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Research Article

ESCITALOPRAM IN THE TREATMENT OF SOCIAL ANXIETY DISORDER: ANALYSIS OF EFFICACY FOR DIFFERENT CLINICAL SUBGROUPS AND SYMPTOM DIMENSIONS

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Escitalopram has demonstrated efficacy for the acute treatment of social anxiety disorder (SAD) in two placebo-controlled trials and for long-term treatment in a relapse-prevention study. Social anxiety disorder is a heterogeneous disorder. This study questions whether this new selective serotonin reuptake inhibitor is effective across different subgroups of patients. Data from two randomised, placebo-controlled, 12-week escitalopram SAD trials were pooled. General linear models were used to determine the efficacy of escitalopram in different patient subgroups. Furthermore, a factor analysis of the primary efficacy scale, the Liebowitz Social Anxiety Scale (LSAS), was undertaken, and a determination made of whether treatment effects were similar for the different symptom dimensions. Escitalopram was effective in both younger and older patients, in male and female patients, and in patients with more and less severe social anxiety symptoms. The LSAS factor analysis showed six factors, which were differentially associated with different areas of disability. Escitalopram was significantly superior to placebo for all six symptom dimensions. The treatment effects of escitalopram were independent of gender, symptom severity and chronicity, and comorbid depressive symptoms. A six-factor model of social anxiety symptoms is supported by the distinctive association between these symptom dimensions and different areas of disability, but did not predict differential response to escitalopram. Depression and Anxiety 20:175–181, 2004. © *2005 Wiley-Liss, Inc.*

Key words: *escitalopram; social anxiety disorder; factor analysis; Liebowitz Social Anxiety Scale; symptom dimension; Sheehan Disability Scale*

INTRODUCTION

Social anxiety disorder (SAD), or social phobia, is increasingly seen as a serious medical condition that is accompanied by significant disability [Ballenger et al., 1998; Magee et al., 1996]. This view has been strengthened by data showing that SAD cannot simply be equated with shyness or with avoidant personality traits, and by evidence that this disorder is a discrete

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entity associated with specific psychobiological dysfunctions and selectively responsive to specific pharmacotherapeutic interventions [Ballenger et al., 1998; Blanco et al., 2003].

Social anxiety symptoms can be conceptualized as lying on a spectrum from severe shyness through to SAD and on to avoidant personality disorder [Schneier et al., 2002]. Within SAD, patients have been divided into those with generalized and non-generalized (discrete, specific, circumscribed) symptoms [Heimberg et al., 1993], and into those with social interaction fears and those with pure speaking fears [Kessler et al., 1998; Eng et al., 2000]. Pharmacotherapeutic dissection of different SAD subtypes may be possible; for example, beta-blockers may be useful for performance anxiety but are ineffective in generalized SAD [Liebowitz et al., 1992].

The Liebowitz Social Anxiety Scale (LSAS) [Liebowitz, 1987] is the most commonly used primary efficacy scale in medication studies of SAD. Nevertheless, there are relatively few studies of its factorial structure [Oakmane et al., 2003; Perugi et al., 2001; Safren et al., 1999], and to our knowledge there are no published studies that have explored the relationship between clinical subgroups of SAD based on LSAS factor loading and treatment outcome. Such work might be able to contribute to the growing literature focused on predicting response to pharmacotherapy in SAD [Stein et al., 2002a]. We pooled data from multisite, randomised, placebo-controlled studies of escitalopram in SAD to predict the response to treatment of different patient subgroups and symptom dimensions.

PATIENTS AND METHODS

CLINICAL TRIALS

The escitalopram SAD trials have been presented in more detail elsewhere. There have been two multinational, randomised, placebo-controlled, parallel group, 12 and 24-week studies [Kasper et al., 2004; Lader et al., 2004]. In the first study, after a 1-week, single-blind placebo lead-in period, patients were randomised to 12 weeks of double-blind treatment with escitalopram (10–20 mg/day) or placebo [Kasper et al., 2004]. In the second study, after the placebo lead-in, patients were randomly assigned to 24 weeks of double-blind treatment with escitalopram (5, 10, or 20 mg/day), paroxetine (20 mg/day) or placebo [Lader et al., 2004]. In addition, data from a longer relapse prevention study were used in the exploratory factor analysis [Montgomery et al., 2003].

Subjects were 18–65 years of age, had a primary diagnosis of generalized SAD according to DSM-IV criteria, and had a total score of at least 70 on the LSAS. Patients were excluded if they had moderate to severe depressive symptoms, or any of a range of comorbid psychiatric or general medical disorders. In the clinical studies, the primary efficacy variable was the LSAS, which was assessed at baseline and at regular intervals up to 12 or 24 weeks of treatment. Secondary outcomes in these two studies included the Montgomery-Åsberg Depression Rating Scale (MADRS) and the Sheehan Disability Scale (SDS).

The LSAS provides an overall measure of social anxiety as a total score and four subscales: performance fear, social fear, performance avoidance, and social avoidance. There are 24 items, and each is rated in terms of both fear and avoidance on a 0–3 scale. The SDS rates disability in three different arenas (social, family, work), each on a 0–10 scale.

For the analysis of treatment outcome, 12-week escitalopram and placebo data from both the 12- and 24-week trials were pooled. For an exploratory factor analysis to determine whether LSAS items measure a smaller number of SAD symptom dimensions, baseline LSAS data from all three trials were used.

STATISTICAL ANALYSES

A first analysis aimed at determining the efficacy of escitalopram versus placebo in different patient subgroups. Median scores were used to define groups based on SAD severity, SAD duration, and extent of comorbid depressive symptoms. General linear models were then used to determine the efficacy of escitalopram in the following patient subgroups: males versus females, more severe (LSAS $\geq$ 95) versus less severe (LSAS $<$ 95), more chronic SAD (SAD duration $\geq$ 18 years) versus less chronic SAD (SAD duration $<$ 18 years), and with more comorbid depressive symptoms (MADRS $>$ 12) versus with fewer depressive symptoms (MADRS $\leq$ 12).

A second analysis determined whether the LSAS items measure a smaller number of symptom dimensions of SAD. This was done using an exploratory factor analysis to model the relationship between manifest variables (the LSAS data) and underlying latent variables (the LSAS factors, or symptom dimensions). An exploratory factor analysis was chosen, as previous factor analyses of the LSAS have reached somewhat different conclusions, and an exploratory analysis does not explicitly posit any particular structural model. Several different statistical methods exist for determining whether the model fits the data; here a maximum likelihood solution was used for factor identification, as this is a method that is independent of the scales of measurement and that allows comparison of factor solutions using objective criteria [Krzanowski, 1991]. A first approach to the factor analysis was to add the paired items (fear and avoidance) of the LSAS so producing 24 items, whereas a second approach was to analyse the fear and avoidance items separately. On the basis of the factor analysis, symptom subscales were then created by adding LSAS scores from items corresponding to factors.

A third analysis assessed relationship between the LSAS factors and disability, as measured by the

Sheehan Disability Scale (SDS). For each of the three components of the SDS (social, family, work) an analysis of covariance (ANCOVA) was fitted, including centre and the symptom subscale scores to examine which of the factors had an effect on these components. The subscale scores were expressed in terms of *z*-scores to take into account that the subscales consist of a different number of items.

Finally, the relationship between symptom subscale scores and treatment outcomes was assessed. ANCOVA analyses were performed on each of the defined subscales adjusting for baseline value of the subscale in question, center, and treatment.

RESULTS

The data for the main analyses was from 1,197 randomized and treated patients. Approximately half were women and their mean LSAS score at baseline ranged from 92.4–96.3 in the different treatment groups (Table 1). For the exploratory factor analysis an additional 517 patients [Montgomery et al., 2003] were included, for a total of 1,714.

Escitalopram was effective in both male ($P < .01$) and female patients ($P < .001$), in both more severe ($P < .001$) and less severe ($P < .001$) patients, in both patients with more chronic ($P < .01$) and less chronic ($P < .001$) SAD, and in patients with ($P < .01$) and without ($P < .001$) comorbid depressive symptoms.

A scree plot of eigenvalues [Krzanowski, 1991] indicated that either a 6- or 7-factor model was reasonable. Formally, the model with seven factors gave the best fit. The model with seven factors, however, had just one item with major loading on the last factor, and that was deemed not satisfactory. A six-factor model was therefore chosen (Table 2), with final communality estimates ranging from 0.16 (Item 21) to 0.90 (Item 3). When factor analysis was done separately on the fear and avoidance items, very similar results were obtained to those seen with the paired items. On the basis of their component items, the six factors could be labeled as follows: Factor 1, social interaction (5, 10, 11, 12, 19, 21); Factor 2, eating and drinking in public (3, 4); Factor 3, speaking in public (2, 6, 14, 15, 16, 20); Factor 4, assertiveness (1, 13, 18, 22, 24); Factors 5, observation fear (8, 9, 17); and Factor 6, partying (7, 23).

The six LSAS factors differentially predicted disability (Figs. 1–3). For SDS Social, LSAS Factors 1–3 and Factor 6 were statistically significant. Factor 6 (partying) had the largest effect whereas Factor 4 (assertiveness) and Factor 5 (observation fear) were non-significant. For SDS Family, the only LSAS factor that was non-significant was Factor 5 (observation fear) whereas the main contribution came from Factor 6 (partying). For SDS Work three LSAS factors were statistically significant: Factor 3 (speaking in public), Factor 4 (assertiveness), and Factor 5 (observation fear). The main contributions came from Factors 3 and 5.

LSAS factors did not significantly predict response to treatment. Escitalopram was significantly superior to placebo for Factors 1–4, 6 ($P < .001$), and Factor 5 ($P < .05$), with effect sizes similar across the different factors (Fig. 4).

DISCUSSION

The development of a view of SAD as a discrete medical disorder that deserves early diagnosis and robust treatment represents a significant advance. SAD is a prevalent and disabling disorder that is characterized by particular psychobiological dysfunctions, and which responds well to select pharmacotherapeutic and psychotherapeutic interventions [Stein et al., 2002b; van der Linden et al., 2000]. Nevertheless, continued progress in understanding and managing SAD may well require progress in delineating the subtypes and spectra of this condition [Heimberg et al., 1993; Schneier et al., 2002].

Several previous studies have attempted to tackle this question by determining predictors of response to treatment [Stein et al., 2002a,c]. These data support previous work indicating that demographic and clinical variables do not consistently predict treatment response. An early suggestion that heart rate predicted response was not confirmed with a larger patient sample [Stein et al., 2002a]. Trail variables (e.g., duration of treatment) show that longer courses of

TABLE 1. Baseline characteristics for the 12- and 24-week social anxiety disorder treatment studies

	Kasper et al., 2004		Lader et al., 2004					
Parameter	PBO	ESC	PBO	ESC 5 mg	ESC 10 mg	ESC 20 mg	PAR	Total
Patients treated (*n*)	177	181	166	167	167	170	169	1,197
Patients completed (*n*)	145	145	116	125	111	121	124	887
% Female	46.9	44.2	51.2	49.7	57.5	52.9	53.8	50.9
Age (yr)	36.2	39.0	37.0	36.3	37.2	37.0	37.4	37.2
MADRS at baseline	7.5	7.6	7.6	6.8	6.8	7.3	7.2	7.3
LSAS at baseline	95.4	96.3	96.0	94.3	92.4	94.0	94.1	94.6

PBO, placebo; ESC, escitalopram; PAR, paroxetine; MADRS, Montgomery-Åsberg Depression Rating Scale; LSAS, Liebowitz Social Anxiety Scale.

TABLE 2. Factor loadings of the 6-factor model

Item	F1	F2	F3	F4	F5	F6
11. Talking with people you don't know very well.	**0.80**	0.02	0.04	0.11	0.01	0.07
12. Meeting strangers.	**0.61**	0.04	0.12	0.13	0.03	0.18
10. Calling someone you don't know very well.	0.46	−0.13	−0.03	0.33	0.08	0.03
19. Looking people you don't know very well in the eye.	0.39	0.05	−0.01	0.28	0.07	0.03
5. Talking to people in authority.	0.35	−0.06	0.13	0.16	0.22	−0.03
21. Trying to make someone's acquaintance.	0.26	−0.02	0.07	0.18	−0.04	0.24
3. Eating in public places.	−0.02	**0.94**	0.03	0.04	0.10	0.09
4. Drinking with others in public places.	−0.04	**0.74**	0.03	0.07	0.12	0.11
20. Giving a report to a group.	−0.05	0.04	**0.63**	−0.09	0.07	−0.00
6. Acting, performing or giving a talk in front of an audience.	−0.00	−0.03	**0.54**	−0.07	0.02	−0.03
15. Being the centre of attention.	0.08	0.01	0.50	−0.02	0.03	0.07
16. Speaking up at a meeting.	0.05	−0.05	0.45	0.06	0.03	0.01
2. Participating in small groups.	0.11	0.11	0.35	0.05	0.12	0.17
14. Entering a room when others are already seated.	0.11	0.17	0.29	0.09	0.13	0.14
22. Returning goods to a store.	0.14	0.04	0.02	**0.59**	0.05	0.05
24. Resisting a high-pressure salesperson.	0.17	0.02	0.00	**0.55**	0.08	0.04
1. Telephoning in public.	0.10	0.05	−0.01	0.38	0.17	0.01
18. Expressing disagreement or disapproval to people you don't know very well.	0.25	−0.04	0.11	0.37	0.04	0.02
13. Urinating in a public bathroom.	0.06	0.08	−0.11	0.34	0.10	0.14
8. Working while being observed.	0.10	0.05	0.11	0.11	**0.73**	0.03
9. Writing while being observed.	0.05	0.11	0.03	0.13	**0.67**	0.04
17. Taking a test.	0.01	0.05	0.13	0.11	0.24	0.06
7. Going to a party.	0.08	0.19	0.07	−0.01	0.10	**0.87**
23. Giving a party.	0.09	0.03	0.09	0.12	0.04	0.49

*Figures in bold are loadings above 0.5. Factor 1 (F1), social interaction; F2, eating and drinking in public; F3, speaking in public; F4, assertiveness; F5, observation fear; F6, partying.

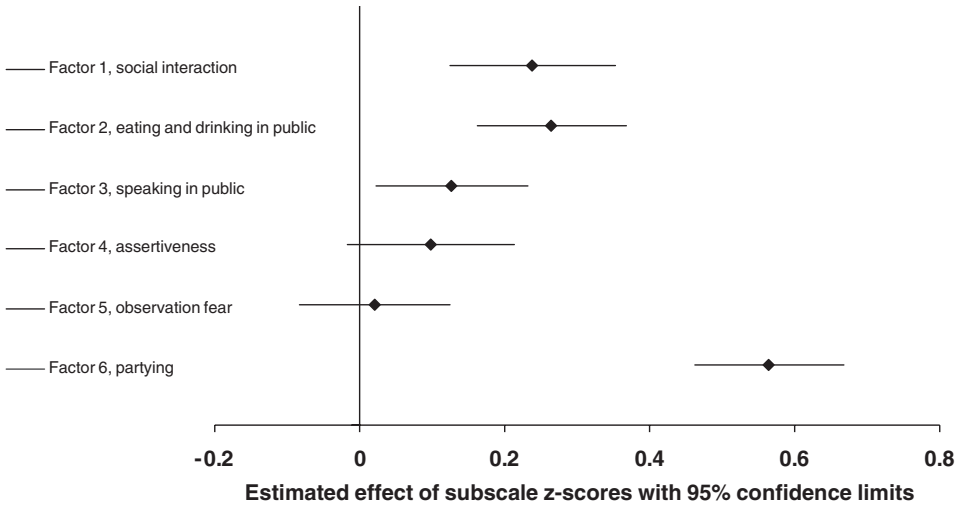

Fig. 1. SDS Social. The effect of subscale z-scores on Sheehan Disability Scale (SDS) Social at baseline adjusting for centre.

pharmacotherapy are associated with better outcome [Stein et al., 2002a]. Factor analysis of the LSAS has not been used previously to predict response to pharmacotherapy.

Previous LSAS factor analyses have differed slightly in their methods and results. The exploratory analysis of the LSAS in 382 SAD patients by Safren et al. [1999] yielded four factors (social interaction, speaking in public, observation fear, eating and drinking in public), and Oakman et al. [2003] supported this with a four-factor confirmatory analysis of the self-report version of the LSAS in 188 patients with various anxiety disorders. Perugi et al. [2001] conducted a principal components analysis of the LSAS in 153 SAD patients, and found a fifth factor, with social interaction divisible into items involving interpersonal anxiety and those involving stranger-authority anxiety. In our analysis, undertaken in a larger sample than previously used, we again found factors for social interaction, speaking in public, eating and drinking in public, and observation

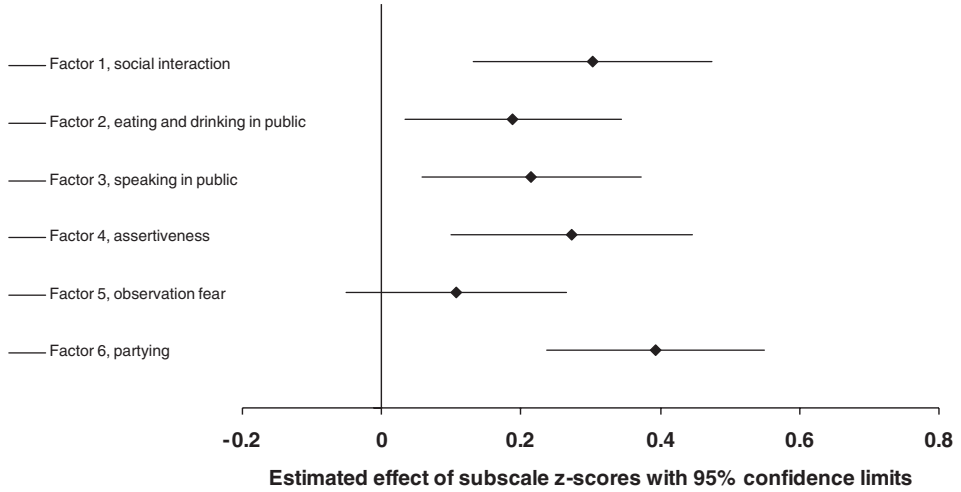

Fig. 2. SDS FAMILY. The effect of subscale z-scores on Sheehan Disability Scale (SDS) Family at baseline adjusting for centre.

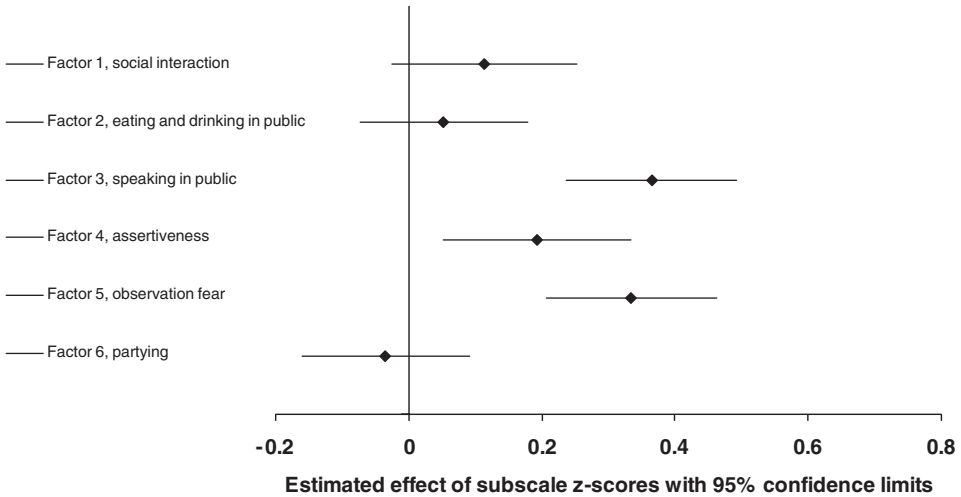

Fig. 3. SDS WORK. The effect of subscale z-scores on Sheehan Disability Scale (SDS) Work at baseline adjusting for centre.

fear. In addition, however, some of the items described as "social interaction" in the previous work fell into separate factors that we termed "assertiveness" and "partying."

The previous factor analysis research on the LSAS has noted that although the LSAS subscales for social interaction and performance symptoms have face validity, factor analysis of the LSAS does not provide construct validity [Oakman et al., 2003; Safren et al., 1999]. Factor analysis of other social anxiety scales also fails to confirm a 2-factor model [Safren et al., 1998]. Nevertheless, some of the factors (social interaction, partying) largely reflect social fears, whereas others (eating and drinking in public, observation fear) largely reflect performance fears.

Factor analyses of the LSAS, including the work here, overlap in part with patterns identified using other measures. Early on, Dixon et al. [1957] described "fear of exhibitionism" in patients with social anxiety, and subsequent work has described the factor of "fear of performing actions while being observed" [Connor et al., 2000; Davidson et al., 1997] or of "anxiety about being observed by others" [Safren et al., 1998]. Differences in factors obtained with different measures can in part by explained by differences in the SAD symptom dimensions that they assess. The LSAS does not include a specific measure of physiological discomfort, whereas work with other measures has led to the description of "fear of loss of control, especially bodily control" and a factor representing physiological symptoms [Connor et al., 2000; Davidson et al., 1997], as well as "fear that others will notice anxiety symptoms" [Safren et al., 1998].

The 6-factor model obtained here was supported by the finding that different factors were associated with

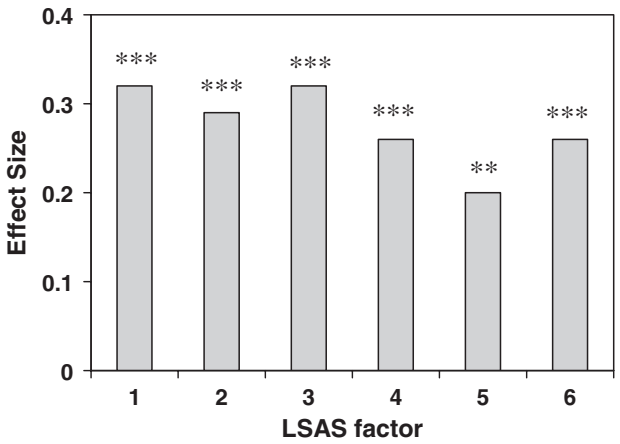

Fig. 4. Effect size in Liebowitz Social Anxiety Scale (LSAS) analyses by subscales (LOCF). Factor 1 - social interaction; factor 2 - eating and drinking in public; factor 3 - speaking in public; factor 4 – assertiveness; factor 5 – observation fear; factor 6 - partying. $^{**}p<0.01$; $^{***}p<0.001$.

different patterns of disability, as assessed by the SDS. There is growing awareness of disability associated with the anxiety disorders in general, and with SAD in particular [Mogotsi et al., 2000]. Compared to other anxiety disorders, SAD may have a particularly negative impact on social function [Lochner et al., 2003]. Theses findings demonstrate that delineating the symptom dimensions of SAD may be useful for understanding fully the nature of the disability associated with this condition. It is notable, for example, that assertiveness and observation fear do not significantly impact on social function, but are particularly relevant to occupational disability.

Escitalopram was significantly superior to placebo for all factors. It seems that current pharmacological interventions for SAD affect the different LSAS items, and their underlying symptom dimensions, to a similar extent. Although generalized SAD seems particularly heritable [Stein et al., 1998], one implication of the current finding is that both interactional and performance symptom dimensions are mediated in part by the serotonin system. This is consistent with previous work demonstrating that both more generalized and less generalized SAD respond to treatment with an SSRI [Stein et al., 2001].

Some limitations of the current study can be mentioned. First, some items in the factor analysis loaded weakly on more than one factor; in particular, several items (e.g., "attempting to make someone's acquaintance") showed aspects of both social interaction and assertiveness difficulties. Previous factor analysis work on social phobia measures has similarly found that certain items load on multiple factors [Safren et al., 1998, 1999]; future work could potentially attempt to develop items that address hypothesized symptom dimensions of SAD with greater specificity. Second, although the LSAS covers a broad range of social anxiety symptoms, other symptoms in SAD may also contribute to its symptom structure. As noted above, the LSAS does not assess common somatic symptoms in SAD (e.g., blushing, tremor). Finally, SAD subjects who qualify for a clinical trial may not be representative of typical patients with this disorder. Patients in the escitalopram trials did not have comorbid disorders, and were required to have a minimum score on the LSAS. Thus more extensively ill subjects, and subjects with milder or with more discrete SAD were excluded from the factor analysis. It is notable that the median baseline LSAS score of 95 in the current dataset represents a comparatively severe level of SAD symptoms.

This study supports the multidimensionality of social anxiety symptoms, and the value of assessing symptom dimensions for understanding aspects of SAD psychopathology such as associated disability. Future scoring of the LSAS might benefit from calculating separate summary scores for different factors [Safren et al., 1999]. Further work is needed to delineate the predictors of non-response to pharmacotherapy, and to understand the mechanisms that underlie treatment-resistant SAD. In the meantime, the current data extend previous work on escitalopram in SAD [Kasper et al., 2004; Lader et al., 2004] by showing its efficacy across a broad range of SAD patients and symptoms of SAD.

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REFERENCES

Ballenger JC, Davidson JA, Lecrubier Y, Nutt DJ, Bobes J, Beidel DC, Ono Y, Westenburg HG. 1998. Consensus statement on social anxiety disorder from the international consensus group on depression and anxiety. J Clin Psychiatry 59:54–60.

Blanco C, Raza MS, Schneier FR, Liebowitz MR. 2003. The evidence-based pharmacological treatment of social anxiety disorder. Int J Neuropsychopharmacol 6:427–442.

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

Davidson JR, Miner CM, DeVeaugh-Geiss J, Tulper LA, Colket JT, Potts NL. 1997. The Brief Social Phobia Scale: A psychometric evaluation. Psychol Med 27:161–166.

Dixon JJ, de Monchaux C, Sandler J. 1957. Patterns of anxiety: An analysis of social anxieties. Br J Med Psychol 30:107–112.

Eng W, Heimberg RG, Coles ME, Schneier FR, Liebowitz MR. 2000. An empirical approach to subtype identification in individuals with social phobia. Psychol Med 30:1345–1357.

Heimberg RG, Holt CS, Schneier FR, Spitzer RL, Liebowitz MR. 1993. The issue of subtypes in the diagnosis of social phobia. J Anxiety Disord 7:249–269.

Kasper S, Stein DJ, Loft H, Nil R. 2004. Escitalopram is effective and well tolerated in the treatment of social anxiety disorder: A randomised, placebo-controlled, flexible-dose study. Br J Psych (in press).

Kessler RC, Stein MB, Berglund P. 1998. Social phobia subtypes in the National Comorbidity Study. Am J Psychiatry 155:613–619.

Krzanowski WJ. 1991. Principles of multivariate analysis. A user's perspective. Oxford, UK: Oxford, Science Publications.

Lader M, Stender K, Bürger V, Nil R. 2004. The efficacy and tolerability of escitalopram in the short- and long-term treatment of social anxiety disorder: A randomised, double-blind, placebo-controlled, fixed-dose study. Depress Anxiety 19:241–248.

Liebowitz MR. 1987. Social phobia. In: Ban TA, Pichot P, Poldinger W, editors. Modern problems of pharmacopsychiatry. Basel: Karger.

Liebowitz MR, Schneier F, Campeas R, Hollender E, Hatterer J, Fryer A, Gorman J, Papp L, Davies S, Gullly R, et al. 1992. Phenelzine vs. atenolol in social phobia: A placebo-controlled comparison. Arch Gen Psychiatry 49:290–300.

Lochner C, Mogotsi M, du Toit PL, Kaminer D, Niehaus DJ, Stein DJ. 2003. Quality of life in the anxiety disorders: A comparison of obsessive-compulsive disorder, social anxiety disorder, and panic disorder. Psychopathology 36:255–262.

Magee WJ, Eaton WW, Wittchen HU, McGonagle KA, Kessler RC. 1996. Agoraphobia, simple phobia, and social phobia in the National Comorbidity Survey. Arch Gen Psych 53:159–168.

Mogotsi M, Kaminer D, Stein DJ. 2000. Quality of life in the anxiety disorders. Harv Rev Psychiatry 8:273–282.

Montgomery SA, Dürr-Pal N, Loft H, Nil R. 2003. Relapse prevention by escitalopram treatment of patients with social anxiety disorder (SAD). Eur Neuropsychopharmacol 13S4:364.

Oakman J, van Ameringen M, Mancini C, Farvolden P. 2003. A confirmatory factor analysis of a self-report version of the Liebowitz Social Anxiety Scale. J Clin Psychol 59:149–161.

Perugi G, Nassin S, Maremmani I, Madaro D, Toni C, Simonini E, Akiskal HS. 2001. Putative clinical subtypes of social phobia: A factor-analytic study. Acta Psychiatr Scand 104:280–288.

Safren SA, Heimberg RG, Horner KJ, Juster HR, Schneier FR, Liebowitz MR. 1999. Factor structure of social fears: The Liebowitz Social Anxiety Scale. J Anxiety Disord 13: 253–270.

Safren SA, Turk CL, Heimberg RG. 1998. Factor structure of the Social Interaction Anxiety Scale and the Social Phobia Scale. Behav Res Ther 36:443–453.

Schneier FR, Blanco C, Antia SX, Liebowitz MR. 2002. The social anxiety spectrum. Psychiatr Clin North Am 25:757–774.

Stein DJ, Stein MB, Goodwin W, Hunter B. 2001. The selective serotonin reuptake inhibitor paroxetine is effective in more generalized and less generalized social anxiety disorder. Psychopharmacology 158:267–272

Stein DJ, Stein MB, Pitts CD, Kumar R, Hunter B. 2002a. Predictors of response to pharmacotherapy in social anxiety disorder: An analysis of 3 placebo-controlled paroxetine trials. J Clin Psychiatry 63:152–155.

Stein DJ, Westenberg H, Liebowitz MR. 2002b. Social anxiety disorder and generalized anxiety disorder: Serotonergic and dopaminergic neurocircuitry. J Clin Psychiatry 63S6:12–19.

Stein DJ, Cameron A, Amrein R, Montgomery SA. 2002c. Moclobemide is effective and well tolerated in the long-term pharmacotherapy of social anxiety disorder with or without comorbid anxiety disorder. Int Clin Psychopharmacol 17: 161–170.

Stein MB, Chartier MJ, Hazen A, Kozak MV, Tancer ME, Lander S, Furer P, Chubaty D, Walker JR. 1998. A direct-interview family study of generalized social phobia. Am J Psychiatry 155: 90–97.

van der Linden GJH, Stein DJ, van Balkom AJLM. 2000. The efficacy of the selective serotonin reuptake inhibitors for social anxiety disorder (social phobia): A meta-analysis of randomized controlled trials. Int Clin Psychopharmacol 15S2:15–24.