., Heimberg et al., 1998) and is currently listed as an "empirically supported treatment" for social phobia by the Society of Clinical Psychology (Division 12 of the American Psychological Association) Task Force on Promotion and Dissemination of Psychological Procedures. In this study, treatment consisted of 12 weekly 2.5-hr sessions conducted in groups of 5–7 patients by 2–3 therapists who followed structured treatment protocols in their delivery of the therapy.

Measures

The ADIS–IV–L (DiNardo et al., 1994) is a widely used, semistructured interview designed to establish reliable diagnoses of *DSM–IV* anxiety, mood, somatoform, and substance-use disorders and to screen for the presence of other conditions such as psychotic disorders. Although interrater reliability was not tested in the present sample, the ADIS–IV–L has demonstrated high reliability for diagnosing social phobia ($\kappa = .77$) and other Axis I disorders (Brown, DiNardo, Lehman, & Campbell, 2001).

The Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987) is a psychometrically well-validated (e.g., Heimberg et al., 1999) 24-item clinician-administered scale that evaluates fear and avoidance of 11 social interaction (e.g., talking with people one does not know very well) and 13 performance (e.g., returning goods to a store, eating in public places) situations. In this study, participants completed the self-report version of the LSAS (LSAS–SR) prior to each treatment session. They were given the following written instructions: *Please rate how fearful and anxious you are and how much you avoid the following 24 situations. Please rate each situation by using a scale from 0 (*no anxiety/avoidance*) to 100 (*severe anxiety/avoidance*).*[1] The mean fear rating across all of the items was used to compute the LSAS–SR Total Fear score, which was the primary measure of social anxiety used in this study. The LSAS–SR has shown good test–retest reliability, high internal consistency, and good convergent and discriminant validity (Baker, Heinrichs, Kim, & Hofmann, 2002; Fresco et al., 2001). In addition, Baker et al. (2002) reported that the scale was sensitive to treatment change. In the present study, the LSAS–SR Fear scale demonstrated excellent internal consistency ($\alpha = .94$).

Participants also completed the Beck Depression Inventory (BDI; Beck, Rush, Shaw, & Emery, 1979) before each session. The BDI is a 21-item self-report inventory that measures the severity of symptoms of depression. A recent evaluation of the BDI among patients with social phobia suggests good internal consistency ($\alpha = .89$) and test–retest reliability (intraclass correlation = .91) as well as adequate convergent and divergent validity (Coles, Gibb, & Heimberg, 2001). Although it is a nonspecific measure (Kendall, Hollon, Beck, Hammen, & Ingram, 1987), the BDI is capable of discriminating between social phobia patients with and without a comorbid mood disorder (Coles et al., 2001). In the present study, the BDI demonstrated good internal consistency ($\alpha = .91$).

Finally, participants completed the Social Phobia and Anxiety Inventory (SPAI; Turner, Beidel, Dancu, & Stanley, 1989) before and after treatment. The SPAI is a psychometrically well-validated (e.g., Beidel, Borden, Turner, & Jacob, 1989; Beidel, Turner, & Stanley, 1989; Turner, Stanley, & Beidel, 1989) 109-item measure that assesses cognitive, physical, and behavioral dimensions of social anxiety as well as agoraphobic avoidance. The measure consists of the Social Phobia and Agoraphobia subscales, each of which is scored separately. In this study, we used the total score, which is computed by subtracting the Agoraphobia subscale from the Social Phobia subscale. Pre- and posttreatment descriptive statistics are provided in Table 1.

Data Analyses

The longitudinal nature of our design produced a multilevel or nested data structure (Kenny, Kashy, & Bolger, 1998; Raudenbush & Bryk, 2001). The lower level, or Level 1 data, consisted of the repeated measures that were collected at each treatment session (i.e., social anxiety and depression). The Level 1 data were nested within upper level, or Level 2, units (i.e., participants). Thus, our data structure was comprised of repeated measures (Level 1 data) nested within individuals (Level 2 data). This data structure is appropriate for contemporary growth curve modeling techniques (see Collins & Sayer, 2001).

The focus of these innovative approaches to analyzing longitudinal data is to examine how an individual's score or standing on a variable changes as a function of time. Therefore, we used the hierarchical linear and nonlinear modeling software program (Raudenbush, Bryk, Cheong, & Congdon, 2001) to examine change over time in a multilevel random coefficient regression framework (see Raudenbush, 2001a, 2001b). An advantage of the multilevel random coefficient regression framework is that it handles unbalanced designs efficiently, allowing the number of observations to vary across participants.

To examine mediation of change, we followed the procedures originally described by Baron and Kenny (1986) and recently applied to a multiple-level framework by Kenny, Korchmaros, and Bolger (2003). As described by Kenny et al. (2003), four statistical criteria must be present in order to establish mediation:

1. The predictor variable must be significantly related to the mediator (path a in Figures 1 and 2).
2. The predictor variable must be significantly related to the outcome variable (path c).
3. When the outcome is regressed simultaneously on the predictor and mediator, the mediator must be significantly related to the outcome (path b).
4. The relationship between the predictor and the outcome with the mediator in the regression equation (path c′) must be significantly attenuated compared to when the outcome was regressed only on the predictor (i.e., path c).

In the present study, we tested two mediational models. In the hypothesized mediational model, we examined whether changes in depression were mediated by changes in social anxiety. In this model, time (with the number of CBGT sessions as the indicator of time) was the predictor variable, social anxiety was the mediator, and depression was the outcome variable. Because the predictor, mediator, and outcome were all Level 1 variables, these analyses examined lower level mediation (Kenny et al., 2003). We also tested an alternate mediational model (i.e., whether changes in anxiety were mediated by changes in depression) by switching the positions of the mediator and outcome in the analyses, a procedure described by Baron and Kenny (1986) as *reverse mediation.*

The strength of mediation (or indirect effect) was indexed in two ways. First, we used the procedure described by Sobel (1982; recently discussed

[1] The LSAS–SR scale of 0–100 used here diverges from the 0–3 scale used in previous studies. Our goal in this study was to allow for a fine-grained analysis of changes in social anxiety during treatment. Thus, we used the 0–100 scale to maximize the sensitivity of detecting even slight symptom changes during treatment. The correlation between the SPAI at pretreatment and the LSAS–SR at Session 1 was moderately strong ($r = .67$, $p = .00$), and significantly larger than the correlation between the SPAI at pretreatment and the BDI at Session 1 ($r = .26$, $p = .04$; $Z_{diff} = -4.36$, $p = .00$). These results support the convergent validity of the LSAS–SR in the present study.

Table 1
Descriptive Statistics for Pre- and Posttreatment Measures Completed by Study Participants

Measure	Pretreatment		Posttreatment	
	M	*SD*	*M*	*SD*
BDI	10.15	8.55	6.79	9.89[b]
LSAS–SR	46.13	17.84[a]	31.30	21.09[b]
SPAI[c]	109.53	22.82	82.08	29.67
ADIS CSR (social phobia)[c]	5.52	0.77	3.88	1.06
Completed treatment sessions[d]			9.06	2.79

Note. BDI = Beck Depression Inventory; LSAS–SR = Liebowitz Social Anxiety Scale—Self-Report version; SPAI = Social Phobia Anxiety Inventory; ADIS CSR = Anxiety Disorders Interview Schedule Clinical Severity Rating.
[a] First treatment session for 66 participants. [b] Multilevel regression estimate at final treatment session (represents the average score on the outcome variable at the final session for all 66 participants). [c] On the basis of 42 participants assessed at both pre- and posttreatment. [d] Sessions in which both LSAS–SR and BDI measures were completed.

in a multilevel framework by Krull & Mackinnon, 1999) to test the significance of the indirect path from the predictor to the outcome through the mediator (i.e., the product of paths a and b). Sobel's test is an index of the strength of the indirect effect of the predictor on the outcome through the mediator. Second, we calculated the percentage of the total effect between the predictor and outcome that was accounted for by the mediated (or indirect) effect, a procedure described by Kenny et al. (2003)[2] for lower level mediation in multilevel models. Both Sobel's test and percentage mediation provide an index of the strength of mediation.

Results

Response to CBGT

Forty-two participants completed the SPAI both pre- and posttreatment. A paired samples *t* test indicated that SPAI scores significantly decreased during the course of treatment (SPAI-pre–SPAI-post = 27.45), $t(41) = 7.50$; $p = .00$, $\eta^2 = .58$. At pretreatment, 38 (91%) of these participants scored above Turner et al.'s (1989) social phobia total score cutoff of 80 on the SPAI, whereas at posttreatment only 23 (54.8%) of these participants scored above 80. Forty-two participants completed both pre- and posttreatment ADIS assessments. At pretreatment, all 42 participants received a diagnosis of social phobia with a CSR ≥4. At posttreatment, 25 (64%) of these participants received a social phobia diagnosis with a CSR ≥4. This represents a significant decrease in CSRs ($M_{pre} = 5.52$ vs. $M_{post} = 3.88$), $t(41) = 9.14$, $p = .00$, $\eta^2 = .67$. At pretreatment, 19 (45.2%) of these 42 participants received comorbid mood diagnoses (8 with CSRs <4; 11 with CSRs ≥4), whereas at posttreatment, this number fell to 12 (1 with a CSR <4; 11 with CSRs ≥4).

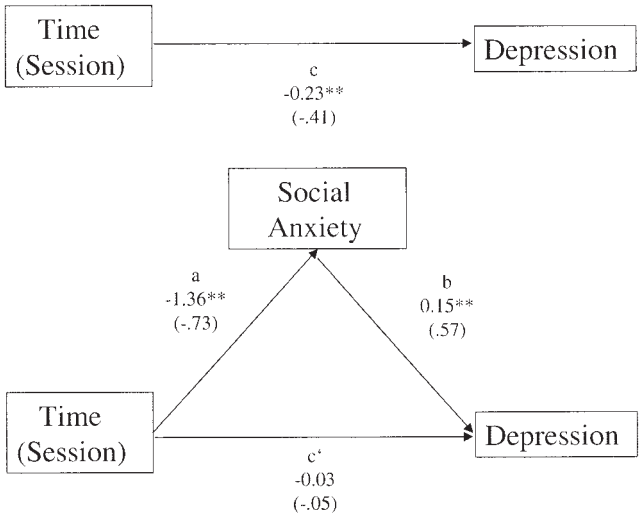

Figure 1. Results of the hypothesized mediational model with social anxiety as a mediator between time and depression (unstandardized regression coefficients presented on top and partial regression coefficients presented in parenthesis; $^{**}p < .01$).

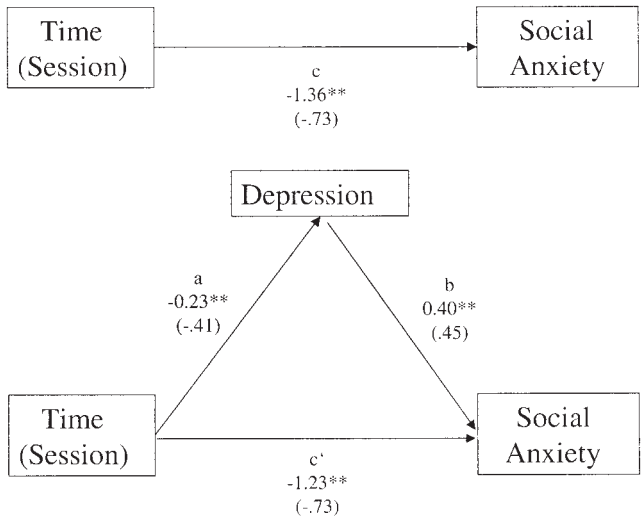

Figure 2. Results of the alternate mediational model with depression as a mediator between time and social anxiety (unstandardized regression coefficients presented on top and partial regression coefficients presented in parenthesis; $^{**}p < .01$).

Mediational Analyses

The results of the analyses for the hypothesized mediational model are presented graphically in Figure 1. The complete statistical results are presented in Table 2. As shown in both the table and figure, when time was entered into the Level 1 regression

[2] The total effect of the predictor on the outcome variable is the sum of the indirect effect and the direct effect (i.e., total effect = ab + c′). In multilevel analyses, the calculation of the total effect of the predictor on the outcome needs to be adjusted for the covariance between paths a and b across upper level units (Kenny et al., 2003). Thus, the covariance between a and b was included in the calculation of the total effect for the current analyses (i.e., total effect = ab + c′ + the covariance between a and b). Therefore, the percent mediation was calculated by the following formula: percent mediation = 100 × [(ab + c′ + the covariance between a and b) – c′]/[(ab + c′ + the covariance between a and b)], or 100 × total effect—c′/(total effect). The covariance between a and b was computed by the procedure described in Kenny et al. (2003).

Table 2
Summary of Level 1 Regression Analyses for the Hypothesized Mediational Model

Step	Path	Predictor variable	Outcome variable	*B*	*SE B*	*T*	pr
1	C	Time	Depression	−0.23	0.06	−3.64	0.41
2	A	Time	Social anxiety	−1.36	0.16	−8.65	0.73
3	B	Social anxiety	Depression	0.15	0.03	5.54	0.57
	C′	Time	Depression	−0.03	0.07	−0.42	0.05

Note. $T < -1.96$ or $T > 1.96$ indicates statistical significance at $p < .05$. pr = partial regression coefficient.

equation predicting depression (path c), the regression coefficient indicated that depression decreased significantly during the course of treatment ($B = -0.23$, $pr = .41$, $p = .00$, $\eta^2 = .17$).[3] When time was entered into the Level 1 regression equation predicting social anxiety (path a), the regression coefficient indicated that social anxiety also decreased significantly during treatment ($B = -1.36$, $pr = .73$, $p = .00$, $\eta^2 = .53$). The effect sizes for these two regression analyses suggest that the decrease across treatment sessions was stronger for social anxiety than for depression (social anxiety: $\eta^2 = .53$; depression: $\eta^2 = .17$). When controlling for social anxiety, time was no longer a significant predictor of depression (path c′); however, social anxiety was a significant predictor of depression at this step (path b: $B = .15$, $pr = .57$, $p = .00$, $\eta^2 = .32$), suggesting that participants who were more socially anxious also tended to be more depressed when controlling for time.

The drop in the regression coefficient for time predicting depression when social anxiety was not in the regression equation compared with when social anxiety was in the equation ($B = -.23$ vs. $B = -.03$) is one indication of mediation. Though the regression coefficient was statistically significant at the $p < .01$ level without social anxiety in the equation, its significance level rose above the statistical cutoff of $p < .05$ when social anxiety was added, a condition referred to as *full* or *complete mediation* (partial mediation occurs when the path decreases but remains significant when controlling for the mediator).

Sobel's test of the indirect effect of time on depression via social anxiety was significant (ab = −.20), $t(62) = -4.66$, $pr = .50$, $p = .00$, $\eta^2 = .25$. Ninety-one percent of the relationship between time and depression was mediated by changes in social anxiety. Thus, all indicators of mediation demonstrated that improvements in social anxiety mediated improvements in depression during the course of treatment.

Next, we examined the alternate mediational model by switching the position of the mediator and the outcome variable in the analyses (i.e., reverse mediation). Figure 2 presents the path diagram of the mediation results, and Table 3 includes the complete statistical results. The regression coefficient for time predicting social anxiety did drop when depression was entered into the equation ($B = -1.36$ vs. $B = -1.23$), and Sobel's test of the indirect path was significant (ab = −.09), $t(62) = -2.69$, $pr = .32$, $p = .01$, $\eta^2 = .10$. However, the relationship between time and social anxiety remained significant, an outcome indicative of only partial mediation. Furthermore, percentage mediation was only 6%. By comparison, percentage mediation was 91% in the hypothesized mediational model.

Because some individuals in the overall sample had little or no symptoms of depression, we examined next whether the mediation model varied as a function of depression as a diagnostic category. For this purpose, we categorized participants into three groups on the basis of their diagnostic status at intake: (a) participants who received an additional diagnosis of clinical depression (MDD, dysthymia, or depressive disorder NOS with ADIS–IV–L CSR ≥4) secondary to social phobia ($n = 16$), (b) participants who had subclinical symptoms of depression (i.e., CSRs of 2 or 3; $n = 9$), and (c) participants with no clinical symptoms of depression ($n = 41$). We then inspected the BDI scores for the three groups and labeled the groups in accordance with Kendall et al.'s (1987) suggestions. The first group was labeled *depressed* (BDI scores: $M = 19.50$, $SD = 10.48$), the second group was labeled *dysphoric* (BDI scores: $M = 14.78$, $SD = 7.41$), and the third group was labeled *nondepressed* (BDI scores: $M = 7.75$, $SD = 5.24$). A one-way ANOVA examining group differences in BDI scores revealed a significant group effect, $F(2, 62) = 16.52$, $p < .001$, with post hoc Bonferroni comparisons indicating that BDI scores in the nondepressed group were significantly lower than the dysphoric group ($p = .03$) and the depressed group, $p = .00$. No significant differences were found between the dysphoric group and the depressed group ($p = .35$). Given their similarity in BDI scores, individuals classified as depressed and dysphoric were combined into one group labeled *depressed or dysphoric* in order to obtain a sample size that was sufficiently large to test the mediational model in subgroups of participants. The mean BDI score for this combined group was 17.80 ($SD = 9.60$).

In order to examine the mediational model in these two subgroups (nondepressed vs. depressed or dysphoric), dummy variables (0 and 1) were created and entered into the Level 2 component of the regression equations. To obtain estimates of the regression coefficients and standard errors for the Level 1 coefficients for each group, the regression analyses were conducted in two ways, once with each group entered as the comparison group. The results of the analyses for the hypothesized mediational model, which are presented in Table 4, revealed that the only path in this model that significantly differed between the groups was path b. The relationship between social anxiety and depression controlling for time in the two groups was $B_{\text{depressed/dysphoric}} = .23$ versus $B_{\text{nondepressed}} = .06$; $t_{\text{difference}}(61) = 3.86$, $p = .00$, $\eta^2 = .20$. For both groups in the hypothesized model, Sobel's tests revealed a significant indirect path from time to depression via social anxiety ($\text{ab}_{\text{depressed/dysphoric}} = -.36$, $t(61) = -3.92$, $p = .00$, $\eta^2 = .20$; $\text{ab}_{\text{nondepressed}} = -.08$, $t(61) = -2.06$, $p = .04$, $\eta^2 = .07$). In

[3] Because multilevel regression analyses do not produce standardized regression coefficients, we included partial regression coefficients for comparison on a common metric.

Table 3
Summary of Level 1 Regression Analyses for the Alternate Mediational Model

Step	Path	Predictor variable	Outcome variable	*B*	*SE B*	*T*	pr
1	C	Time	Social anxiety	−1.36	0.16	−8.65	0.73
2	A	Time	Depression	−0.23	0.06	−3.64	0.41
3	B	Depression	Social anxiety	0.40	0.10	4.01	0.45
	C′	Time	Social anxiety	−1.23	0.14	−8.60	0.73

Note. $T < -1.96$ or $T > 1.96$ indicates statistical significance at $p < .05$. pr = partial regression coefficient.

contrast, Sobel's tests for the alternate model (i.e., the indirect path from time to social anxiety through depression) were not statistically significant ($ab_{depressed/dysphoric} = -.10$, $t(61) = -1.64$, $p = .10$, $\eta^2 = .07$; $ab_{nondepressed} = -.06$, $t(61) = -1.59$, $p = .11$, $\eta^2 = .07$). These results suggest that the clinical status of depression did not significantly alter the mediational relationship between treatment changes in BDI scores and social anxiety.

Discussion

In the present study, we investigated interactive changes in social anxiety and depression among patients with social phobia who participated in weekly sessions of cognitive–behavioral group therapy. As expected, both social anxiety and depression improved significantly during treatment. However, anxiety and depression changed in a predominantly nonreciprocal manner, with decreases in social anxiety fully mediating (and accounting for 91% of) decreases in depression, and decreases in depression only partially mediating (and accounting for 6% of) decreases in social anxiety. These findings indicate, therefore, that during CBGT for social phobia, depression improves over the course of treatment because social anxiety improves, whereas social anxiety improves largely via mechanisms unrelated to the amelioration of depression.

Whether and how treatments designed specifically to target principal anxiety disorders also lead to changes in comorbid mood symptoms that are not explicitly the focus of therapy is an issue that is relevant to our understanding of the psychopathology and treatment of anxiety and depression, as well as how best to classify these disorders in the *DSM*. According to Clark and Watson's (1991) tripartite theory, high negative affect is a common factor that is shared by both anxiety and depression, whereas low positive affect and high autonomic arousal are uniquely characteristic of depression and anxiety, respectively. Research testing the validity of the tripartite model in outpatients with anxiety and mood disorders has found that social phobia and depression are both distinguished by high negative affect and low positive affect and that neither is characterized uniquely by physiological hyperarousal (Brown, Chorpita, & Barlow, 1998). Given the overlap in latent higher order trait dimensions between social phobia and depression, as well as the ubiquity of negative self-related cognitions in both disorders, advocates of the unitary view of anxiety and depression might predict that CBGT would tap shared elements of affective and cognitive distress in anxiety and depression, leading to reciprocal and simultaneous changes in both. Our results, however, suggest that in patients with social phobia, the CBGT treatment response is characterized by early, specific improvements in social phobia symptomatology, which, in turn, cause improvements in symptoms of depression.

Several limitations of this study should be acknowledged. Because of the absence of a wait-list control group, changes in anxiety and depression may have been caused by the passage of time rather than the effects of treatment per se. Future studies should administer weekly measures of anxiety and depression to participants in a wait-list condition. Additional limitations of our study were its use of single self-report measures of anxiety and depression that were administered multiple times over the course of treatment and its inclusion of low numbers of participants with diagnosable comorbid depression. Validity would have been enhanced by the use of multiple indicators and methods of measuring symptom changes during therapy. Yet, it should be noted that conducting thorough, consistent symptom measurement can be a difficult and time-consuming endeavor in the context of weekly therapy sessions and, although important, its implementation will

Table 4
Summary of Regression Analyses for the Hypothesized Mediational Model in Depressed or Dysphoric Versus Nondepressed Participants

Group	Step	Path	Predictor variable	Outcome variable	*B*	*SE B*	*T*	pr
Depressed/dysphoric	1	C	Time	Depression	−0.24	0.13	−1.91	0.24
	2	A	Time	Social anxiety	−1.54	0.26	−5.95	0.61
	3	B	Social anxiety	Depression	0.23[a]	0.04	5.21	0.55
		C′	Time	Depression	0.07	0.14	0.49	0.06
Nondepressed	1	C	Time	Depression	−0.22	0.06	−3.64	0.42
	2	A	Time	Social anxiety	−1.23	0.19	−6.46	0.64
	3	B	Social anxiety	Depression	0.06[a]	0.03	2.17	0.27
		C′	Time	Depression	−0.13	0.07	−2.00	0.25

Note. $T < -1.96$ or $T > 1.96$ indicates statistical significance at $p < .05$. pr = partial regression coefficient.
[a] Indicates a statistically significant difference between the two groups.

pose challenges for future studies. Future studies should, however, include a greater number of socially phobic patients with moderate to high levels of depression.

All patients in our sample had a principal diagnosis of social phobia and secondary symptoms of depression and received therapy specifically designed to target social anxiety. It is unclear, therefore, whether our results are generalizable to individuals with principal depression and secondary symptoms of social anxiety, or to those who receive more general forms of CBT that are not specifically structured to target social phobia. Also, although a range of socioeconomic backgrounds was represented in the sample, participants were almost exclusively Caucasian. Thus, our findings may not be generalizable to non-Caucasian individuals. Furthermore, the female to male ratio in the present study was 1:1.28. The proportion of men in our study was slightly higher than that in previous studies by Erwin et al. (2002; 1:1.07 [$N = 141$]), Chambless et al. (1997; 1:0.77 [$N = 62$]), and Persons et al. (2003; 1:0.66 [$N = 58$]). It should be pointed out, however, that the gender distribution in the present study was most similar to the Erwin et al. (2002) study, which included the greatest number of participants. Finally, comparisons of our results with those of Persons et al. (2003) are limited because their study examined general symptoms of anxiety and depression in patients treated with CBT naturalistically and individually in independent practice, whereas ours focused more specifically on symptoms of social anxiety and depression in patients treated in the context of a structured group treatment protocol for social phobia at a large outpatient clinic. It is possible that the differences in our results were a function of the differences between studies in the specificity of patient population and treatment type.

Despite its limitations, the present study represents, to our knowledge, the first multilevel mediational study examining the interactive, longitudinal relationship between social phobia and depression at multiple time points during empirically supported CBT. The results shed new light on existing research that has investigated the relationship between social phobia and depression (e.g., Chambless et al., 1997; Erwin et al., 2002), and suggest that in patients with social phobia, secondary symptoms of depression are ameliorated via effective, cognitive–behavioral treatment that targets primary symptoms of social anxiety.

Future research should investigate the mechanisms by which CBGT leads to reductions in social anxiety symptoms and by which decreases in social anxiety lead to improvements in secondary symptoms of depression. Consistent with the cognitive model, previous studies have shown that changes in social anxiety may be mediated by reductions in negative self-related thinking, particularly in patients' estimations of the costs of negative social events (e.g., Hofmann, 2004). How and why decreases in social anxiety mediate decreases in depression is an intriguing question that awaits empirical study. Current theories suggest that social anxiety blocks the path to positively reinforcing attachment relationships (Eng, Heimberg, Hart, Schneier, & Liebowitz, 2001). Thus, reduction in social anxiety may lead to improvements in depression through the mechanism of increased positive reinforcement in interpersonal domains.

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