

# Cognitive-Behavioral and Pharmacological Treatment for Social Phobia: A Meta-Analysis

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**We conducted a meta-analysis using all available controlled treatment outcome studies of cognitive-behavioral and pharmacological treatments for social phobia ( $N = 24$  studies,  $N = 1079$  subjects). The mean social anxiety effect size for cognitive-behavioral treatments was .74 and for pharmacological treatments was .62. Both were significantly different from zero and the difference between them was not significant. Among cognitive-behavioral treatments, exposure-interventions yielded the largest effect size (ES) whether alone (ES = .89) or combined with cognitive restructuring (ES = .80). Selective serotonin reuptake inhibitors (ES = 1.89) and benzodiazepines (ES = .72) yielded the largest effect sizes for pharmacotherapy. According to cost projections, group cognitive-behavioral treatment offered the most cost-effective intervention.**

**Key words:** meta-analysis, social phobia, treatment outcome. [*Clin Psychol Sci Prac* 4:291-306, 1997]

The clinician or researcher interested in determining the most effective treatment interventions or agents for social phobia is faced with a difficult task. First, a wide variety of studies using cognitive-behavioral or pharmacological interventions for social phobia have reported positive findings, making it difficult to identify treatments that are clearly superior to others. These studies have included pharmacological agents like benzodiazepines (BZDs; Davidson et al., 1993; Munjack, Baltazar, Bohn, Cabe, & Appleton, 1990), monoamine oxidase inhibitors (MAOIs;

Gelernter et al., 1991; Liebowitz et al., 1992), beta blockers (Liebowitz et al., 1988), and selective serotonin reuptake inhibitors (SSRIs; Katzelnick, Kobak, & Greist, 1995; van Vliet, den Boer, & Westerberg, 1994), as well as cognitive-behavioral interventions like Heimberg's cognitive-behavioral group treatment (CBGT; Heimberg, Becker, Goldfinger, & Vermilyea, 1985; Heimberg, Dodge et al., 1990; Hope, Heimberg, & Bruch, 1995), situational exposure alone (Alden, 1989; Butler, Cullington, Munby, Amies, & Gelder, 1984; Mattick, Peters, & Clarke, 1989), and rational emotive therapy (Schelver & Gutsch, 1983).

Second, making meaningful comparisons between these studies is complicated by a number of methodological issues. Studies vary widely in their use of outcome measures, which include clinician-rated instruments, physiological measures of anxiety, behavioral avoidance tests, and self-report measures of anxiety, depression, and cognitive change. Consequently, finding a common metric to compare these "apples and oranges" is an elusive proposition. Studies also differ on several other methodological components including whether they use control groups, the types of control groups employed (e.g., pill placebo, wait list, psychological placebo), placebo response rates, lengths of treatment, differences in sample selection (e.g., percentage of subjects in their sample who meet criteria for comorbid conditions including avoidant personality disorder, APD), and whether treatment is offered individually or in groups.

Third, interpreting findings from some studies may be complicated by problems of statistical power. As noted by Feske and Chambless (1995), several social phobia studies comparing two active treatments have reported statistically null findings that may have been due to a lack of adequate power to detect true differences (e.g., Emmel-

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kamp, Mersch, Vissia, & van der Helm, 1985; Gelernter et al., 1991; Mattick et al., 1989; Scholing & Emmelkamp, 1993). In four of these comparative studies the sample size did not exceed 12 subjects per cell, and in none of the studies did the sample size exceed 21 subjects per treatment condition (Feske & Chambless, 1995).

In the present article, we employ meta-analytic techniques to help address some of these difficulties of interpretation in the social phobia literature. One advantage of meta-analytic techniques is that they anchor treatment outcome results to quantifiable standard units of comparison (effect sizes) that can be used to identify the relative effectiveness of various interventions. Effect sizes can be used to make cross-study comparisons of specific social phobia outcome variables (e.g., fear of negative evaluation, social avoidance). In addition, they can be used to quantify the extent to which methodological differences in studies influence treatment outcome. Effect sizes can be combined across studies to increase the power for detecting differential treatment or methodological effects. Finally, effect sizes offer advantages over literary reviews that compare studies' findings based on levels on statistical significance since two studies with different levels of significance can, in fact, have very similar effect sizes (Glass, McGaw, & Smith, 1981).

In a recently completed meta-analysis, Feske and Chambless (1995) examined whether cognitive-behavioral therapy (CBT) for social phobia was superior to exposure treatment alone. The authors compared 12 studies of CBT to 9 studies of exposure treatment and found no differences between these treatment modalities. Both treatments led to significant pretest to posttest and follow-up improvements on self-report measures of social anxiety, cognitive symptoms, and depressed/anxious mood.

In the present meta-analysis we were interested in expanding the scope of Feske and Chambless' meta-analysis beyond psychological studies to include pharmacological studies; as such, we present the first meta-analytical findings that directly compare these two treatment approaches. Pharmacological agents have been increasingly prescribed for the treatment of social phobia over the last two to three decades with numerous studies demonstrating efficacy (Marshall, 1992; Rosenbaum & Pollock, 1994). In this analysis we were interested in examining a number of questions, including what types of cognitive-behavioral and pharmacological treatments are most efficacious, whether length of treatment affects out-

come, and how methodological factors contribute to treatment outcome (e.g., format of treatment, placebo response rates).

We present results from 24 studies ( $N = 1079$  subjects) providing data on 40 separate treatment interventions. These studies represent investigations of the efficacy of cognitive-behavioral treatments, BZDs, SSRIs, and beta blockers. In addition, we examine attrition rates for each intervention and include analyses of the relative cost of the different interventions. Finally, we discuss the implications of our findings for the acute and long-term treatment effects of this debilitating disorder.

## METHOD

### Selection of Studies

We selected treatment outcome studies for patients with social phobia that employed a control group (no-treatment or wait-list control, drug or psychological placebo). Although numerous studies were excluded from our meta-analysis because they did not employ adequate control groups, we felt that this criterion was important because failure to use an adequate control group prohibits the attribution of change to active treatment elements as opposed to the nonspecific effects of being in a treatment study (e.g., the instillation of hope, or weekly supportive contact). We also excluded open-trial pharmacological studies because they tend only to report outcome on subjects who complete the trial, thereby artificially inflating effectiveness rates.

Cognitive-behavioral interventions were defined as studies that utilized cognitive-restructuring, situational exposure, social skills training, systematic desensitization, flooding, and anxiety management techniques. In addition, because we were interested in clinical samples of individuals with social phobia we required that subjects either met *DSM-III*, *DSM-III-R*, or *DSM-IV* criteria (American Psychiatric Association, 1980, 1987, 1994, respectively) or, for articles published earlier than 1980, would have if these criteria had been applied.

Journal articles were located using five strategies. First, a computer-based CD-ROM *PsychLIT* (American Psychological Association, 1994) search was conducted from the beginning of this database in 1974 to the present. *PsychLIT*'s database consists of citations from 1300 journals from 50 countries. The search was conducted using the following key terms alone and in combination: social phobia, social anxiety, avoidant personality disorder, treatment, treatment outcome, clinical trial, comparative

study, and double-blind. These terms were searched both in free-text form and as major key terms in the database. Second, a search using the same key terms was implemented with the *MEDLINE* (National Library of Medicine, 1996) CD-ROM database from 1966 to the present. *MEDLINE* is a database derived from more than 3000 medical and biomedical journals. Third, the reference sections of articles located from both CD-ROM searches were perused for relevant citations. Fourth, unpublished articles were sought by examining *Dissertation Abstracts* from 1980 to the present. Fifth, articles "in press" were included if we had knowledge of them due to their presentation at national conferences prior to January 1996.

A total of 41 articles were initially identified using these four strategies, and 24 of these met the criteria for inclusion in this meta-analysis. Of the 17 studies that were excluded, 10 were pharmacotherapy studies and 7 were psychosocial therapy studies. Eleven of these articles failed to use an adequate control comparison (e.g., an open trial of a drug), four used mixed samples of social phobics and patients with other anxiety disorders (e.g., agoraphobia or panic disorder), one reported data identical to a later study published by the same authors, and one was a case study report. A total of 24 studies with 40 separate treatment by control comparisons met the criteria for inclusion in this meta-analysis.

#### Meta-Analysis Procedure

We were initially struck by the heterogeneity of dependent measures used to assess treatment outcome in studies of social phobia. Although some studies used behavioral avoidance tests as their primary outcome measure, others used self-report questionnaires, blind and unblinded clinician-rated measures of change, and physiological measures (e.g., heart rate or galvanic skin response). In their meta-analysis of panic disorder, Clum, Clum, and Surls (1993) found that the type of dependent measure used in a study significantly biased the relative strength of the study's effect size (ES). For example, measures of behavioral assessment (ES = .06) in this meta-analysis were significantly smaller than nonblind clinician ratings of improvement (ES = .89), whereas self-report questionnaires provided the most nonbiased estimate (ES = .57). In order to reduce this potential source of measurement bias in our own meta-analysis, we decided to derive effect sizes from validated self-report questionnaires. Self-report questionnaires were the most frequently employed assessment tool and were used in all of our 24 studies.

Three effect sizes were calculated for each social phobia study: a measure of social anxiety or avoidance, a cognitive measure, and a measure of depression. These three symptom dimensions characteristic of social phobia were adopted based on methodology used by Feske and Chambless (1995), who also derived their effect sizes from self-report questionnaires. Measures of social anxiety or avoidance included the Social Avoidance and Distress scale (Watson & Friend, 1969), Social Phobia subscale of the Fear Questionnaire (Marks & Mathews, 1979), Social Situations Questionnaire (Bryant & Trower, 1974), Liebowitz Social Phobia scale (Liebowitz, 1987), Fear of Negative Evaluation scale (FNES; Watson & Friend, 1969), the Social Phobia and Anxiety Inventory (Turner, Beidel, Dancu, & Stanley, 1989), and the Personal Report of Confidence as a Speaker (Paul, 1966). Although the FNES is frequently used as a measure of cognitions, we concur with Heimberg's (1994) finding that the FNES confounds the assessment of anxiety and cognition, and that it may be a better measure of social anxiety. Cognitive measures commonly included the Social Cognition Inventory (van Kamp & Klip, 1981), Social Anxiety Self-Statement Questionnaire (Mersch et al., 1993), Irrational Beliefs Test (Jones, 1969), the Social Interaction Self-Statement Test (Glass, Merluzzi, Biever, & Larsen, 1982), and the Cognitions During the Talk scale (Hoffman & Newman, 1992). Measures of depression consisted of the Beck Depression Inventory (Beck, Steer, & Garbin, 1988) and the Depression subscale of the Symptom Checklist (Derogatis, 1975).

For studies that employed multiple measures of the same construct (e.g., four measures of social anxiety), mean effect sizes were calculated by averaging across all of the dependent measures (Rosenthal, 1991). This was done to avoid spurious inflation of effect sizes from studies employing a number of dependent measures. Effect sizes were derived using Smith and Glass's delta procedures (Glass et al., 1981). Effect sizes were calculated by subtracting the mean of the posttreatment control group from the posttreatment experimental group(s), and then dividing by the standard deviation of the control group at posttreatment:

$$ES = \frac{M_t - M_c}{SD_c}$$

A number of different methods were used if the means and standard deviations of a comparison group were not reported. If the *t* statistic was reported, ES was calculated

as  $t\sqrt{(1/N_i + 1/N_j)}$ . If the results were reported by  $F$  score,  $t$  was calculated as  $\sqrt{F}$ ; then the above formula for  $t$  was applied. If only the significance level was reported, procedures based on Glass et al. (1981, pp. 128–129) were utilized to determine  $t$ , and then  $t$  was entered into the above formula. If proportions of the number of individuals “improved” or “not improved” were reported in the study, effect sizes were computed from a table reported by Glass et al. (1981, p. 139). These proportions were rounded to the nearest 5% for purposes of comparison.

Dependent measures mentioned in the Method section but not presented in the Results section of a study were assumed to be nonsignificant. Nonsignificant results were assumed to be  $p = .50$  unless otherwise specified. Both of these decisions were conservative and were likely to reduce the overall effect size; however, this approach was chosen so that greater confidence could be placed in the results. Effect sizes were computed for each dependent variable at posttreatment and, when available, at follow-up. In studies for which there was more than one control group employed (e.g., wait-list control and attention placebo) we derived effect sizes from the group that best controlled for nonspecific treatment effects. For studies using a crossover design (e.g., Katzelnick et al., 1995), effect sizes were calculated at the point of first crossover. The effect sizes of all constructs for CBT and pharmacotherapy studies were tested for heterogeneity using the Rosenthal (1991)  $\chi^2$  test for heterogeneity. We tested each set of studies (CBT and pharmacotherapy, separately) for heterogeneity of effect sizes for each symptom dimension (anxiety severity, depression). In addition, we performed tests of heterogeneity for all other mean effect sizes reported in the results section.

## RESULTS

Effect sizes for measures of social anxiety, cognitive change, and depression in each study are presented in Table 1, along with type of control group employed, sample size, and dropout rates for each experimental and control group. Studies were classified for purposes of comparison as either cognitive-behavioral treatment or pharmacological treatment. Effect sizes in Table 1 are for short-term (i.e.,  $\leq 14$  weeks) treatment outcome.

### Sample Characteristics

We were initially interested in examining whether several variables related to sample characteristics (e.g., sex, duration of disorder, comorbidity) had a systematic effect on

the magnitude of the effect size. We used the social anxiety effect size in these calculations and in subsequent effect size statistical analyses because it was the most common target of treatment interventions, and because it was assessed in every study.

Nearly all studies (21 of 24) assessed the male:female ratio in their samples. Findings suggested a nearly equal distribution: 54.3% of subjects were men and 45.7% were women. Simple regression analyses revealed no significant relationship between sex distribution across studies and the effectiveness of the active treatment ( $R = .29$ ;  $df = 1$ ,  $19$ ;  $p = .20$ ). This conclusion is tentative, however, given that study results were not broken down by sex, precluding more definitive evaluation.

Duration of social phobia was assessed in only 7 of 24 studies; the mean duration of disorder was 15.92 years ( $SD = 3.48$ ; range = 12.4–22.0). The small number of studies reporting this variable precluded further statistical analyses.

Studies varied widely with regard to the degree to which they assessed comorbid Axis I criteria. Although some studies used any comorbid anxiety disorder as an exclusionary criteria (e.g., Liebowitz et al., 1988; Munjack et al., 1990; van Vliet, den Boer, & Westenberg, 1992; van Vliet et al., 1994), others (7 studies) included subjects with these diagnoses, while still others (5 studies) did not report or comment on these diagnoses. Both Heimberg, Dodge et al. (1990) and Hope et al. (1995) excluded subjects with comorbid panic disorder and generalized anxiety disorder (GAD) only if these conditions were equal or greater in severity to patient's difficulties with social phobia. Turner, Beidel, and Jacob (1994) excluded subjects with any secondary Axis I diagnosis with the exception of GAD, simple phobia, or dysthymia. Eight studies specified that current major depressive disorder was an exclusionary criteria. Thirteen studies specified that current substance abuse or psychotic disorders were exclusionary criteria. The actual rate of comorbid panic disorder across studies was 11%, and the rate for comorbid GAD was 15%. However, the small number of studies ( $n = 5$ ) that reported these data prevented us from assessing the influence of these rates on treatment outcome effect size. Only three studies discriminated subjects with generalized social phobia from those with discrete social phobia.

Assessment of Axis II pathology was also varied. The presence of comorbid APD was assessed in only 8 of 24 studies, and in these the mean percentage of affected sub-

**Table 1.** Mean effect sizes for measures of social anxiety, cognitive change, and depression in pharmacological and cognitive-behavioral interventions at posttreatment

| Study Treatment Group(s)<br>(Control Group)              | N  | Dropout<br>Rate | Tx Length | Social<br>Anxiety<br>ES | Cognitive<br>Change ES | Depression<br>ES |
|--|----|-----------------|-----------|-------------------------|------------------------|------------------|
| <b>Cognitive-Behavioral Treatments</b>                   |    |                 |           |                         |                        |                  |
| Marzillier et al. (1976)                                 | 21 |                 | 14 weeks  |                         |                        |                  |
| Social skills training                                   |    | 11%             |           | 0.86                    | —                      | —                |
| Systematic desensitization<br>(Wait-list control)        |    | 50%<br>0%       |           | 0.50                    |                        |                  |
| Kanter & Goldfried (1979)                                | 68 |                 | 7 weeks   |                         |                        |                  |
| Rational restructuring                                   |    | 12%             |           | 1.75                    | 2.35                   | —                |
| Self-control desensitization (SCD)                       |    | 19%             |           | 0.87                    | 0.82                   | —                |
| Rational Restructuring + SCD<br>(Wait-list control)      |    | 15%<br>0%       |           | 1.30                    | 1.97                   | —                |
| Frisch et al. (1982)                                     | 34 |                 | 8 weeks   |                         |                        |                  |
| Social skills training (SST)                             |    | 0%              |           | 0.24                    | 0.07                   | —                |
| SST + stress management training<br>(Minimal Tx control) |    | 3%<br>0%        |           | 0.18                    | 0.12                   | —                |
| Schelver & Gutsch (1983)                                 | 45 |                 | 5 weeks   |                         |                        |                  |
| Rational emotive therapy<br>(Attention placebo control)  |    | 8%<br>7%        |           | 0.49                    | —                      | —                |
| Butler et al. (1984)                                     | 45 |                 | 7 weeks   |                         |                        |                  |
| Exposure + anxiety management                            |    | 0%              |           | 0.90                    | —                      | 0.87             |
| Exposure + attention placebo<br>(Wait-list control)      |    | 0%<br>0%        |           | 0.74                    | —                      | 0.78             |
| Jerremalm et al. (1986)                                  | 38 |                 | 12 weeks  |                         |                        |                  |
| Applied relaxation                                       |    | 20%             |           | 0.32                    | —                      | 0.42             |
| Self-instructional training<br>(Wait-list control)       |    | 20%<br>0%       |           | -0.13                   | —                      | 0.68             |
| Alden (1989)   | 76 |                 | 10 weeks  |                         |                        |                  |
| Exposure (Exp)   |    | 1%              |           | 1.40                    | —                      | —                |
| Social skills training (SST)                             |    | 0%              |           | 0.71                    | —                      | —                |
| Intimacy focus (Exp + SST)<br>(No treatment control)     |    | 4%<br>—         |           | 1.06                    | —                      | —                |
| Mattick et al. (1989)                                    | 43 |                 | 6 weeks   |                         |                        |                  |
| Cognitive restructuring                                  |    | 18%             |           | 0.29                    | 0.39                   | —                |
| Exposure   |    | 9%              |           | 0.44                    | 0.16                   | —                |
| Combination<br>(Wait-list control)                       |    | 9%<br>10%       |           | 0.61                    | 0.25                   | —                |
| Heimberg et al. (1990)                                   | 49 |                 | 12 weeks  |                         |                        |                  |
| CBGT   |    | 20%             |           | 0.20                    | 0.20                   | 0.36             |
| (Education + group support)                              |    | 17%             |           |                         |                        |                  |
| Heimberg et al. (1993)                                   | 49 |                 | 12 weeks  |                         |                        |                  |
| CBGT   |    | ND              |           | 0.78                    | 0.82                   | 1.08             |
| (Education + group support)                              |    | ND              |           |                         |                        |                  |
| Newman et al. (1994)                                     | 36 |                 | 8 weeks   |                         |                        |                  |
| Exposure   |    | 6%              |           | 0.66                    | 0.87                   | —                |
| (Wait-list control)                                      |    | 3%              |           |                         |                        |                  |
| Mersch (1995)  | 34 |                 | 14 weeks  |                         |                        |                  |
| Exposure   |    | 6%              |           | 0.79                    | 0.66                   | —                |
| Exposure + RET + SST<br>(Wait-list control)              |    | 6%<br>6%        |           | 0.36                    | 0.41                   | —                |
| Hope et al. (1995)                                       | 43 |                 | 12 weeks  |                         |                        |                  |
| CBGT   |    | 7%              |           | 1.99                    | 0.71                   | —                |
| Exposure<br>(Wait-list control)                          |    | (Overall)       |           | 2.30                    | 0.98                   | —                |
| <b>Cognitive-Behavioral Treatment vs. Pill Placebo</b>   |    |                 |           |                         |                        |                  |
| Turner et al. (1994)                                     | 72 |                 | 12 weeks  |                         |                        |                  |
| Flooding   |    | 19%             |           | 1.77                    | —                      | —                |
| Atenolol   |    | 13%             |           | 0.60                    | —                      | —                |
| (Pill placebo)   |    | 5%              |           |                         |                        |                  |
| <b>Pharmacological Treatment vs. Pill Placebo</b>        |    |                 |           |                         |                        |                  |
| Liebowitz et al. (1988)                                  | 41 |                 | 8 weeks   |                         |                        |                  |
| Atenolol   |    | 18%             |           | -0.22                   | —                      | 0.10             |
| Phenelzine   |    | 21%             |           | 0.68                    | —                      | -0.05            |
| (Pill placebo)   |    | 6%              |           |                         |                        |                  |
| Munjack et al. (1990)                                    | 23 |                 | 8 weeks   |                         |                        |                  |
| Clonazepam   |    | 17%             |           | 1.13                    | —                      | 0.41             |
| (Non-Tx control)   |    | 9%              |           |                         |                        |                  |

Table 1. Continued

| Study Treatment Group(s)<br>(Control Group) | N  | Dropout<br>Rate | Tx Length | Social<br>Anxiety<br>ES | Cognitive<br>Change ES | Depression<br>ES |
|---|----|-----------------|-----------|-------------------------|------------------------|------------------|
| Clark & Agras (1991)                        | 34 |                 | 6 weeks   |                         |                        |                  |
| Buspirone                                   |    | 22%             |           | -0.50                   | -0.62                  | —                |
| Buspirone + CBT                             |    | 0%              |           | 0.50                    | 0.15                   | —                |
| CBT + pill placebo                          |    | 22%             |           | -0.25                   | 1.00                   | —                |
| (Pill placebo)                              |    | 0%              |           |                         |                        |                  |
| Gelernter et al. (1991)                     | 65 |                 | 12 weeks  |                         |                        |                  |
| Alprazolam + exposure instru.               |    | 7%              |           | 0.31                    | —                      | —                |
| Phenelzine + exposure instru.               |    | 13%             |           | 0.65                    | —                      | —                |
| CBT   |    | 15%             |           | 0.13                    | —                      | —                |
| (Pill placebo + instru.)                    |    | 0%              |           |                         |                        |                  |
| Liebowitz et al. (1992)                     | 74 |                 | 8 weeks   |                         |                        |                  |
| Atenolol                                    |    | 35%             |           | -0.13                   | —                      | -0.50            |
| Phenelzine                                  |    | 16%             |           | 0.23                    | —                      | 0.11             |
| (Pill placebo)                              |    | 8%              |           |                         |                        |                  |
| van Vliet et al. (1992)                     | 30 |                 | 12 weeks  |                         |                        |                  |
| Brofaromine                                 |    | 0%              |           | 0.77                    | —                      | 0.52             |
| (Pill placebo)                              |    | 7%              |           |                         |                        |                  |
| Versiani et al. (1992)                      | 78 |                 | 8 weeks   |                         |                        |                  |
| Moclobemide                                 |    | 19%             |           | 0.74                    | —                      | 0.98             |
| Phenelzine                                  |    | 0%              |           | 0.88                    | —                      | 1.30             |
| (Pill placebo)                              |    | 62%             |           |                         |                        |                  |
| Davidson et al. (1993)*                     | 39 |                 | 10 weeks  |                         |                        |                  |
| Clonazepam                                  |    | 26%             |           | 5.95*                   | —                      | 2.00             |
| (Pill placebo)                              |    | 25%             |           |                         |                        |                  |
| van Vliet et al. (1994)                     | 30 |                 | 12 weeks  |                         |                        |                  |
| Fluvoxamine                                 |    | 3%              |           | 2.73                    | —                      | 4.60             |
| (Pill placebo)                              |    | 3%              |           |                         |                        |                  |
| Katzelnick et al. (1995)                    | 12 |                 | 10 weeks  |                         |                        |                  |
| Sertraline                                  |    | 0%              |           | 1.05                    | —                      | —                |
| (Pill placebo)                              |    |                 |           |                         |                        |                  |

Note: ND = not determinable.

\*Study excluded from overall analyses due to  $\chi^2$  test of heterogeneity (Rosenthal, 1991).

jects was 27.3% (SD = 36.1). In some studies APD was an inclusionary criteria (Alden, 1989), whereas in others it was used as an exclusionary criteria (Butler et al., 1984; Heimberg, Dodge et al., 1990; van Vliet et al., 1994). Turner, Beidel, and Jacob (1994) also excluded subjects with borderline, paranoid, schizotypal, schizoid, paranoid, and antisocial personality disorders. Alden (1989) excluded subjects with pathological personality scores above the 50th percentile on the Millon Clinical Multiaxial Inventory. The majority of studies ( $n = 15$ ) did not assess the presence of other personality disorders.

#### Cognitive-Behavioral Interventions

Studies of cognitive-behavior therapy utilized cognitive restructuring, situational exposure, social skills training, systematic desensitization, flooding, and anxiety management techniques. A total of 16 studies with 27 treatment groups employed cognitive-behavioral interventions without pharmacotherapy. Treatment occurred in both group (9 studies, 17 treatment interventions) and individ-

ual treatment formats (7 studies, 10 treatment interventions). The majority of studies (87%) combined in vivo exposure during sessions with homework assignments of self-directed exposure. In two studies, exposure was described and assigned without direct in-session exposure. Summary results of mean effects sizes for CBT and pharmacotherapy are presented in Table 2.

The mean social anxiety effect size across all CBT studies was .74 (95% CI = .54-.94), mean cognitive effect size was .74, and mean depression effect size was .67. Comparison of the social anxiety measure to the null hypothesis (ES = .0) was statistically significant [ $t(26) = 6.23; p < .0001$ ]. Comparisons of the cognitive change [ $t(15) = 4.58; p < .001$ ] and depression measures [ $t(5) = 6.25; p < .001$ ] to the null hypothesis were also statistically significant. Studies using exposure techniques alone (ES = .89) and combination exposure plus cognitive restructuring (ES = .80) yielded the largest effect sizes. Studies employing cognitive restructuring alone (ES = .60) and social skills training (ES = .60) yielded moderate effect

**Table 2.** Summary statistics of comparisons within and between cognitive-behavioral therapy and pharmacotherapy at posttreatment

| Type of Treatment             | N  | Mean Anxiety ES | Dropout Rate |
|-------------------------------|----|-----------------|--------------|
| Cognitive-behavioral therapy  | 27 | 0.74            | 10.7%        |
| Cog. restructuring + exposure | 8  | 0.80            | 10.3%        |
| Exposure alone                | 9  | 0.89            | 12.6%        |
| Cog. restructuring alone      | 4  | 0.60            | 14.5%        |
| Social skills training        | 3  | 0.60            | 3.7%         |
| Other                         | 3  | 0.47            | 7.7%         |
| Pharmacotherapy               | 13 | 0.62            | 13.7%        |
| MAOIs                         | 5  | 0.64            | 13.8%        |
| BZDs                          | 2  | 0.72            | 12.0%        |
| SSRIs                         | 2  | 1.89            | 1.5%         |
| Beta blockers                 | 3  | -0.08           | 22.0%        |
| Buspirone                     | 1  | -0.50           | 22.0%        |

sizes. Dropout rates were generally similar across CBT studies and at a rate of roughly 10%, with social skills training yielding the smallest dropout rates. Nearly all studies used wait-list or placebo attention controls. The one study that used exposure instructions as a control group (Gelernter et al., 1991) yielded the smallest effect size for additive CBT techniques, suggesting the strength of exposure techniques as a treatment component.

Duration of CBT treatment was assessed by multiplying the number of hours of CBT treatment per week by the number of weeks of treatment. For example, subjects receiving 2 h of CBT per week for 12 weeks would receive a total of 24 sessions. The mean duration of CBT treatment was 15.0 sessions with a range of 5–30 sessions. Simple regression analyses revealed no significant relationship between duration of CBT treatment and change in social anxiety ( $R = .03$ ;  $df = 1, 25$ ;  $p = .37$ ).

We next examined the question of whether treatment format was systematically related to treatment outcome. Seventeen interventions used a group treatment format and 10 used an individual format. We found no significant differences [ $t(25) = -1.86$ ;  $p = .07$ ] between group treatment ( $ES = .88$ ) over individual treatment ( $ES = .44$ ).

#### Pharmacotherapy Interventions

Ten studies that included 13 treatment interventions provide data on the efficacy of pharmacotherapy relative to pill placebo ( $n = 8$  studies), pill placebo plus exposure instructions ( $n = 1$  study), or a no-treatment control group ( $n = 1$  study). We eliminated one study from the overall analysis (Davidson & Versiani, 1994;  $ES = 6.95$ ) because its absence allowed the remaining studies to satisfy

the  $\chi^2$  test for heterogeneity of effect sizes (see Rosenthal, 1991).

The mean effect size for social anxiety was .62 (95% CI = .42–.82), and for depression was .83. Measures of cognitive change were assessed in only one study (Clark & Agras, 1991), and these findings suggested that individuals in the drug group experienced more negative cognitions at posttreatment relative to pill placebo. Comparison of the social anxiety effect size to the null hypothesis ( $ES = .0$ ) was significant [ $t(11) = 2.51$ ;  $p < .03$ ]. Comparisons of the depression measure to the null hypothesis were not statistically significant [ $t(8) = 1.65$ ;  $p = .14$ ]. The mean dropout rate for these studies was 13.7%; control groups in these studies dropped out at a rate of 11.1%. Eliminating the two studies that did not use pure pill placebo control groups did little to change the overall effect sizes ( $ES = .52$  for social anxiety;  $ES = .88$  for depression). Including the Davidson et al. (1993) study in our analyses raised the mean effect size for pharmacotherapy considerably, to 1.03.

Monoamine oxidase inhibitors (MAOIs) in this meta-analysis included phenelzine ( $n = 4$  studies) and moclobemide [a reversible inhibitor of monoamine oxidase (RIMA);  $n = 1$  study], and their mean effect size was .64 with a dropout rate of 13.8%. Two controlled studies assessed the efficacy of high-potency BZDs (alprazolam and clonazepam); their mean effect size was .72, and their dropout rate was 12.0%. More recent and promising findings have been demonstrated with SSRIs including fluvoxamine (van Vliet, den Boer, & Westerberg, 1994;  $ES = 2.73$ ; dropout rate = 3%) and sertraline (Katzelnick et al., 1995;  $ES = 1.05$ ; dropout rate = 0%). The beta blocker atenolol was found to yield a negative effect size ( $ES = -.08$ ) with two studies finding that this drug was less effective than placebo. Similarly, buspirone did not appear to be effective for social phobia because it did worse than a pill placebo condition.

#### Combined Pharmacological and Cognitive-Behavioral Treatments

Only two studies attempted to combine medication and CBT components and the overall social anxiety effect size was ( $ES = .49$ ). Clark and Agras (1991) utilized buspirone, a drug not generally found effective for social phobia, and found that CBT plus buspirone did better than CBT plus a pill placebo. The Gelernter et al. (1991) study combined the MAOI phenelzine with exposure instructions

and registered an effect size of .65; alprazolam plus exposure yielded an effect size of .31 in this study. Determination of the relative efficacy of combined pharmacotherapy and CBT approaches warrants additional systematic study.

#### **Pharmacotherapy Versus Cognitive-Behavioral Treatment Versus Combined Treatment**

Comparisons of studies utilizing cognitive-behavioral treatments ( $ES = .74$ ) to those using pharmacotherapy ( $ES = .62$ ) and combined treatments ( $ES = .49$ ) were not statistically significant [ $F(2, 41) = .31; p = .74$ ]. In addition, mean attrition rates were not significantly different among subjects receiving a cognitive-behavioral intervention (10.7%) and those receiving a medication intervention (13.7%), or combined treatment [6.7%;  $F(2, 41) = .63; p = .54$ ]. Including the Davidson et al. (1993) study, comparisons of CBT to pharmacotherapy remained non-significant [ $F(1, 42) = .49; p = .62$ ]. It should be noted that these comparisons need to be interpreted in light of the relatively low available statistical power.

Any valid comparison of the effect sizes of pharmacotherapy and cognitive-behavioral interventions requires some discussion of their respective use of control groups. The strength of a control group and the increment of its effect size have an inverse relationship. Interventions that employ strong control groups are likely to yield relatively smaller effect sizes. However, direct effect size comparisons on the basis of type of control in this meta-analysis would be biased by their systematically differential use across types of treatment (e.g., nearly all studies using pill placebo controls are pharmacotherapy studies). Results from a previous meta-analytic review of panic disorder (Gould, Otto, & Pollack, 1995) indicated that pill placebo control groups are relatively stronger (35% panic-free rates among completers) than wait-list controls (28% panic-free rates), although this difference may have been influenced by the greater attrition rate in pill placebo groups. Findings from a meta-analytic study of depression (Robinson, Berman, & Neimayer, 1990) also suggested that studies employing wait-list controls were more likely to yield relatively larger effect sizes. In the present meta-analysis, pharmacotherapy interventions were far more likely to employ a pill placebo control (91%), and did not use wait-list controls (0%). In contrast, cognitive-behavioral interventions were far less likely to use a pill placebo condition (20%), and more likely to use a wait-list control (47%). Consequently, it appears that CBT

interventions may enjoy an advantage in terms of these direct comparisons.

Do CBT and pharmacotherapy studies differ in terms of sample severity? This question remains open to debate given that sample severity was largely not addressed across studies. In addition, there was no consistent metric for assessing sample severity across studies in this meta-analysis.

#### **Meta-Analysis of Long-Term Treatment Outcome**

Examination of the long-term efficacy of social phobia interventions is restricted by the difficulties in maintaining patients in control conditions over time. In most studies, subjects in control conditions are given a therapeutic treatment intervention at posttreatment, thereby making it impossible to derive a "controlled" effect size at follow-up. In fact, in only two studies (Heimberg, Dodge et al., 1990; Heimberg, Salzman, Holt, & Blendell, 1993) were long-term controlled effect sizes able to be determined. To address this problem, we calculated effect sizes using a within-groups procedure similar to that employed by Chambless and Gillis (1993), and to the one we employed in our meta-analysis of panic disorder (Gould, Otto, & Pollack, 1995). We felt the benefit of including more studies in the long-term outcome analyses outweighed the disadvantage of calculating effect sizes that were not derived from control conditions. Effect sizes were calculated by subtracting the mean of the treatment group at follow-up from the mean of the treatment group at posttreatment, and then dividing by the standard deviation of the treatment group at posttreatment:

$$ES = \frac{M_i(\text{follow-up}) - M_i(\text{posttreatment})}{SD_i(\text{posttreatment})}$$

Using this formula we were able to estimate the within group change from posttreatment to follow-up. Effect sizes with negative values indicate that subjects did not maintain their treatment gains after treatment; positively valued effect sizes suggest that subjects continued to improve after treatment. Follow-up effect sizes were drawn from the same outcome measures as posttreatment effect sizes. We used a minimum follow-up period of 3 months; although a longer follow-up period would have been preferable, only two studies included follow-ups of as much as 6 months. One study (Mersch, 1995) employed a 1.5 year follow-up, but these data excluded treatment nonresponders; we chose to use their shorter (3 month) follow-up measures that did include these nonre-

**Table 3.** Mean effect sizes for measures of social anxiety in pharmacological and cognitive-behavioral interventions at posttreatment and follow-up

| Study Treatment Group(s)<br>(Control Group)          | N  | Dropout<br>Rate | Post-Tx Social<br>Anxiety ES | Follow-up<br>Length | Change* from<br>Post-Tx to FU<br>Social Anxiety ES |
|--|----|-----------------|------------------------------|---------------------|--|
| <b>Cognitive-Behavioral Treatments</b>               |    |                 |                              |                     |  |
| Kanter & Goldfried (1979)                            | 68 |                 |                              |                     |  |
| Rational restructuring                               |    | 12%             | 1.75                         | 3 months            | .22  |
| Self-control desensitization (SCD)                   |    | 19%             | 0.87                         |                     | .35  |
| Rational restructuring + SCD<br>(Wait-list control)  |    | 15%<br>0%       | 1.30                         |                     | .22  |
| Alden (1989)   | 76 |                 |                              |                     |  |
| Exposure (Exp)                                       |    | 1%              | 1.40                         | 3 months            | -.10   |
| Social Skills Training (SST)                         |    | 0%              | 0.71                         |                     | 0  |
| Intimacy focus (Exp + SST)<br>(No treatment control) |    | 4%<br>—         | 1.06                         |                     | .50  |
| Mattick et al. (1989)                                | 43 |                 |                              |                     |  |
| Cognitive restructuring                              |    | 18%             | 0.29                         | 3 months            | .62  |
| Exposure   |    | 9%              | 0.44                         |                     | .61  |
| Combination<br>(Wait-list control)                   |    | 9%<br>10%       | 0.61                         |                     | -.08   |
| Heimberg et al. (1990)                               | 49 |                 |                              |                     |  |
| CBGT<br>(Education + group support)                  |    | 20%<br>17%      | 0.20                         | 3 months            | .11  |
| Heimberg et al. (1993)                               | 49 |                 |                              |                     |  |
| CBGT<br>(Education + group support)                  |    | ND<br>ND        | 0.78                         | 6 months            | .48  |
| Mersch (1995)  | 34 |                 |                              |                     |  |
| Exposure   |    | 6%              | 0.79                         | 3 months            | .30  |
| Exposure + RET + SST<br>(Wait-list control)          |    | 6%              | 0.36                         |                     | .28  |
| Hope et al. (1995)                                   | 43 |                 |                              |                     |  |
| CBGT   |    | 7%              | 1.99                         | 6 months            | .08  |
| Exposure<br>(Wait-list control)                      |    |                 | 2.30                         |                     | -.18   |
| <b>Pharmacological Treatment vs. Pill Placebo</b>    |    |                 |                              |                     |  |
| Versiani et al. (1992)                               | 78 |                 |                              |                     |  |
| Moclobemide  |    | 19%             | 0.74                         | 3 months            | .11  |
| Phenelzine<br>(Pill placebo)                         |    | 0%              | 0.88                         |                     | .03  |

\*Negative values indicate that social anxiety measures increased relative to values at posttreatment. FU = follow-up.

sponders. Within-group effect sizes are presented in Table 3.

Ten studies from our sample did not have follow-up conditions. In three studies, the follow-up measures fell short of our 3 month minimal follow-up inclusion criterion (Frisch, Elliott, Atsides, Salva, & Denney, 1982; Gelernter et al., 1991; Liebowitz et al., 1992), and in three others there was insufficient data to derive an effect size (Marzillier, Lambert, & Kellett, 1976; Turner, Beidel, & Jacob, 1994; van Vliet et al., 1992). We were able to determine long-term effect sizes in eight studies that included 17 interventions. The mean within-group effect size for all studies at follow-up was .21, suggesting that subjects continued to make modest improvement in their social anxiety after treatment had ended. Notably, almost all (seven) of these studies employed CBT interventions

(mean ES = .23), and one study examining pharmacotherapy had an effect size that indicated no further treatment gains over the follow-up period (ES = .07). Subjects were medication free at follow-up in the pharmacotherapy study.

#### The Cost of Treatment

We assessed the relative financial costs of cognitive-behavioral and pharmacological treatment for social phobia based on monthly costs for a typical course of these treatments. We estimated individual CBT sessions at a cost of \$90.00 per session, and group CBT sessions at a cost of \$40.00 per session. Twenty- to thirty-minute pharmacological management and CBT booster sessions were computed at a rate of \$60.00 per session. The cost of the initial evaluation session was not included in these calcula-

tions and was assumed to be equivalent across treatment domains. Medication dosages were based on therapeutic levels of each agent used in studies in this meta-analysis (Davidson et al., 1993; Liebowitz et al., 1985; van Vliet et al., 1994). Medication costs were computed based on average prescription costs per 30 days of each medication in the Boston area to reflect the typical consumer purchasing practices: phenelzine (15 mg tablets, qid), fluvoxamine (50 mg tablets, tid), and clonazepam (1 mg tablets, bid; 2 mg tablets, qd). When possible, estimates were based on larger pill sizes to ensure that medication costs were minimized; generic versions of these drugs are not yet available.

The frequency of CBT sessions was based on our own group program for social phobia with weekly sessions for the first 10 sessions, sessions every other week for the last two sessions, and three booster sessions during the first year. We also included costs of four booster sessions during the second year (on a separate line of Table 3), although many social phobia programs do not furnish booster sessions 1 year posttreatment. Our current social phobia group program is modeled after the CBGT program developed by Heimberg and his colleagues (Heimberg et al., 1985; Heimberg & Barlow, 1991) and includes (1) development of a cognitive-behavioral explanation of

social phobia, (2) cognitive restructuring of maladaptive and anxiogenic thinking, (3) development of a social phobia anxiety hierarchy, (4) use of within-group exposure simulations, and (5) in vivo exposure homework assignments.

Pharmacological treatment sessions were scheduled for twice during the first month, monthly for the first 3 months thereafter, and then progressing from every other month to every third month by the end of the first year. Given that most patients with social phobia remain on medication for at least 2 years, we assumed ongoing medication treatment during the 2 years of our estimates. Dosages of medication were selected to reflect common dosages in drug trials. Our findings are summarized in Table 4.

Results indicated that the least costly intervention was clearly CBGT, with a yearly total of approximately \$600. The least costly pharmacological intervention was 2.0 mg tablets of clonazepam with a yearly cost of approximately \$1000. Individual CBT along with phenelzine and clonazepam (1.0 mg) were of comparable cost; however, their yearly costs totaled approximately twice the charge of CBGT (\$1200). Fluvoxamine was clearly the most costly intervention. At the end of the second year, CBGT was by far the least costly intervention. Given CBGT's roughly

**Table 4.** Cost of different modalities of treatment over a 2-year period

| Treatment Modality                                 | Months of Treatment |            |            |             |             |             |             |
|--|---------------------|------------|------------|-------------|-------------|-------------|-------------|
|  | 1                   | 2          | 3          | 6           | 9           | 12          | 24          |
| <i>Cognitive-Behavior Therapy (CBT)</i>            |                     |            |            |             |             |             |             |
| CBT or CBGT sessions per month                     | 4                   | 4          | 2          | 1           | 1           |             |             |
| Cumulative number of sessions                      | 4                   | 8          | 10         | 14          | 15          | 15          | 15(19)      |
| Cumulative CBT (ind.) session cost (\$)            | 360                 | 720        | 900        | 1260        | 1350        | 1350        | 1350        |
| (With 4 booster sessions during year 2)            |                     |            |            |             |             |             | (1710)      |
| Cum. CBGT (Group) Session Cost (\$)                | 160                 | 320        | 400        | 560         | 600         | 600         | 600         |
| (With 4 booster sessions during year 2)            |                     |            |            |             |             |             | (760)       |
| <i>Pharmacotherapy</i>                             |                     |            |            |             |             |             |             |
| Sessions per month                                 | 2                   | 1          | 1          | 1           | 0           |             |             |
| Cumulative number of sessions                      | 2                   | 3          | 4          | 6           | 7           | 8           | 12          |
| Cumulative session cost (\$)                       | 120                 | 180        | 240        | 360         | 420         | 480         | 720         |
| <i>Medication costs (\$)</i>                       |                     |            |            |             |             |             |             |
| 60 mg phenelzine                                   | 57                  | 114        | 171        | 342         | 513         | 684         | 1368        |
| 150 mg fluvoxamine                                 | 199                 | 398        | 597        | 1194        | 1791        | 2388        | 4776        |
| 2.0 mg clonazepam (1.0 mg tabs)                    | 63                  | 126        | 189        | 378         | 567         | 756         | 1512        |
| 2.0 mg clonazepam (2.0 mg tabs)                    | 43                  | 86         | 129        | 258         | 387         | 516         | 1024        |
| <i>Total cumulative pharmacotherapy costs (\$)</i> |                     |            |            |             |             |             |             |
| 60 mg phenelzine                                   | <b>177</b>          | 294        | <b>411</b> | <b>702</b>  | <b>933</b>  | <b>1164</b> | <b>2088</b> |
| 150 mg fluvoxamine                                 | <b>319</b>          | <b>578</b> | <b>837</b> | <b>1554</b> | <b>2211</b> | <b>2868</b> | <b>5496</b> |
| 2.0 mg clonazepam (1.0 mg tabs)                    | <b>183</b>          | 306        | <b>429</b> | <b>738</b>  | <b>987</b>  | <b>1236</b> | <b>2232</b> |
| 2.0 mg clonazepam (2.0 mg tabs)                    | <b>163</b>          | 266        | 369        | <b>618</b>  | <b>807</b>  | <b>996</b>  | <b>1744</b> |

Note: Boldface numbers signify when costs exceed CBGT, and boldface italics signify when costs exceed individual CBT.

equal efficacy to pharmacotherapy, our findings suggest that CBGT is the most cost-effective treatment intervention currently available.

Other factors beyond monetary costs should also be considered when weighing the advantages and disadvantages of each treatment modality. In general, CBT requires more total therapy visits, greater transportation costs, more time spent doing CBT homework assignments, and potentially more interference with daily life activities. Alternatively, medications may cause difficulties with side effects and problems related to withdrawal.

## DISCUSSION

This review provides effect size analyses for studies of cognitive-behavioral and pharmacologic treatments of social phobia published during the last 20 years. Findings from the meta-analysis can be summarized into several major points. First, both pharmacological and cognitive-behavioral treatment interventions for social phobia are superior to control conditions and appear to be effective interventions for social phobia in the short term. In addition, cognitive-behavioral treatments appear to maintain or even increase their salutary effects during a 3–6 month follow-up period. Only one study provided longer term follow-up on pharmacotherapy and indicated no slippage of treatment effects. Cognitive-behavioral interventions render large effect sizes for three measures of outcome that include indices of social anxiety ( $ES = .74$ ), cognitive change ( $ES = .76$ ) and depression ( $ES = .67$ ). Greater confidence can be placed in measures of social anxiety relative to the other two symptom dimensions because of the larger number of studies assessing this measure. Pharmacological interventions also yield large effect sizes for measures of social anxiety ( $ES = .62$ ) and depression ( $ES = .83$ ), although their effects on cognitive change were unclear given the infrequent assessment of this variable. Our findings are consistent with previous reviews and analyses supporting the effectiveness of pharmacological (Pollack & Gould, 1996; Rosenbaum & Pollock, 1994) and cognitive-behavioral (Chambless & Gillis, 1993; Feske & Chambless, 1995; Gould & Otto, 1995; Heimberg, 1993) treatments.

Among cognitive-behavioral interventions, studies that employ exposure techniques ( $ES = .89$ ) appear to yield somewhat more favorable outcomes than studies using cognitive restructuring alone ( $ES = .60$ ). If cognitive change is a necessary condition for improvement in

social phobia, then perhaps this change can occur without the use of cognitive restructuring techniques. For example, Newman, Hoffmann, Werner, Roth, and Taylor (1994) found that cognitive restructuring can occur with the use of exposure techniques alone. Feske and Chambless (1995), in their meta-analysis, also found that CBT and exposure were equivalent on measures of change in cognitive symptoms. Studies that employed group treatments ( $ES = .88$ ) appeared to be more effective than those employing individual treatment ( $ES = .44$ ), although only at the level of a trend. This finding supports the importance of exposure in CBT for social phobia; group treatments offer a “built-in” exposure format that confer an advantage over individual treatments.

Among pharmacological interventions, our meta-analysis suggests the therapeutic efficacy of a number of agents. Selective serotonin reuptake inhibitors (SSRIs) yielded strong outcomes and had low dropout rates. These agents are generally well tolerated in clinical practice; however, the limited number of controlled trials to date requires further study of these agents to assess their spectrum of efficacy and their relative benefits compared to MAOIs and BZDs. Monoamine oxidase inhibitors, both the irreversible agents (e.g., phenelzine) and the RIMAs, have large effect sizes, although the dietary restrictions required for safe use of these agents restrict their acceptability to patients. High-potency BZDs also appear to be effective agents, although concerns regarding their use in patients with alcohol abuse may limit their application. Beta blockers appear effective for some patients with performance anxiety but are not typically useful for the generalized symptoms of social phobia.

Medication treatments may be particularly appropriate for those persons refusing CBT because CBT entails exposure to highly anxiety-provoking situations. However, medications also carry potential side effect and withdrawal problems. Studies testing the efficacy of BZDs for the treatment of panic disorder have shown that relapse is common even after successful withdrawal (Fyer et al., 1987; Marks et al., 1993). This increased risk of relapse may also be true for patients receiving BZDs for social phobia. Women in their child-bearing years may also be less than ideal candidates for medications given the unknown teratogenic effects of these agents.

Meta-analytic comparisons of short-term outcome in studies using cognitive-behavioral interventions ( $ES = .74$ ) relative to those using pharmacological interventions

(ES = .62) were not statistically significant, suggesting that both treatments are approximately equally effective in the acute treatment of social phobia. Attrition rates for the two interventions were also not different. These findings should be interpreted in light of the low statistical power for these comparisons relative to meta-analyses of other disorders reported in the literature. In addition, CBT may be favored in these direct comparisons because of its use of a relatively weaker control comparison group. In general, there is a dearth of well-controlled studies that allow us to make comparisons between specific types of CBT interventions, and between these types and medication approaches. Our cost analysis suggests that CBGT is the most cost-effective intervention for social phobia given the assumptions of our model; however, we caution that the validity of our model needs to be tested under actual conditions of clinical practice.

The influence on effect size of duration of disorder, comorbid anxiety disorders, comorbid personality disorders, and generalized versus discrete subtype was not determinable given the low rate and variable manner with which these sample characteristics were reported. We also found no relationship between sex and treatment outcome, although definitive conclusions about this relationship could be better established if results in these studies were broken down in terms of sex. Comorbidity rates may be particularly important given data suggesting that 70% of social phobics suffer from at least one other anxiety disorder (Van Amerigen, Mancini, Stryan, & Donison, 1991), and that comorbidity is likely to have a negative impact on the course of treatment (Turner, Beidel, Borden, Stanley, & Jacob, 1991). Similarly, the classification of subtype distributions may be relevant in light of data suggesting that generalized social phobics are more severely impaired and treatment resistant than are discrete social phobics (Brown, Heimberg, & Juster, 1994; Heimberg et al., 1990).

A potential criticism of this and other meta-analyses, as they are considered in relation to treatment decisions occurring in clinical settings, is that the sample studied and data obtained may not be fully representative of social phobics who decline participation in a randomized controlled trial. However, this criticism is tempered by data reported by Heimberg and his colleagues (Juster, Heimberg, & Engelberg, 1995) demonstrating that those social phobics refusing versus those accepting random assign-

ment did not respond differentially to CBT. Another potential shortcoming of this study is that use of self-report measures, rather than blinded clinical assessment, while increasing reliability and decreasing bias between studies, may have given an incomplete picture of the nature and degree of change in patients over time. Further research is necessary to assess the role of patient and clinician conceptualization of disorder and treatment modality preferences in assessing the relative risks and benefits of pharmacologic and cognitive-behavioral interventions.

Future research on social phobia should address several methodological and general research issues. One of the major shortcomings in the social phobia literature is the paucity of reliable follow-up data. Medication studies have largely failed to assess patients beyond initial 3-month trials, and need to examine the course of disorder after discontinuation from these agents particularly given tentative evidence that CBT and pharmacotherapy may have very different patterns of maintenance of treatment gains over follow-up periods (Juster & Heimberg, 1995). Cognitive-behavioral studies also need to improve tracking longer-term treatment outcome and tracking patients who drop out of follow-up conditions. For example, only 50% of the original patients in the Heimberg et al. (1993) study took part in the follow-up evaluations that took place over a period of 5.5 years. Studies would also benefit from determining which subjects are given additional therapy during the follow-up period, and the nature of that therapy. For example, Mersch, Emmelkamp, and Lips (1991) found that 44% of subjects receiving either social skills training or rational emotive therapy for social phobia had been given additional therapy at 14 months posttreatment. All types of treatment studies need to do a better job at determining the differences between patients who maintain their gains, and those who show an initial positive response, only to relapse during follow-up.

Future studies of social phobia also should make better diagnostic determinations of which social phobia patients have APD. Because APD is thought to be a more treatment-resistant disorder (Feske, Perry, Chambless, Renneberg, & Goldstein, 1996), it can potentially confound research findings; only one third of the studies in this meta-analysis reported APD in their samples. Evaluation of the potential overlap between social phobia and APD has been complicated by significant changes in the criteria for both conditions from the *DSM-III* to the

*DSM-III-R*, and remains unresolved with the advent of the *DSM-IV*. To date there is no consensus as to whether APD is on the extreme continuum of social phobia disorder, or a distinct disorder. Indeed, APD by itself may be an excellent predictor of treatment nonresponse across interventions. Better evaluation of, and counterbalancing for the percentage of subjects in a sample that meet APD criteria can improve trials. In addition, more general evaluation of the interaction between other individual difference variables (e.g., personality traits, or cognitive styles) and specific treatment techniques is in order. Is social phobia mediated primarily by cognitive deficits in some patients, and by conditioned behavioral inhibition in others? Are patients with identifiable cognitive deficits more likely to respond to specific cognitive techniques, or can significant improvement be mediated by noncognitive interventions?

For patients meeting the social phobia diagnosis, better tracking of generalized versus specific subtype may allow even finer determinations of severity within samples given that generalized subtype may be less responsive to all treatments (Brown et al., 1995). Similarly, better evaluation of problems of comorbid anxiety and affective disorders would improve comparability between study samples. In the recently published National Comorbidity Survey (Magee, Eaton, Wittchen, McGonagle, & Kessler, 1996) 56.9% of 1077 subjects with social phobia had at least one other anxiety disorder. In the same survey, 41.4% of subjects with social phobia had a history of at least one affective disorder. Because comorbid disorders are likely to interfere with treatment progress, their identification would help identify potential predictors of treatment nonresponse.

Treatment contamination may be a particularly relevant issue for social phobia because of the ease with which exposure instructions may be conveyed to patients. Some medication studies (e.g., Gelertner et al., 1991) encouraged participants to engage in systematic self-exposure to situations that they avoid. With this design it becomes difficult to determine to what extent changes during treatment were due to the medication, or to the exposure instructions. Treatment contamination may also occur in less detectable ways. Power and Sharp (1995), for example, questioned the efficacy of CBT for panic disorder based on the inclusion, in some studies, of patients using concurrent psychotropic medication. Effective

monitoring and/or restriction of CBT subjects' use of concurrent medication and medication subjects' concurrent use of CBT is essential for ensuring valid interpretation of data from these trials. For CBT studies, treatment integrity could also be enhanced by using manualized treatments and reviewing recorded sessions to ensure that therapists are not drifting from the protocol.

Another methodological issue that can potentially influence comparisons between medication and CBT studies concerns control conditions. Pharmacotherapy studies may be at a slight disadvantage in controlled meta-analytic comparisons because of the relative strength of pill placebo control groups. Psychosocial studies could better equate this difference by using credible attention placebo controls that controlled for the nonspecific effects of being in a treatment condition. Attention placebo controls could be designed to include as many of the characteristics of the experimental treatment as possible (e.g., number of sessions, experiment length, experimenter involvement) without the hypothesized active treatment ingredients. For example, Heimberg and his colleagues (Heimberg, Dodge et al., 1990; Heimberg et al., 1993) have created a credible attention placebo called Educational Supportive Group Psychotherapy that encompasses these nonspecific elements, and that could be emulated in other CBT studies.

Future research concerning enhancing the effectiveness of social phobia treatments may benefit from further examining combination CBT/pharmacotherapy treatments. Only two studies in this meta-analysis used combination interventions, and both studies yielded moderate effect sizes. In addition, only the Clark and Agras (1991) study used the full compliment of CBT components. Future studies could examine the timing, sequencing, and mixture of treatment elements in combination interventions that would render the most potent treatment packages. Just as important, these studies could help better discern whether the effects of combination interventions are additive, synergistic, or even detrimental. For example, one could posit that the addition of medications to CBT could have a potentially detrimental effect because of the tendency for individuals to attribute their success to the medication and not to their own cognitive and behavioral strategies to cope with social anxiety. Brief application of medication treatment (e.g., BZD treatment during 4–6 weeks of CBT, followed by medication taper)

may be one method of successfully combining medications and CBT without detracting from the promising long-term outcome afforded by CBT.

In addition, our own current clinical impression is that treatment of social phobia can be aided by borrowing strategies from current CBT protocols for panic disorder (Gould & Otto, 1995). In particular, in our own group program we have added interoceptive exposure and breathing retraining to our program of standard cognitive restructuring and in vivo exposure for social phobia. It is our belief that direct training in the modification of emotional and cognitive responses to anxiety symptoms achieved through interoceptive exposure aids fear reduction and helps ensure more productive and successful in vivo exposure experiences. Encouraging data from the application of interoceptive exposure to specific phobias provide tentative support for this proposition (Zarate, Rapee, Craske, & Barlow, 1988), and awaits further empirical assessment.

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