Cognitive Therapy vs Interpersonal Psychotherapy in Social Anxiety Disorder

A Randomized Controlled Trial

Ulrich Stangier, PhD; Elisabeth Schramm, PhD; Thomas Heidenreich, PhD; Matthias Berger, MD; David M. Clark, DPhil

Context: Cognitive therapy (CT) focuses on the modification of biased information processing and dysfunctional beliefs of social anxiety disorder (SAD). Interpersonal psychotherapy (IPT) aims to change problematic interpersonal behavior patterns that may have an important role in the maintenance of SAD. No direct comparisons of the treatments for SAD in an outpatient setting exist.

Objective: To compare the efficacy of CT, IPT, and a waiting-list control (WLC) condition.

Design: Randomized controlled trial.

Setting: Two academic outpatient treatment sites.

Patients: Of 254 potential participants screened, 117 had a primary diagnosis of SAD and were eligible for randomization; 106 participants completed the treatment or waiting phase.

Interventions: Treatment comprised 16 individual sessions of either CT or IPT and 1 booster session. Twenty weeks after randomization, posttreatment assessment was conducted and participants in the WLC received 1 of the treatments.

Main Outcome Measures: The primary outcome was treatment response on the Clinical Global Impression Improvement Scale as assessed by independent masked evaluators. The secondary outcome measures were independent assessor ratings using the Liebowitz Social Anxiety Scale, the Hamilton Rating Scale for Depression, and patient self-ratings of SAD symptoms.

Results: At the posttreatment assessment, response rates were 65.8% for CT, 42.1% for IPT, and 7.3% for WLC. Regarding response rates and Liebowitz Social Anxiety Scale scores, CT performed significantly better than did IPT, and both treatments were superior to WLC. At 1-year follow-up, the differences between CT and IPT were largely maintained, with significantly higher response rates in the CT vs the IPT group (68.4% vs 31.6%) and better outcomes on the Liebowitz Social Anxiety Scale. No significant treatment × site interactions were noted.

Conclusions: Cognitive therapy and IPT led to considerable improvements that were maintained 1 year after treatment; CT was more efficacious than was IPT in reducing social phobia symptoms.

Arch Gen Psychiatry. 2011;68(7):692-700

Author Affiliations:
Department of Psychology, University of Frankfurt, Frankfurt (Dr Stangier); Department of Psychiatry and Psychotherapy, University of Freiburg, Freiburg (Drs Schramm and Berger); Department of Social Work, Health and Nursing, University of Applied Sciences, Esslingen (Dr Heidenreich), Germany; and Department of Psychology, Kings College London, London, England (Dr Clark).

SOCIAL ANXIETY DISORDER (SAD) is a common mental disorder that is associated with considerable vocational and psychosocial handicap and an increased risk of comorbid disorders, such as depression, other anxiety disorders, and alcohol abuse.[1,2] If untreated, SAD generally takes a long-term course.[3]

Biological, cognitive, and interpersonal factors has been implicated in the causes of SAD,[4,5] and each had led to the development of distinctive treatments. Among psychological treatments, group cognitive behavior therapies (CBTs) (Heimberg et al[6] and Davidson et al[7]) and individual cognitive therapy (CT) have been shown to be effective. Cognitive therapy is based on the cognitive model of Clark and Wells[8] of the maintenance of SAD. Efficacy has been demonstrated against exposure therapy, group CT, selective serotonin reuptake inhibitor treatment, and waiting-list control (WLC) conditions in 4 randomized controlled trials.[9-12]

Whereas the cognitive approach mainly emphasizes intrapersonal mechanisms, other researchers have more strongly emphasized interpersonal relationship patterns and the fulfillment of social roles in the maintenance of SAD.[13] Accordingly, interpersonal psychotherapy (IPT), which was originally developed by Klerman et al[14]

and Weissman et al[15] for unipolar depression and which focuses on the modification of dysfunctional patterns of interpersonal relationships, may represent a useful alternative to CT. Randomized controlled trials have established that IPT is effective in depression[16] and in eating disorders.[17] After encouraging results in an open trial[18] of patients with SAD, Lipsitz et al[19] in 2008 conducted a randomized controlled trial that confirmed the improvements observed with IPT in the open trial but found no significant differences between IPT and supportive therapy.

Few direct comparisons between CBTs and IPT have been conducted. The National Institute of Mental Health Treatment of Depression Collaborative Research Program[20] found that both treatments were effective, but in post hoc analysis, some evidence indicated that IPT was more effective with the most severely depressed patients. Two trials[17,21] of bulimia nervosa demonstrated the superior effectiveness of CBT over IPT at the posttreatment assessment but not at the 1-year follow-up. A Norwegian group[22] compared predominantly group-based versions of IPT and CT in patients with SAD in a residential setting and found limited, not significantly different, improvements of symptoms in both approaches. However, both treatments differed substantially from the individual IPT and CT programs that have received the strongest support in randomized controlled trials. Interpretation of the trial findings is further complicated by low therapist competency ratings.

The aim of the present study was to compare in SAD the short- and long-term efficacy of individual CT and IPT with that of a WLC condition. To control for therapy site allegiance effects and for capacity to deliver the treatments with a sufficient degree of competence,[23,24] the investigation was conducted at 2 research centers, 1 of which (Frankfurt, Germany) had previously specialized in CT and 1 of which (Freiburg, Germany) had previously specialized in IPT. Therapists at each site were trained to provide both treatments.

METHODS

DESIGN

At each trial site, patients were randomly assigned to the CT, IPT, or WLC group. Randomization was stratified according to site and presence or absence of comorbid depression. After patient eligibility was assessed and informed consent was obtained, patients were formally enrolled in the study. Allocation was based on a computer-generated list that was concealed from the investigators. Treatment comprised up to 16 individual sessions conducted on a mainly weekly basis. A booster session was offered 2 months after the end of treatment. The WLC group received treatment after a 20-week waiting period. The main assessment points were before treatment/wait, after treatment/wait, and 1 year after treatment completion. Two treatment sites that were each experienced in conducting trials with 1 of the 2 treatment approaches participated: Frankfurt University (CT; U.S. and T.H.) and Freiburg University (IPT; E.S. and M.B.). The study design, thus, included 3 factors: (1) treatment condition (CBT vs IPT vs WLC), (2) a repeated-measures factor (pretreatment vs posttreatment vs follow-up), and (3) treatment site (Frankfurt vs Freiburg) to control for any site allegiance effects.

PATIENTS

Participants were recruited via the private practices of psychiatrists and psychologists, outpatient clinics, and advertisements in local newspapers and on the Internet, with use of the different referral routes varying with the local circumstances of each site. All individuals interested in participating in the study took part in a telephone screening based on the Social Phobia Inventory.[25] Patients who seemed eligible were invited for a diagnostic interview. The study was approved by the ethical committees at the University of Frankfurt and the University of Freiburg. Participants were provided with a complete study description, and written consent was obtained.

Social anxiety disorder and other psychiatric diagnoses were assessed using *Structured Clinical Interview for DSM-IV* Axis I and Axis II disorders.[26-28] All the diagnostic evaluations were conducted by trained and certified clinical psychologists and were reviewed by senior study investigators (U.S., E.S., and T.H.). The 17-item Hamilton Rating Scale for Depression (HRSD)[29,30] was used to assess severity of depression. On the basis of 6 videotaped interviews, the intraclass correlation coefficient for the HRSD was 0.97.

Individuals were invited to participate if they met the following inclusion criteria: diagnosis of SAD according to the *DSM-IV,* any comorbid mental disorder provided that severity did not exceed that of SAD, and age 18 to 65 years. The exclusion criteria were psychosis, current substance dependency or abuse, Axis II personality disorders from the dramatic or odd cluster, severe depression (HRSD score >23), acute suicidality, current psychopharmacologic or other psychotherapeutic treatment, and preference for psychopharmacologic treatment.

Of 697 individuals who contacted the study centers, 254 were assessed by interview; 137 individuals were excluded owing to a failure to meet the inclusion criteria or for other reasons (**Figure 1**). Of 44 patients who refused to participate, 8 who met the inclusion criteria withdrew after signing the consent form but before randomization. The remaining 117 individuals met the inclusion criteria and were randomized. Thirty-eight participants were allocated to CBT, 38 to IPT, and 41 to WLC. Nineteen therapists (16 clinical psychologists and 3 psychiatrists) with advanced or completed psychotherapy/ clinical training participated in the trial. The 8 therapists treating patients receiving CT and 11 therapists treating patients receiving IPT had comparable levels of clinical experience (CT: 5.3 years; IPT: 6.6 years; $t_{17}=-0.73$, $P=.48$), experience with the treatment (CT or CBT: 4.5 years; IPT: 4.1 years; $t_{17}=0.78$, $P=.44$), and experience with the treatment of SAD (CT: 1.5 years; IPT: 1.5 years; $t_{17}=0.04$, $P=.97$). In each treatment condition, therapists received 40 hours of training workshops and adhered to treatment manuals (D.M.C., unpublished data, 1997; translated and revised by Stangier, Ehlers, and Clark[31]; J. D. Lipsitz, PhD, and J. C. Markowitz, PhD, unpublished data, 1996). The workshops for CT were conducted by 3 of us (U.S., T.H., and D.M.C.) and for IPT by Dr Lipsitz and one of us (E.S.). Each therapist treated at least 2 pilot cases under supervision before participating in the trial. Additional training in the form of detailed feedback on videotapes or case descriptions was provided by one of us (D.M.C.) and Dr Lipsitz. At both trial sites, continuous supervision was established for therapists in each condition. After reaching an adequate level of adherence, therapists treated an average of 4 patients each.

TREATMENTS

The treatments comprised 16 individual sessions conducted over 20 weeks. Most sessions were 50 minutes, but the protocol allowed therapists to extend up to 6 sessions to a maximum of
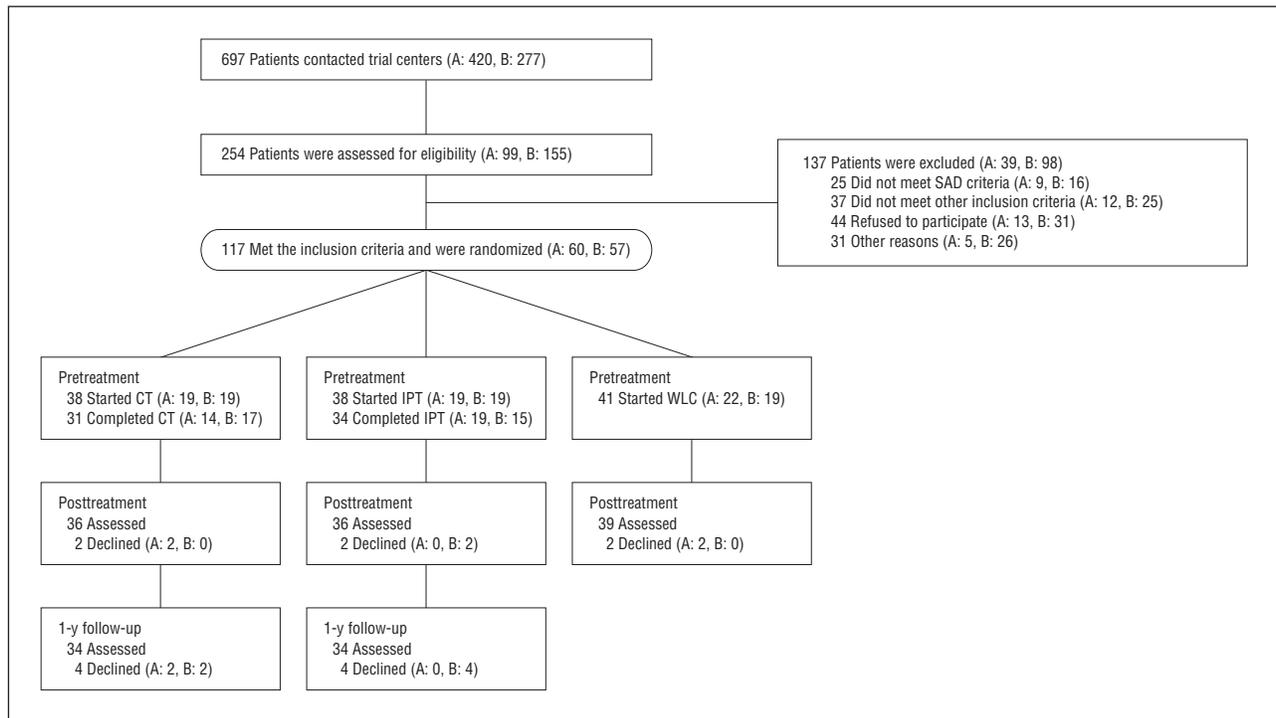
Figure 1. Flowchart of attrition. A indicates the Freiburg site; B, the Frankfurt site; CT, cognitive therapy; IPT, interpersonal psychotherapy; SAD, social anxiety disorder; and WLC, waiting-list control condition.

100 minutes to facilitate behavioral experiments (CT) or in-depth discussions and role-plays (IPT). With respect to mean session length, no significant differences between both treatments (mean [SD] number of minutes per session: IPT, 65.3 [9.8]; CT, 67.8 [14.4]; t_{50}, 0.77; $P = .45$). Both treatments were manualized (D.M.C., unpublished data, 1997; translated and revised by Stangier, Ehlers, and Clark[31]; J. D. Lipsitz and J. C. Markowitz, unpublished data, 1996). Patients on the waiting list received no treatment for 20 weeks, after which they were offered 1 of the 2 treatments. None of the patients received any other form of psychotherapy or pharmacotherapy during the treatment phase of the study. The sessions were videotaped. A randomly selected subset of CT videotapes was audited by one of us (D.M.C.), and written feedback was sent to the therapist. Adherence to the CT manual was reviewed by 2 of us (U.S. and T.H.) during routine, videotape-based supervision. Similarly, IPT videotapes were systematically checked by 1 of us (E.S.), and additional feedback was provided by Dr Lipsitz. The integrity and boundaries of each therapy were carefully monitored. Checklists of "encouraged" and "prohibited" interventions were completed by the therapist after each session to ensure that techniques unique to the other treatment were not applied.

Cognitive Therapy

The CT program was based on the cognitive model of SAD of Clark and Wells[8] and included the following components[8,9]: (1) establishing a personal version of the model using the patient's own thoughts, images, focus of attention, safety behaviors, and symptoms; (2) conducting role-play–based behavioral experiments to demonstrate the adverse effects of self-focused attention and safety behaviors; (3) practicing external focus of attention in nonsocial and social situations; (4) restructuring distorted self-imagery using videotape feedback and other methods; (5) discussing surveys providing feedback on

other people's beliefs about the significance of blushing, stuttering, sweating, etc; and (6) behavioral experiments to test negative beliefs in anxiety-provoking social situations while giving up safety behaviors and adopting an external focus of attention. Therapists were instructed not to use components of IPT, such as exploring and modifying interpersonal relationships or using role-plays to enhance communication of affect and social skills.

Interpersonal Psychotherapy

For SAD, IPT was based on a revised version of the standard manual[13,14] developed by Lipsitz and Markowitz (J. D. Lipsitz and J. C. Markowitz, unpublished data, 1996) and used in trials by Lipsitz et al.[18,19] During the first phase of treatment, the Interpersonal Inventory is conducted with the aim of relating social anxiety symptoms to 1 of the 4 problem areas. J. Lipsitz (written communication, 2002) replaced the problem area "social deficits" with the concept of "role insecurity/role deficits" as being more specific to SAD. Most commonly used in this trial was the area of role transition, either in terms of life changes or in terms of a therapeutic role transition. Therapeutic role transition means that the patient recognizes that SAD is not part of his or her personality but rather a temporary state or role. In the second stage of treatment, the formulated problem area is addressed by clarifying roles and their associated emotions, giving advice, using role-play if indicated, and encouraging the patient to communicate and express feelings. As in standard IPT, the interventions generally aim to enable the patient to build a social network by forming and maintaining close and trusting relationships. During the last phase of treatment, therapy completion is explicitly addressed, progress is discussed, and therapeutic gains are consolidated to prevent future relapses. In the present study, therapists were instructed not to use CT interventions for safety behaviors, attentional processes, behavioral experiments, and cognitive restructuring.

Table 1. Sample Characteristics

Characteristic	Cognitive Therapy (n = 38)	Interpersonal Psychotherapy (n = 38)	Waiting-List Control (n = 41)	P Value
Age, mean (SD), y	34.6 (12.9)	33.9 (9.5)	38.1 (12.9)	.15[a]
Female sex, No. (%)	17 (44.7)	22 (57.9)	26 (63.4)	.23[b]
High school diploma, No. (%)	25 (65.8)	25 (65.8)	22 (53.7)	.62[b]
Age at onset of SAD, mean (SD), y	13.1 (7.2)	14.8 (8.0)	18.3 (11.8)	.12[a]
Duration of SAD, mean (SD), y	19.7 (11.3)	18.6 (11.8)	16.8 (11.3)	.68[a]
Generalized subtype of SAD, No. (%)	25 (65.8)	21 (55.3)	21 (51.2)	.51[b]
Any additional Axis I diagnoses, No. (%)	21 (55.3)	24 (63.2)	19 (46.3)	.32[b]
Comorbid mood disorders, No. (%)	14 (36.8)	13 (34.2)	14 (34.1)	.96[b]

Abbreviation: SAD, social anxiety disorder.
[a] By analysis of variance.
[b] By χ^2 test.

ASSESSMENT PROCEDURES

The primary outcome measure was treatment response as assessed by the Clinical Global Impression Scale (CGI-I).[32] In agreement with Heimberg et al[6] and Davidson et al,[7] we chose CGI-I as the primary outcome measure because it is a standard primary outcome measure in psychopharmacologic studies and provides information that is of high clinical relevance. The psychometric properties of CGI-I have been found to be good.[33] Independent assessors masked to the treatment condition completed the 7-point rating scale at the posttreatment and 1-year follow-up assessments. Patients rated 1 or 2 (markedly or moderately improved) were classified as responders, and those rated 3 or higher were classified as nonresponders.

The secondary outcome measures were independent assessor ratings on the Liebowitz Social Anxiety Scale (LSAS)[34-36] and the HRSD[37] and the patient-completed Social Phobia and Anxiety Inventory (SPAI) (T. Fydrich, PhD, A. Scheurich, PhD, and E. Kasten, Dipl Psych, unpublished data, 1995). Each was completed at the pretreatment/wait, posttreatment/wait, and 1-year follow-up assessments. At the end of the first session, patients rated the credibility of their treatment using a rating scale developed by Borkovec and Nau.[38] In addition, a therapist version of this questionnaire was used to assess allegiance. After each therapy session, patients and therapists separately completed the Bernese Post-Session Report,[39] which includes satisfactorily reliable patient- and therapist-rated therapeutic alliance scales. For the present analysis, alliance ratings after the first therapy session were used.

STATISTICAL ANALYSES

Data were analyzed using a commercially available software package (SPSS; SPSS Inc, Chicago, Illinois). All the statistical analyses were intent-to-treat. Patients who were allocated to CT or IPT were considered to have had an adequate dose of therapy if they attended at least 12 (of 16) sessions. Individuals who attended fewer sessions were still assessed and included in the intent-to-treat analysis. Missing data were replaced using the last-observation-carried-forward approach. Categorical analyses were conducted using binary logistic regression. Dimensional measures were submitted to analyses of covariance in which pretreatment scores were controlled for. Analyses of covariance were performed separately for the posttreatment and 1-year follow-up assessments. To determine whether treatment site affected outcome, all the analyses included an estimation of site and treatment × site interaction effects. Statistical significance was set at $P < .05$ (2-tailed).

RESULTS

DESCRIPTION OF THE SAMPLE

Patient characteristics are given in **Table 1**. No significant differences were noted between treatment conditions regarding any of the sociodemographic or clinical variables. Fifty-eight percent of patients met the criteria for the generalized subtype of SAD. Fifty-four percent of patients also met the diagnostic criteria for 1 or more other current Axis I disorders: major depressive disorder (24.6%), dysthymia (13.6%), specific phobia (5.9%), and panic disorder (3.4%). Sixty-seven percent of patients met the criteria for 1 or more personality disorders, primarily avoidant type (50.8%).

TREATMENT AND ASSESSMENT COMPLIANCE

Figure 1 shows the flow of patients through the trial. Eleven of 76 patients (14.5%) attended fewer than 12 of 16 sessions and were considered to have received a suboptimal dose of treatment (7 patients receiving CT [18.4%] and 4 patients receiving IPT [10.5%], $\chi_1^2 = 0.96$, $P = .26$). Separate analyses for both sites reveal that no significant difference was noted between CT and IPT in the attrition rate in Frankfurt (CT = 2, IPT = 4; $\chi_1^2 = 0.79$, n = 38, $P = .66$), but in Freiburg, the rate of patients not receiving an adequate treatment dose was significantly higher for CT than for IPT (CT = 5, IPT = 0; $\chi_1^2 = 5.76$, n = 38, $P = .046$). For these patients, the number of sessions ranged from 2 to 10. Six patients (5%) did not attend the posttreatment/wait assessment interview and were coded as nonresponders. Eight of 76 patients (10.5%) did not participate in the 1-year follow-up assessment (CT = 4, IPT = 4). There were no suicides, suicide attempts, or other major adverse events.

TREATMENT CREDIBILITY, THERAPEUTIC ALLIANCE, AND ADHERENCE

No significant differences were noted between IPT and CT in either patient or therapist ratings of treatment credibility or in the quality of the therapeutic alliance. For
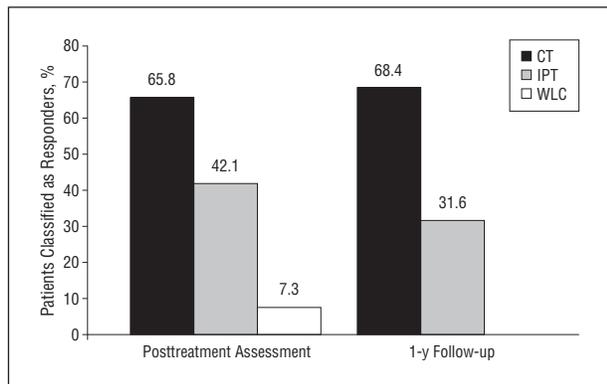
Figure 2. Percentage of patients in the intent-to-treat sample (n=117) classified as responders by independent assessors at the posttreatment and 1-year follow-up assessments according to study group. Treatment response ratings were based on the Clinical Global Impression Scale.[33] Treatment responders were defined by a change score reflecting marked or moderate improvement. CT indicates cognitive therapy; IPT, interpersonal psychotherapy; and WLC, waiting-list control.

both treatments, credibility and therapeutic alliance scores were high. Mean (SD) treatment credibility patient ratings were as follows: IPT, 7.47 (1.26); CT, 7.82 (1.34); $F_{1,67}=1.27$, $P=.26$. Mean (SD) therapist ratings were as follows: IPT, 8.2 (0.9); CT, 8.7 (0.7); $t_{17}=1.57$, $P=.14$. Mean (SD) quality of the therapeutic alliance ratings by patients were as follows: CT, 1.58 (0.68); IPT, 1.45 (0.65); $F_{1,67}=0.58$, $P=.45$.

Adherence to treatment protocols was assessed using the postsession therapeutic technique checklists. According to these checklists, therapists used interventions that were categorized as being unique to the relevant treatment only (CT: 54.5%, IPT: 65.5%) or that were shared by both treatments (CT: 45.4%, IPT: 35.5%). No interventions that were categorized as "prohibited" were reported.

TRIAL SITE EFFECTS

No significant site effects on the social phobia outcome measures were noted. However, there was a significant site effect on the HRSD at the posttreatment/wait assessment but not at the follow-up assessment. Irrespective of the intervention received, patients in Freiberg had higher HRSD scores at the posttreatment/wait assessment than did patients in Frankfurt. No significant treatment × site interaction effects were noted on any outcome measure at either the posttreatment/wait or the 1-year follow-up assessment. This means that the differences in outcomes among the 3 groups (CT, IPT, and WLC) reported later herein were not significantly affected by the site at which treatment was provided.

PRIMARY OUTCOME

Figure 2 shows the results for the primary outcome measure: the independent assessor ratings of treatment response using the CGI-I. At the posttreatment/wait assessment, 25 of the 38 patients (65.8%) who had undergone CT, 16 of the 38 (42.1%) who had undergone IPT, and 3 of the 41 (7.3%) in the WLC group were classified as re-

sponders. Both CT and IPT were superior to WLC (CT: Wald $\chi_1^2=21.4$, $P<.001$; IPT: Wald $\chi_1^2=10.55$, $P<.001$). In addition, CT proved superior to IPT (Wald $\chi_1^2=4.21$, $P=.04$). At 1-year follow-up, the difference between CT and IPT was maintained. Twenty-six of the 38 patients (68.4%) who had undergone CT and 12 of the 38 patients (31.6%) who had undergone IPT were classified as responders (Wald $\chi_1^2=9.82$, $P=.002$). During follow-up, significantly more patients who had received IPT sought additional psychological or pharmacologic treatment for SAD (CT, 12.1% of patients [1 psychological, 1 medication, and 2 combined treatment]; IPT, 38.2% of patients [7 psychological, 4 medication, and 2 combined treatment]; $\chi_1^2=6.03$, n=67, $P=.01$). From posttreatment assessment to 1-year follow-up, 15.2% of patients receiving CT changed from nonresponse to response and 9.1% of patients from response to nonresponse. In the IPT group, these rates were 8.8% and 20.6%, respectively. Because additional treatments during follow-up may have produced further improvement, we also analyzed the proportion of patients who were classified as responders at 1-year follow-up and had not received additional treatment for SAD. The difference between CT and IPT remained significant ($P<.01$). Responder proportions were 48.6% for CT and 18.4% for IPT.

SECONDARY OUTCOMES

Table 2 provides the secondary outcome measures. At the posttreatment/wait assessment, the independent assessor ratings on the LSAS indicated that patients who received either CT or IPT showed greater improvement than did patients in the WLC group. Cognitive therapy also proved superior to IPT. The SPAI showed a similar pattern of results, although the difference between CT and IPT did not reach significance ($P=.07$). On the HRSD, the CT and IPT groups showed greater improvement than did the WLC group and did not differ from each other. At 1-year follow-up, CT remained superior to IPT on the LSAS but did not differ from IPT on the SPAI ($P=.10$) or the HRSD ($P>.40$).

EFFECT SIZES

For the primary outcome measure (CGI-I responder status), number needed to treat (NNT) is an appropriate way of quantifying effect sizes. The NNT refers to the number of patients who need to be treated with the "more effective" intervention to obtain 1 more responder as if the same number of patients had received the "less effective" intervention. For the contrasts at the posttreatment/wait assessment, NNTs were as follows: 5 for CT vs IPT, 2 for CT vs WLC, and 3 for IPT vs WLC. At 1-year follow-up, the NNT for CT compared with IPT was 3. **Table 3** provides the controlled effect sizes for the secondary outcome measures.

COMMENT

The results of the present study suggest that CT and IPT are effective treatments for SAD. Each treatment was associated with significantly greater improvement com-

Table 2. Secondary Outcome Measures at the Pretreatment, Posttreatment, and Follow-up Assessments[a]

Assessment	Cognitive Therapy (n = 38)	Interpersonal Psychotherapy (n = 38)	Waiting-List Control (n = 41)	Statistic	P Value
LSAS score, mean (SD)					
Pretreatment	69.17 (23.36)	68.35 (22.60)	62.75 (26.76)	$F_{2,111} = 0.99$	.38
Posttreatment	39.49_A (21.09)	$48.16B$ (22.36)	59.90_C (29.05)	$F_{2,110} = 21.41$	<.001
1-y Follow-up	33.96_A (20.57)	$43.33B$ (25.18)	NA	$F_{1,71} = 5.72$	.02
HRSD score, mean (SD)					
Pretreatment	8.11 (5.43)	8.24 (5.93)	7.81 (6.06)	$F_{2,111} = 0.12$	.89
Posttreatment	5.43_A (5.74)	4.50_A (4.00)	$8.03B$ (6.13)	$F_{2,110} = 6.04$	<.001
1-y Follow-up	4.47 (5.39)	5.31 (4.93)	NA	$F_{1,71} = 0.63$	.43
SPAI score, mean (SD)					
Pretreatment	76.14 (16.98)	77.94 (15.44)	73.14 (23.52)	$F_{2,111} = 0.06$	.94
Posttreatment	51.20_A (20.61)	59.75_A (18.38)	69.19_B (28.26)	$F_{2,108} = 13.86$	<.001
1-y Follow-up	49.74 (24.06)	55.80 (19.82)	NA	$F_{1,70} = 2.68$	.11

Abbreviations: HRSD, Hamilton Rating Scale for Depression; LSAS, Liebowitz Social Anxiety Scale (total score); NA, not applicable; SPAI, Social Phobia and Anxiety Inventory.
[a]Within an assessment occasion, means with no subscript letters and those that share the same subscript letter do not differ. Means with nonoverlapping subscript letters differ at a level of at least $P < .05$. At the pretreatment assessment, the group effect is based on 2-way (treatment × site) analysis of variance. At all other assessment points, the group effect is based on 2-way (treatment × site) analysis of covariance, with pretreatment scores as the covariate.

Table 3. Controlled Effect Sizes at the Posttreatment and 1-Year Follow-up Assessments[a]

Measure	Posttreatment Assessment			Follow-up Assessment CT vs IPT
	CT vs WCL	IPT vs WLC	CT vs IPT	
LSAS	1.47	0.95	0.57	0.55
SPAI	1.21	0.79	0.42	0.38
HRSD	0.55	0.76	−0.21	−0.18

Abbreviations: CT, cognitive therapy; HRSD, Hamilton Rating Scale for Depression; IPT, interpersonal psychotherapy; LSAS, Liebowitz Social Anxiety Scale; SPAI, Social Phobia and Anxiety Inventory; WLC, waiting-list control.
[a]Controlled effect sizes were computed by dividing the difference between covariance-adjusted means by the square root of the average of the variances for the groups.

pared with the WLC group. In addition, a significant advantage was found for CT over IPT on the primary outcome measure (CGI-I responder status). At posttreatment assessment, 65.8% of patients treated with CT showed marked improvement in social-phobic symptoms compared with 42.1% of those treated with IPT. At 1-year follow-up, the superiority of CT over IPT persisted, with the former showing significantly higher response rates. We additionally observed a significantly higher rate of additional nonprotocol treatment during follow-up in the IPT vs the CT group.

To ensure a valid and fair comparison of treatments, we controlled for several potential sources of bias. First, therapeutic allegiance was controlled by using 2 research sites, 1 of which had previously specialized in IPT and 1 in CT. Second, therapists at both centers received training from acknowledged experts in each treatment. Third, the therapists who provided the 2 treatments had similar levels of clinical experience and were not significantly different in their expectations for the improvements in their patients, although it cannot be excluded that slight, nonsignificant differences in outcome expectations between IPT and CT therapists might reflect different acknowledgment of empirical support for the efficacy of the treatments (at the beginning of the study, 2 controlled studies evaluating CT[8,10] were opposed to 1

open trial of IPT[18]). Fourth, the quality of the therapeutic alliance, as rated by therapists and patients, was similar in the 2 treatments. Fifth, patients' expectations after the first session were similarly high in the 2 treatments. Sixth, overall treatment compliance rates did not differ between the 2 treatments. At the site that had previously specialized in IPT, more patients in the IPT group than those in the CT group received an adequate dose of treatment, but if this were important, one would expect a higher response rate to IPT, which is the opposite of what was found. Seventh, no significant treatment × site interaction effects were noted for the primary or secondary outcome measures. Overall, it seems that there is no good reason to suppose that observed differences in outcome between CT and IPT were caused by variation in allegiance or other common, nonspecific therapy factors.

Treatment integrity was supported by providing treatment manuals developed by 1 of us (D.M.C., unpublished data, 1997; translated and revised by Stangier, Ehlers, and Clark[31]; and Lipsitz and Markowitz, unpublished data, 1996). In addition, 1 of us (D.M.C.) and Dr Lipsitz conducted intensive workshops for both groups of therapists. The quality of each treatment was monitored by the primary investigators (U.S. and E.S.) and by experienced on-site supervisors. Finally, although inde-

pendent ratings from videotapes might be the best method to assess protocol adherence, the adherence checklists completed by the therapists after each treatment session indicated that the applied interventions agreed with the respective treatment manuals. These design elements add to the confidence with which results of the study are interpreted.

This is the first trial, to our knowledge, to demonstrate that IPT is superior to a no-treatment control condition in patients with SAD and, as such, provides further support for the efficacy of this approach. Recently, Lipsitz et al[19] found that IPT and supportive therapy were associated with substantial improvements in SAD and did not differ from each other. Although they did not directly report effect sizes, their IPT outcomes seem to correspond to the effect sizes for social-phobia measures. In addition, responder rates were also comparable with those found in the present study. There is, thus, evidence to assume that the IPT treatment in this study performed as well as in previous studies. The effect sizes for CT on social-phobia measures at the posttreatment assessment were somewhat lower than were those obtained in some preceding trials investigating individual CT based on the model by Clark et al,[8,9,11] and they were comparable with those reported by Mörtberg et al.[12] In addition, the effect sizes of CT and IPT in this trial were larger compared with the average effect sizes for psychological and pharmacologic treatments for SAD.[40-42] Because we excluded patients with severe depression, effect sizes were lower for depression than for social phobia measures, but both treatments had a significant effect in reducing depression. In contrast to the results for social anxiety, IPT was associated with a nonsignificantly greater reduction in depression than was CT. Thus, although IPT was less effective than CT in changing social anxiety, it was at least as successful in reducing depressive symptoms.

Which active ingredients of CT might have contributed to the larger effects on SAD compared with IPT? Because the 2 treatments differ with respect to the explicit targets for psychotherapeutic change, CT might tackle aspects that are of greater relevance to the etiology of SAD. Experimental studies have provided evidence to suggest that increased self-focused attention,[43] recurrent images,[44] memory biases,[45] and safety behaviors[46] contribute to the maintenance of social-phobic beliefs. Furthermore, cognitive variables have been shown to essentially contribute to the mediation of effects in CBTs of SAD.[47] In addition, CT comprises several techniques that have been shown to be effective in the treatment of SAD, including behavioral experiments, attentional training, modification of safety behaviors,[48] videotape feedback,[49] and imagery modification.[50]

In IPT, the central mechanism of action is proposed to be the resolution of interpersonal problem areas.[51] Interpersonal theories suggest that social-phobic individuals establish negative interpersonal cycles, leading to nonassertive behaviors and social avoidance.[52] Some researchers, however, suggest that these negative interpersonal cycles are the result of dysfunctional cognitive processes (eg, the anticipation of negative responses from others) and self-protective behavioral strategies.[12] Although we did not directly assess interpersonal behaviors in the present study, we used the Inventory of Interpersonal Problems to evaluate self-rated interpersonal functioning. Contrary to expectations, we found no significant differences between the 2 treatments at the posttreatment assessment and significantly larger interpersonal improvements in the CT group at 1-year follow-up. A possible explanation for this result is that interpersonal problems are more likely to be resolved when the underlying dysfunctional cognitions and safety and avoidance behaviors are effectively modified.

In contrast to previous trials,[16,17] we did not observe a "slower action" of IPT than of CBT. Rather, IPT could not compensate for the posttreatment differences at 1-year follow-up. Because SAD, similar to dysthymia, is a chronic disorder characterized by marked avoidance behavior, we assume that IPT may not provide sufficiently structured help (eg, exercises and homework) to overcome avoidance. Interpersonal psychotherapy was originally tailored for acute major depression. In this disorder, and possibly in bulimia nervosa,[16,17] IPT might be more beneficial because acute interpersonal problems are more closely related to their etiology.[53] In other disorders, such as dysthymia and SAD, however, interpersonal problems might be speculated to represent a consequence rather than a causative factor.[5] Future research should investigate whether the efficacy of IPT can be increased by developing more structured interventions focusing on disorder-specific problems in SAD (eg, self-protective behaviors) or incorporate techniques that have been proved effective in CT (eg, videotape feedback).

The present study has several limitations. First, although CT was superior to IPT on the primary outcome measure (CGI-I response rate) and the other assessor ratings of social anxiety (LSAS), the self-reported social-anxiety measure (SPAI) showed only a statistical trend in favor of CT in the intent-to-treat analysis. This difference became significant in a post hoc analysis restricted to patients considered to have received an adequate dose of therapy ($\geq$12 sessions). It is unclear why the self-report measure seems to have been less sensitive to differential treatment effects. However, this pattern of results has also been observed in some trials of pharmacologic treatments for SAD.[54] Second, although we included patients with secondary comorbid conditions, such as mild or moderate secondary depression, the characteristics of this sample (exclusion of severe comorbid Axis I and II disorders, recruitment by advertisements, and university setting) are not fully representative of clinical practice.[55] The generalizability of the trial findings to routine clinical care might, thus, be limited. Third, although therapist-completed ratings indicated no protocol violations, we cannot exclude the possibility that some interventions that should be unique to one treatment were occasionally used in the other treatment without being detected.

In conclusion, the results of this study demonstrate the efficacy of CT and IPT in the treatment of SAD. However, the data also provide evidence of a superiority of CT over IPT, suggesting that CT should be the preferred psychological treatment for SAD. The lack of differences between the 2 treatment sites with respect to the efficacy of CT contradicts a potential effect of allegiance and suggests that successful dissemination is possible.

For IPT, further developments might help to improve efficacy by more specifically addressing empirically supported interpersonal problems and avoidance in SAD.

REFERENCES

1. Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE. Prevalence, severity, and comorbidity of 12-month *DSM-IV* disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry*. 2005;62(6):617-627.

2. Ruscio AM, Brown TA, Chiu WT, Sareen J, Stein MB, Kessler RC. Social fears and social phobia in the USA: results from the National Comorbidity Survey Replication. *Psychol Med*. 2008;38(1):15-28.

3. Keller MB. Social anxiety disorder clinical course and outcome: review of Harvard/Brown Anxiety Research Project (HARP) findings. *J Clin Psychiatry*. 2006;67(12)(suppl 12):14-19.

4. Mathew SJ, Ho S. Etiology and neurobiology of social anxiety disorder. *J Clin Psychiatry*. 2006;67(12)(suppl 12):9-13.

5. Rapee RM, Spence SH. The etiology of social phobia: empirical evidence and an initial model. *Clin Psychol Rev*. 2004;24(7):737-767.

6. Heimberg RG, Liebowitz MR, Hope DA, Schneier FR, Holt CS, Welkowitz LA, Juster HR, Campeas R, Bruch MA, Cloitre M, Fallon B, Klein DF. Cognitive behavioral group therapy vs phenelzine therapy for social phobia: 12-week outcome. *Arch Gen Psychiatry*. 1998;55(12):1133-1141.

7. Davidson JR, Foa EB, Huppert JD, Keefe FJ, Franklin ME, Compton JS, Zhao N, Connor KM, Lynch TR, Gadde KM. Fluoxetine, comprehensive cognitive behavioral therapy, and placebo in generalized social phobia. *Arch Gen Psychiatry*. 2004;61(10):1005-1013.

8. Clark DM, Wells A. A cognitive model of social phobia. In: Heimberg RG, Liebowitz MR, Hope DA, Schneier FR, eds. *Social Phobia: Diagnosis, Assessment and Treatment*. New York, NY: Guilford Press; 1995:69-93.

9. Clark DM, Ehlers A, McManus F, Hackmann A, Fennell M, Campbell H, Flower T, Davenport C, Louis B. Cognitive therapy versus fluoxetine in generalized social phobia: a randomized placebo-controlled trial. *J Consult Clin Psychol*. 2003;71(6):1058-1067.

10. Clark DM, Ehlers A, Hackmann A, McManus F, Fennell M, Grey N, Waddington L, Wild J. Cognitive therapy versus exposure and applied relaxation in social phobia: a randomized controlled trial. *J Consult Clin Psychol*. 2006;74(3):568-578.

11. Stangier U, Heidenreich T, Peitz M, Lauterbach W, Clark DM. Cognitive therapy for social phobia: individual versus group treatment. *Behav Res Ther*. 2003;41(9):991-1007.

12. Mörtberg E, Clark DM, Sundin O, Aberg Wistedt A. Intensive group cognitive treatment and individual cognitive therapy vs. treatment as usual in social phobia: a randomized controlled trial. *Acta Psychiatr Scand*. 2007;115(2):142-154.

13. Alden LE, Taylor CT. Interpersonal processes in social phobia. *Clin Psychol Rev*. 2004;24(7):857-882.

14. Klerman GL, Weissman MM, Rounsaville BA, Chevron ES. *Interpersonal Psychotherapy of Depression*. New York, NY: Basic Books; 1984.

15. Weissman MM, Markowitz JC, Klerman GL. *Comprehensive Guide to Interpersonal Psychotherapy*. New York, NY: Basic Books; 2000.

16. Elkin I, Shea MT, Watkins JT, Imber SD, Sotsky SM, Collins JF, Glass DR, Pilkonis PA, Leber WR, Docherty JP, Fiester SJ, Parloff MB. National Institute of Mental Health Treatment of Depression Collaborative Research Program: general effectiveness of treatments. *Arch Gen Psychiatry*. 1982;46(11):971-982.

17. Agras WS, Walsh T, Fairburn CG, Wilson GT, Kraemer HC. A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Arch Gen Psychiatry*. 2000;57(5):459-466.

18. Lipsitz JD, Markowitz JC, Cherry S, Fyer AJ. Open trial of interpersonal psychotherapy for the treatment of social phobia. *Am J Psychiatry*. 1999;156(11):1814-1816.

19. Lipsitz JD, Gur M, Vermes D, Petkova E, Cheng J, Miller N, Laino J, Liebowitz MR, Fyer AJ. A randomized trial of interpersonal therapy versus supportive therapy for social anxiety disorder. *Depress Anxiety*. 2008;25(6):542-553.

20. Elkin I, Gibbons RD, Shea MT, Sotsky SM, Watkins JT, Pilkonis PA, Hedeker D. Initial severity and differential treatment outcome in the National Institute of Mental Health Treatment of Depression Collaborative Research Program. *J Consult Clin Psychol*. 1995;63(5):841-847.

21. Fairburn CG, Jones R, Peveler RC, Hope RA, O'Connor M. Psychotherapy and bulimia nervosa: longer-term effects of interpersonal psychotherapy, behavior therapy, and cognitive behavior therapy. *Arch Gen Psychiatry*. 1993;50(6):419-428.

22. Borge FM, Hoffart A, Sexton H, Clark DM, Markowitz JC, McManus F. Residential cognitive therapy versus residential interpersonal therapy for social phobia: a randomized clinical trial. *J Anxiety Disord*. 2008;22(6):991-1010.

23. Jacobson NS, Hollon SD. Cognitive-behavior therapy versus pharmacotherapy: now that the jury's returned its verdict, it's time to present the rest of the evidence. *J Consult Clin Psychol*. 1996;64(1):74-80.

24. Luborsky L, Diguer L, Seligman DA, Rosenthal R, Krause ED, Johnson S, Halperin G, Bishop M, Berman JS, Schweizer E. The researcher's own therapeutic allegiances: a "wild card" in comparisons of treatment efficacy. *Clin Psychol Sci Pract*. 1999;6(1):95-106.

25. Connor KM, Davidson JRT, Churchill LE, Sherwood A, Foa E, Weisler RH. Psychometric properties of the Social Phobia Inventory (SPIN): new self-rating scale. *Br J Psychiatry*. 2000;176(4):379-386.

26. First MB, Spitzer RL, Gibbon M, Williams JBW. *Structured Clinical Interview for DSM-IV (Axis I Disorders)*. Arlington, VA: American Psychiatric Publishing Inc; 1997.

27. First MB, Spitzer RL, Gibbon M, Williams JBW. *User's Guide for the Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II)*. Washington, DC: American Psychiatry Press; 1997.

28. Wittchen HU, Zaudig M, Fydrich T. *Strukturiertes Klinisches Interview für DSM-IV: SKID, Achse I und Achse II*. Göttingen: Hogrefe; 1997.

29. Hamilton M. A rating scale for depression. *J Neurol Neurosurg Psychiatry*. 1960;23(1):56-62.

30. Hamilton M. Hamilton Depression Scale. In: Collegium IPS, ed. *Skalen für die Psychiatrie*. 4th ed. Weinheim, Germany: Beltz; 1996.

31. Stangier U, Ehlers A, Clark D. *Soziale Phobie: Fortschritte der Psychotherapie*. Göttingen, Germany: Hogrefe; 2006.

32. Zaider TI, Heimberg RG, Fresco DM, Schneier FR, Liebowitz MR. Evaluation of the Clinical Global Impression Scale among individuals with social anxiety disorder. *Psychol Med*. 2003;33(4):611-622.

33. Liebowitz MR, Heimberg RG, Schneier FR, Hope DA, Davies S, Holt CS, Goetz D, Juster HR, Lin SH, Bruch MA, Marshall RD, Klein DF. Cognitive-behavioral group therapy versus phenelzine in social phobia: long-term outcome. *Depress Anxiety*. 1999;10(3):89-98.

34. Liebowitz MR. Social phobia. *Mod Probl Pharmacopsychiatry*. 1987;22:141-173.

35. Stangier U, Heidenreich T. Liebowitz Social Anxiety Scale. In: Collegium IPS, ed. *Internationale Skalen für Psychiatrie*. 5th ed. Weinheim, Germany: Beltz; 2005.

36. Mennin DS, Fresco DM, Heimberg RG, Schneier FR, Davies SO, Liebowitz MR. Screening for social anxiety disorder in the clinical setting: using the Liebowitz Social Anxiety Scale. *J Anxiety Disord*. 2002;16(6):661-673.

37. Turner SM, Beidel DC, Dancu CV, Stanley MA. An empirically derived inventory to measure social fears and anxiety: the Social Phobia and Anxiety Inventory. *Psychol Assess*. 1989;1(1):35-40.

38. Borkovec TD, Nau SD. Credibility of analogue therapy rationales. *J Behav Ther Exp Psychiatry*. 1972;3(4):257-260.

39. Flückiger C, Regli D, Zwahlen D, Hostettler S, Caspar F. The Bernese Post-Session Report: therapists' and patients' version: an instrument for the assessment of psychotherapy processes [in German]. *Z Klin Psychol Psychother*. 2010;39(2):71-79.

40. Hofmann SG, Smits JAJ. Cognitive-behavioral therapy for adult anxiety disorders: a meta-analysis of randomized placebo-controlled trials. *J Clin Psychiatry*. 2008;69(4):621-632.

41. Acarturk C, Cuijpers P, van Straten A, de Graaf R. Psychological treatment of social anxiety disorder: a meta-analysis. *Psychol Med*. 2009;39(2):241-254.

42. Ipser JC, Kariuki CM, Stein DJ. Pharmacotherapy for social anxiety disorder: a systematic review. *Expert Rev Neurother*. 2008;8(2):235-257.

43. Hofmann SG. Self-focused attention before and after treatment of social phobia. *Behav Res Ther*. 2000;38(7):717-725.

44. Hirsch CR, Clark DM, Mathews A, Williams R. Self-images play a causal role in social phobia. *Behav Res Ther*. 2003;41(8):909-921.

45. Amir N, Foa EB, Coles ME. Implicit memory bias for threat-relevant information in individuals with generalized social phobia. *J Abnorm Psychol*. 2000;109(4):713-720.

46. McManus F, Sacadura C, Clark DM. Why social anxiety persists: an experimental investigation of the role of safety behaviours as a maintaining factor. *J Behav Ther Exp Psychiatry*. 2008;39(2):147-161.

47. Hofmann SG. Cognitive mediation of treatment change in social phobia. *J Consult Clin Psychol*. 2004;72(3):393-399.

48. McManus F, Clark DM, Grey N, Wild J, Hirsch C, Fennell M, Hackmann A, Waddington L, Liness S, Manley J. A demonstration of the efficacy of two of the components of cognitive therapy for social phobia. *J Anxiety Disord*. 2009;23(4):496-503.

49. Harvey AG, Clark DM, Ehlers A, Rapee RM. Social anxiety and self-impression: cognitive preparation enhances the beneficial effects of video feedback following a stressful social task. *Behav Res Ther*. 2000;38(12):1183-1192.

50. Wild J, Hackmann A, Clark DM. When the present visits the past: updating traumatic memories in social phobia. *J Behav Ther Exp Psychiatry*. 2007;38(4):386-401.

51. Markowitz JC, Bleiberg KL, Christos P, Levitan E. Solving interpersonal problems correlates with symptom improvement in interpersonal psychotherapy: preliminary findings. *J Nerv Ment Dis*. 2006;194(1):15-20.

52. Neal JA, Edelmann RJ. The etiology of social phobia: toward a developmental profile. *Clin Psychol Rev*. 2003;23(6):761-786.

53. Parker G, Parker I, Brotchie H, Stuart S. Interpersonal psychotherapy for depression? the need to define its ecological niche. *J Affect Disord*. 2006;95(1-3):1-11.

54. Liebowitz MR, Schneier F, Campeas R, Hollander E, Hatterer J, Fyer A, Gorman J, Papp L, Davies S, Gully R, Klein DR. Phenelzine vs atenolol in social phobia: a placebo-controlled comparison. *Arch Gen Psychiatry*. 1992;49(4):290-300.

55. Lincoln TM, Rief W. How much do sample characteristics affect the effect size? an investigation of studies testing the treatment effects for social phobia. *J Anxiety Disord*. 2004;18(4):515-529.

Corrected on July 4, 2011