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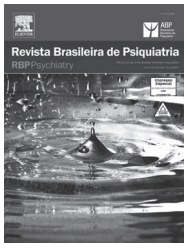

REVIEW ARTICLE

A review on predictors of treatment outcome in social anxiety disorder

Sara Costa Cabral Mululo,[1] Gabriela Bezerra de Menezes,[1] Paula Vigne,[1] Leonardo F. Fontenelle[1,2]

[1] Programa de Pesquisa em Ansiedade e Depressão, Instituto de Psiquiatria, Universidade Federal do Rio de Janeiro (IPUB/UFRJ), Rio de Janeiro, Brazil
[2] Instituto D'Or de Pesquisa e Ensino, Rio de Janeiro & Departmento de Psiquiatria e Saúde Mental, Instituto de Saúde da Comunidade, Universidade Federal Fluminense (MSM/UFF), Niterói, Brazil

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DESCRIPTORS
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Abstract
Objective: This article aims to review the clinical features and therapeutic characteristics that may predict treatment response in patients with social anxiety disorder (SAD). *Methods*: A systematic review of trials identified through databases of ISI, Medline, PsycInfo, Cochrane, LILACS, Current Controlled Trials, and in references of previously selected articles published in English up to December 2010. In our literature search, we used the words prediction/predictors and social anxiety disorder or social phobia. *Results*: Early onset, greater disease severity, comorbidity with other anxiety disorders (including generalized anxiety disorder and simple phobia), and high expectations about the role of the therapist emerged as potential predictors of less effective treatment in SAD. *Conclusions*: Knowledge of various clinical and treatment features may help professionals to predict possible responses to therapeutic interventions in patients with SAD. However, given the diversity of measures used to assess response, further studies should be performed with standardized methods to investigate the aspects related to treatment resistance in SAD.

Corresponding author: Leonardo F. Fontenelle; Rua Visconde de Pirajá 547 Sala 719; 22410-003, Ipanema, Rio de Janeiro, Brazil; Fax: (.55 21) 2239-4919; *E-mail*: lfontenelle@gmail.com

DESCRITORES:
Transtornos fóbicos;
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Uma revisão sobre fatores de predição de resposta ao tratamento no transtorno de ansiedade social

Resumo
Objetivo: Este artigo tem por objetivo descrever as principais características clínicas e terapêuticas que possam predizer resposta ao tratamento em pacientes com transtorno de ansiedade social (TAS). *Métodos*: Revisão sistemática de ensaios clínicos identificados através das bases de dados *ISI*, *Medline*, *PsycInfo*, *Cochrane*, *LILACS*, *Current Controlled Trials* e em referências bibliográficas de artigos previamente selecionados publicados em inglês até dezembro de 2010. As seguintes palavras-chave foram utilizadas em nossa busca bibliográfica: *prediction/predictors* e *social anxiety disorder ou social phobia*. *Resultados*: Início precoce, maior gravidade da doença, comorbidade com outros transtornos de ansiedade (incluindo o transtorno de ansiedade generalizada e fobia simples) e alta expectativa sobre o papel do terapeuta emergiram como potenciais fatores de predição menor eficácia do tratamento do TAS. *Conclusões*: O conhecimento de uma variedade de características clínicas e de tratamento pode auxiliar os profissionais a preverem possíveis respostas às intervenções terapêuticas nos pacientes com TAS. No entanto, devido à diversidade de medidas utilizadas para avaliar a resposta, novos estudos com o objetivo de investigar aspectos relacionados à resistência ao tratamento do TAS devem ser realizados com métodos mais padronizados.

Introduction

Social anxiety disorder (SAD) is characterized by intense and persistent fear of being negatively evaluated by others, which leads to significant avoidance or intense psychological distress in various social situations.[1] SAD has high prevalence rates, early and insidious onset, and a chronic course.[2] It is associated with low levels of quality of life; significant feelings of incapacity; and notorious social, educational, and occupational disability.[3] Despite remaining largely underdiagnosed,[4] recent studies are beginning to unveil SAD neurobiological underpinnings, thus leading to greater knowledge regarding its pathophysiology.[5]

Currently, the most empirically effective treatments of SAD include pharmacotherapy with selective serotonin reuptake inhibitors (SSRIs) or serotonin and norepinephrine reuptake inhibitors (SNRIs), and cognitive behavioral therapy (CBT), either together or separately.[6,7,8] Although these strategies lead to a therapeutic response in a large proportion of individuals, a significant number of patients with SAD (up to 50%) does not respond at all or remain significantly symptomatic after being submitted to first-line treatments.[9,10,11]

This article aims to review the clinical and therapeutic characteristics that have been reported to be associated with response to treatment prescribed to patients with SAD. Potentially, the characterization of these features may save patients' time by avoiding ineffective treatments, which may be sometimes associated with severe side effects and/or economic burden. It could also help to identify factors that should be taken into account if one aims at elaborating more specific treatment for SAD individuals who do not respond to conventional strategies.

Methods

A systematic review of clinical trials that describe the profile of patients with SAD who respond to treatment was performed in the following databases: ISI/Web of science, Medline, PsycInfo, Cochrane Library, LILACS, and Current Controlled Trials (articles not published yet). An additional search for articles of interest was performed in the bibliographic list of selected articles. The following keywords were used in the bibliographical search: prediction/predictors and social anxiety disorder or social phobia. Clinical trials originally published in English up to December 2010 were assessed.

Inclusion criteria consisted of studies composed of subjects with a primary diagnosis of SAD according to the DSM-III-R or DSM-IV and describing an assessment of treatment response predictors in this condition. We included papers that addressed the following aspects as potential response predictors: (i) clinical features (age of onset, duration of SAD, severity of symptoms, generalized subtype of SAD, family history of SAD, and psychiatric comorbidity); (ii) sociodemographic characteristics (age, gender, educational level, and marital status); and (iii) treatment features (treatment history, duration of current treatment, expectancy regarding treatment, and group cohesion [in case of group therapy]). For the sake of space, other potential predictors (e.g., personality traits) were not addressed in our review.

Results

Medline search identified 392 articles, of which 10 met the inclusion criteria. In PsycInfo, from the 58 studies identified, 5 were included. The ISI/Web of Science database led to the identification of 679 articles, of which 4 were included. A Cochrane search resulted in 158 studies, of which 11 were selected, including 10 that had already been localized on Medline and Psyinfo, and one that appeared for the first time.

A LILACS search led to only one article, which did not meet the inclusion criteria. Unpublished articles searched on Current Controlled Trials were not found as well. Four additional articles were identified in the reference lists

of previously selected articles. These combined search strategies led to a total of 24 selected articles. Most studies (n = 19) reported independent predictors of response using regression analysis, while the remaining ones[12-16] used univariate analysis.

Clinical characteristics

Studies describing the impact of different clinical characteristics (including age of onset, duration, symptom severity, subtype, and family history) on SAD treatment outcomes are listed in Table 1.

Age of onset

The impact of the age of SAD onset on treatment outcome was investigated in four studies. In one of them, no impact of age of onset on the response of 57 patients treated with group CBT was found.[17] In the second trial with 204 patients treated with sertraline or placebo, patients with late-onset (≥ 19 years) generalized SAD tended to have a better response to treatment with sertraline than those with early-onset generalized SAD.[18] Accordingly, in two additional studies; e.g., one including 102 patients with generalized SAD submitted to individual or group CBT[19] and the other including 80 SAD patients treated with individual and group cognitive or interpersonal therapy,[20] early (childhood) onset emerged as a predictor of poor response to treatment.

Duration of illness

Duration of SAD was not associated with a worse therapeutic outcome in four studies, including trials with moclobemide,[21] brofaromine and fluvoxamine,[22] paroxetine,[23] and group CBT.[17] Only one placebo-controlled trial found baseline duration of generalized SAD to be a significant predictor of poor response to treatment (in this case, sertraline).[24] In this study, none of the independent variables were significant as predictors of response to placebo.

Severity

The impact of SAD severity on treatment outcome was analyzed in three studies.[22,23,25] Increased severity of symptoms predicted worse response in 30 patients treated with brofaromine or fluvoxamine (with higher Symptom Checklist-90 interpersonal sensitivity scores and heart rate predicting 92% of non-responders),[22] and in 76 patients submitted

Table 1 Studies describing the impact of different clinical characteristics on treatment outcomes of social anxiety disorder

Study	Design	Intervention	N	Clinical characteristics	Influence on response
Brown et al.[28]	Controlled clinical trial	CBTG ESTG PHE PLO	NI	Generalized subtype	Negative
Slaap et al.[22]	Controlled clinical trial	BROFARO FLUVO	15 15	Short duration of SAD Greater Severity Generalized subtype Family history	Neutral Negative Neutral Negative
Turner et al.[12]	Controlled clinical trial	Flooding ATE PLO	NI	Generalized subtype	Neutral
Versiani et al.[21]	Naturalistic	MCLO	93	Short duration of SAD Generalized subtype	Neutral Negative
Scholing et al.[25]	Open trial	CBTG and/or individual	73	Greater Severity	Negative
Van Ameringen et al.[24]	Controlled clinical trial	SERT PLO	NI	Short duration of SAD	Positive
Stein et al.[23]	Controlled clinical trial	PARO PLO	491 329	Short duration of SAD Greater Severity	Neutral Neutral
Van Ameringen et al.[18]	Controlled clinical trial	SERT PLO	NI	Early onset of SAD	Negative
Lincoln et al.[26]	Naturalistic	CBTG	287	Generalized subtype	Negative
Chen et al.[17]	Open trial	CBTG	57	Early onset of SAD Short duration of SAD Generalized subtype	Neutral Neutral Neutral
Dalrymple et al.[19]	Open trial	CBTG and/or individual	102	Early onset of SAD	Negative
Marom et al.[27]	Naturalistic trial	CBTG	219	Generalized subtype	Negative
Borge et al.[20]	Controlled clinical trial	COTG and individual IT individual and group	40 40	Early onset of SAD	Negative

ATE: Atenolol; BROFARO: Brofaromine; CBTG: Cognitive Behavior Therapy Group; ESTG: Educational Supportive Therapy group; FLUVO: Fluvoxamine; IT: Interpersonal Therapy; MCLO: Moclobemide; NI: Not Informed; PARO: Paroxetina; PLO: Placebo; SAD: Social Anxiety Disorder; SERT: Sertraline; PHE: Phenelzine; COTG: Cognitive Therapy In Group.

to individual or group CBT[25] (with greater impairment or Global Clinician rating of Social Phobia severity predicting a slight proportion [4-8%] of treatment outcome). In the third study,[23] the severity levels (according to the baseline scores on the Liebowitz Social Anxiety Scale) of 829 SAD patients did not predict differences in outcome after treatment with paroxetine or placebo.

Subtype

No differences in terms of response to treatment was found between generalized and non generalized SAD patients in three clinical trials: one article with brofaromine or fluvoxamine,[22] one involving group CBT,[17] and one comparing atenolol to exposure psychotherapy (EXP) with flooding.[12] However, in other four studies using group CBT,[26,27] moclobemide,[21] or a set of interventions (including group CBT, educational support therapy, phenelzine, and placebo pill),[28] generalized SAD patients exhibited worse response to treatment compared to non-generalized patients in the post-test. In one of these studies, the generalized subtype was characterized by a trend towards worse short-term response in group CBT, although this difference reached statistical significance after one-year of follow up.[26]

Family history

Only one article suggested that the presence of a family history of SAD predicts worse response to treatment with brofaromine or fluvoxamine in SAD patients.[22] Therefore, the relationship between family history and response to treatment should be considered relatively fragile at this moment.

Comorbidities

Studies describing the impact of different psychiatric comorbidities (including personality, anxiety and mood disorders, and substance abuse) on SAD treatment outcomes are depicted in Table 2.

Personality disorders

Eleven trials assessed the influence of avoidant personality disorder (PD) on treatment response of patients with SAD. In five of these studies, patients with SAD and avoidant PD displayed worse outcome after treatment (including moclobemide,[21] atenolol or EXP with flooding,[12] group CBT,[29] EXP,[13] and moclobemide or individual cognitive therapy)[30] compared to patients with SAD without avoidant PD. Nonetheless, in one of these studies,[30] this difference did not remain significant after 15 months of treatment.

In five additional articles, the comorbidity of SAD with avoidant PD did not predict differences in response to interventions with sertraline,[18] EXP,[14] individual or group CBT,[25] individual or group cognitive therapy, individual or group interpersonal therapy[20] and a set of interventions (including group CBT, educational support therapy, phenelzine and placebo pill).[28] Finally, in contrast to the results described above, a study with 295 generalized SAD patients reported that patients with avoidant personality disorder (APD) had exhibited greater changes in SAD early in treatment than those without APD.[15]

Two studies reported that other types of PD (e.g., obsessive-compulsive and dependent) had no influence in response of 84 SAD patients who received either atenolol or EXP with flooding treatment,[12] and 93 SAD patients who were treated with moclobemide.[21] Histrionic PD co-morbidity with SAD, on the other hand, had a negative impact on response to treatment with atenolol or EXP with flooding.[12]

Anxiety disorders

A study with 52 SAD patients treated with group CBT plus video feedback of exposures revealed that co-morbidity with any anxiety disorder was associated with a worse therapeutic outcome.[31] Similarly, the presence of simple phobia had a negative impact on treatment response of 84 patients with SAD treated with atenolol or EXP with flooding.[12] Finally, generalized anxiety disorder (GAD) was associated with worse treatment response of 93 patients with SAD treated with moclobemide,[21] and of 84 patients treated with atenolol or EXP with flooding.[12]

Mood disorders

The influence of comorbid mood disorders on the treatment outcome of patients with SAD was associated with mixed results. For instance, in at least three studies,[25,26,29] the presence of major depression was associated with worse response of SAD patients treated with group and individual CBT[25] or group CBT,[26,29] respectively. Similarly, comorbid dysthymia had a negative impact on response to treatment with atenolol or EXP with flooding[12] and with moclobemide.[21] However, in one of these articles, the negative influence of depression on the therapeutic outcome was not maintained in the 18-month follow up.[25]

In two trials[27,32] no difference was found in the post-test of SAD patients with comorbid depression treated with group CBT. However, in one of these studies,[27] patients with SAD and comorbid depression displayed an increased relapse rate after one year. On the other hand, the presence of previous episode of depression in patients treated with moclobemide did not predict any difference in treatment outcome.[21]

Alcohol abuse

In one of the studies described above,[21] the occurrence of alcohol abuse co-morbidity was by far the strongest predictor of worse therapeutic outcome in 93 SAD patients treated with moclobemide.

Sociodemographic characteristics

Studies describing the impact of different sociodemographic features (including age, gender, educational level, and marital status) on SAD treatment outcomes are listed in Table 3.

Age

According to several aforementioned studies, the patient's age did not predict the therapeutic response of SAD patients to sertraline,[18] moclobemide,[21] paroxetine,[23] brofaromine or fluvoxamine,[22] group CBT,[17,32] and group or individual CBT.[19]

Gender

Similarly, no impact of gender on treatment response of SAD patients was found in most trials reviewed above, including studies that used group CBT,[17] group or individual CBT,[19]

Table 2 Studies describing the impact of different comorbidities on treatment outcomes of social anxiety disorder

Study	Design	Intervention	N	Comorbidities	Influence on response
Brown et al.[28]	Controlled clinical trial	CBTG	NI	Avoidant PD	Neutral
		ESTG			
		PHE			
		PLO			
Turner et al.[12]	Controlled clinical trial	Flooding	NI	Avoidant PD	Negative
		ATE		Obsessive Compulsive PD	Neutral
		PLO		Dependent PD	Neutral
				Histrionic PD	Negative
				GAD	Negative
				Simple Phobia	Negative
				Dysthimia	Negative
Versiani et al.[21]	Naturalistic trial	MCLO	93	Avoidant PD	Negative
				Obsessive Compulsive PD	Neutral
				Dependent PD	Neutral
				GAD	Negative
				Dysthimia	Negative
				Previous Depression	Neutral
				Alcohol Abuse	Negative
Chambless et al.[29]	Open trial	CBTG	62	Avoidant PD	Negative
				Depression	Negative
Feske et al.[13]	Naturalistic trial	EXP	48	Avoidant PD	Negative
Van Velzen et al.[14]	Open trial	EXP	61	Avoidant PD	Neutral
Scholing et al.[25]	Open trial	CBTG and/or individual	73	Avoidant PD	Neutral
				Depression	Negative
Oosterbaan et al.[30]	Controlled clinical trial	COT individual	28	Avoidant PD	Negative
		MCLO	27		
		PLO	27		
Van Ameringen et al.[18]	Controlled clinical trial	SERT	NI	Avoidant PD	Neutral
		PLO			
Lincoln et al.[26]	Naturalistic trial	CBTG	287	Depression	Negative
Huppert et al.[15]	Controlled clinical trial	FLU	NI	Avoidant PD	Positive
		CBTG			
		FLU+CBTG			
		CBTG+PLO			
Marom et al.[27]	Naturalistic trial	CBTG	219	Depression	Neutral
Alfano et al.[32]	Controlled clinical trial	CBTG	NI	Depression	Neutral
		TNE			
Chen et al.[31]	Open trial	CBTG+VF	52	Anxiety Disorders	Negative
Borge et al.[20]	Controlled clinical trial	COTG and individual	40	Avoidant PD	Neutral
		IT individual and group	40		

ATE: Atenolol; CBTG: Cognitive Behavior Therapy Group; EXP: exposure psychotherapy; COT: Cognitive Therapy; COTG: Cognitive Therapy In Group; ESTG: Educational Supportive Therapy Group; FLU: Fluoxetine; GAD: Generalized Anxiety Disorder; IT: Interpersonal Therapy; MCLO: Moclobemida; NI: Not Informed; PD: Personality Disorder; PHE: Phenelzine; PLO: Placebo; SERT: Sertraline; TNE: Treatment not Specific; VF: Video feedback.

paroxetine,[23] moclobemide,[21] brofaromine or fluvoxamine.[22] In one naturalistic study, no difference regarding treatment outcomes was found when CGI scores of both genders were compared at the end-point after treatment with SSRIs and/or benzodiazepines.[16] However, male gender was associated with worse response of SAD patients to sertraline or placebo[18] and to group plus video feedback CBT.[31]

Educational/Marital status

Educational and marital status had no impact on the response of 57 SAD patients in group CBT in one study,[17] and of 102 patients treated with group or individual CBT in another one.[19]

Treatment characteristics

Studies describing the impact of different therapeutic features on the outcome of SAD (including history, duration and expectancy of current treatment, and group cohesion [in case of group therapy]) are listed in Table 4.

Previous treatment

In three aforementioned studies, previous treatment (either pharmacotherapy or psychotherapy) had no impact on the response of SAD patients to moclobemide,[21] brofaromine and fluvoxamine,[22] and in group CBT.[17]

Table 3 Studies describing the impact of different sociodemographic features the treatment outcomes of social anxiety disorder

Study	Design	Intervention	N	Sociodemographic characteristics	Effect on response
Slaap et al.[22]	Controlled clinical Trial	BROFARO FLUVO	15 15	Age Gender	Neutral Neutral
Versiani et al.[21]	Naturalistic trial	MCLO	93	Age Gender	Neutral Neutral
Stein et al.[23]	Controlled clinical Trial	PARO PLO	491 329	Age Gender	Neutral Neutral
Van Ameringen et al.[18]	Controlled clinical Trial	SERT PLO	NI	Age Female Gender	Neutral Positive
Chen et al.[17]	Open trial	CBTG	234	Age Gender Educational Marital status	Neutral Neutral Neutral Neutral
Dalrymple et al.[19]	Open trial	CBTG and/or individual	57	Age Gender Educational Marital status	Neutral Neutral Neutral Neutral
Menezes et al.[39]	Naturalistic trial	SSRI and/or BENZO	34	Gender	Neutral
Alfano et al.[32]	Controlled clinical Trial	CBTG TNS	NI	Age	Neutral
Chen et al.[31]	Open trial	CBTG+VF	80	Male Gender	Negative

BENZO: Benzodiazepine; BROFARO: Brofaromine; CBTG: Cognitive Behavior Therapy Group; FLUVO: Fluvoxamine; MCLO: Moclobemide; NI: Not Informed; PARO: Paroxetine; PLO: Placebo; SERT: Sertraline; SSRI: selective serotonin reuptake inhibitors; TNS: Treatment not Specific; VF: Video feedback.

Table 4 Studies describing the impact of different treatment characteristics on treatment outcomes of social anxiety disorder

Study	Design	Intervention	N	Treatment characteristics	Effect on Response
Slaap et al.[22]	Controlled clinical trial	BROFARO FLUVO	15 15	Previous treatment	Neutral
Versiani et al.[21]	Naturalistic	MCLO	93	Previous treatment	Neutral
Chambless et al.[29]	Open trial	CBTG	62	Low expectancy regarding treatment effectiveness	Negative
Stein et al.[23]	Controlled clinical trial	PARO PLO	491 329	Short duration of treatment	Negative
Erwin et al.[33]	Controlled clinical trial	CBTG	234	Low expectancy regarding treatment effectiveness	Negative
Chen et al.[17]	Open trial	CBTG	57	Previous treatment Short duration of treatment	Neutral Neutral
Taube-Schiff et al.[35]	Open trial	CBTG	34	Group cohesion	Positive
Delsignore et al.[34]	Open trial	CBTG	49	Low expectancy regarding the therapist role	Neutral
Borge et al.[20]	Controlled clinical trial	COTG and individual IT individual and group	40 40	Low expectancy regarding treatment effectiveness	Negative

BROFARO: Brofaromine; CBTG: Cognitive Behavior Therapy Group; COTG: Cognitive Therapy in Group; FLUVO: Fluvoxamine; IT: Interpersonal Therapy; MCLO: Moclobemide; PARO: Paroxetine, PLO: Placebo.

Duration of treatment

In a study with 829 SAD patients,[23] treatment with paroxetine for 12 weeks was associated with increased response compared to treatment for only 8 weeks. Conversely, another study that used group CBT with a protocol of 11-20 sessions found that the number of treatment sessions did not predict response of 57 patients with SAD.[17]

Expectancy regarding treatment effectiveness

Low expectancy regarding treatment effectiveness and reduced confidence concerning treatment predicted worse response in studies that comprised 62[29] and 234 patients[33] with SAD assigned to group CBT, and 80 patients with SAD assigned to cognitive therapy or interpersonal therapy.[20]

Expectancy regarding the therapist role

In one study, the expectations of 49 SAD patients about the role of the therapist in their treatment did not have any influence on their response in group CBT in the short-term.[34] However, in the three-month follow up, patients who attributed greater responsibility for change to their therapists had worse therapeutic response.

Group cohesion in group CBT

The perception of group cohesion of individuals attending group CBT and its impact on treatment outcome were studied in 34 patients with SAD.[35] In this study, increases in group cohesion ratings over the course of treatment significantly predicted therapeutic response.

Discussion

The present work aimed at reviewing the overall profile of individuals with SAD who respond and not respond to standard treatment, with the ultimate purpose of contributing to future research on the development of more effective interventions.[36] We found that early onset, increased duration and severity of illness, presence of generalized subtype, positive family history of SAD, and male gender were all predictors of worse response to treatment in at least one study. Among these, early onset and severity of illness were the most replicated findings.

In terms of co-morbidity, major depression, dysthymia, generalized anxiety disorder, simple phobia, avoidant and histrionic PD, and alcohol abuse predicted negative responses to treatment in at least one trial. Among these conditions, anxiety disorders emerged as a predictor of poor therapeutic response in more than one study.

Finally, regarding treatment characteristics, low expectancy regarding treatment efficacy, high expectancy regarding the role of the therapist in treatment, and decreased group cohesion (in case of group cognitive behavioral treatment) were associated with worse therapeutic responses. Among the later, low expectancy regarding treatment efficacy was the most replicated finding.

Importantly, we found a handful of studies suggesting the association between early onset of SAD and poor response to treatment. While this finding provides some support to the view that the early-onset of SAD represents a valid subtype of SAD, with greater psychiatric comorbidity, higher levels of incapacitation, and worse prognosis,[19] there is still controversy on this subtype definition, as different studies used different age criteria for "early" or "late" onset. Previous reviews suggested that the unwelcome influence of both age at onset and severity of symptoms on treatment outcome holds true for other anxiety disorders.[37]

Furthermore, we also found some evidence that greater severity of social anxiety symptoms predicts decreased efficacy in the response of patients with SAD to different types of treatment (both pharmacotherapy and psychotherapy). This finding is particularly relevant when one considers that all studies reporting them focused their analysis on patients who completed treatment (and not on an intention-to-treat sample), thus suggesting that decreased treatment efficacy cannot be ascribed to the tendency that patients with more severe symptoms have to discontinue treatment more frequently.

Data supporting an effect of comorbid psychiatric disorders in SAD treatment response are mixed, particularly for depression. It is difficult to explain these findings because, while major depression and dysthymia could perpetuate and strengthen SAD patients negative beliefs about themselves, some of the SAD treatments (including SSRIs and cognitive re-structuring) may effectively treat some forms of depression, particularly those that are secondary. Accordingly, it is interesting to note that many of the comorbid conditions found to predict worse response of SAD to treatment (e.g., alcohol abuse,[21] specific phobia,[12] histrionic PD[12]) are not clearly responsive to treatment (e.g., antidepressants) themselves. It is worth noting that findings regarding the role of several comorbid conditions were assessed in only one study, which restrains us to consider these results as conclusive.

Finally, we also found that low expectancy regarding treatment effectiveness,[29] which might be related to comorbid depression,[38] was associated with worse response to treatment in different studies. Clearly, patients with SAD might believe that the treatment will not work in their case, that they will not be able to follow the therapeutic steps, or that another person will be totally responsible for his improvement. These cognitive distortions should be identified and managed early in the treatment.

Conversely, lower expectations about the role of the therapist were related to better treatment outcomes, particularly in the follow-up sessions.[34] Likewise, in one study, increased feelings of group cohesion between participants and therapist in group CBT was associated with better outcomes.[35] It would be interesting to test whether add-on strategies (aimed at increasing treatment expectancy, attributing responsibility for change to patients, and stressing the importance of group cohesion in group CBT) increase the effectiveness of treatment provided to patients with SAD.

Indeed, our review has a number of limitations. First, several sociodemographic and clinical variables associated with worse outcomes (e.g., previous treatment, dependent and obsessive compulsive PD comorbidity) had their relationship with outcome assessed by a very small number of studies. Second, as there is no current consensus on what should be the optimal criteria for treatment response in SAD, one should examine our results with caution.[12,25,29] It has been argued that the concept of response and remission in SAD must be multidimensional,[39] embracing the

reappraisal of the diagnosis,[40] social anxiety symptoms, and comorbid conditions.[36] Of note, none of the reviewed studies adopted such criteria. Third, there is a clear methodological heterogeneity among selected studies, including study design,[15,17,23] type of intervention,[21,25-27,29,32] and duration of treatment (varying from five or seven days[26] to two years).[21] Accordingly, one must consider that, regardless of treatment, longer follow-ups are usually associated with substantial reduction in anxiety symptoms, a phenomenon that can be explained by the tendency of SAD individuals to orientate their life choices so that anxiogenic situations become less prominent.[41]

Conclusion

The recent increase in the number of studies in the field of response and resistance in SAD is complicated by the difficulty of replicating and comparing these studies. Even when they are conducted with similar patients, treatment, and methods, a considerable variety of treatment response and resistance definitions is used.[25] As a consequence, the current sociodemographic and clinical profile of SAD patients who respond to treatment remains elusive, as it does not point to a very clear direction.

To date, early onset, severity of illness, comorbid anxiety disorders (particularly generalized anxiety disorder and simple phobia), and low expectancy regarding treatment efficacy emerged as potential correlates and/or predictors of low therapeutic response. Clearly, the field of treatment response needs conceptual and methodological redefinitions if it aims to lead to more solid results. A better knowledge about the profile of the significant proportion of SAD patients who remain resistant to treatment is important for discussing the emergence of novel therapeutic alternatives, both in terms of psychotherapy and pharmacotherapy.

Disclosure

Sara C. Mululo
Employment: *Instituto de Psiquiatria, Universidade Federal do Rio de Janeiro (IPUB/UFRJ) Rio de Janeiro, Brazil.*
Gabriela B de Menezes
Employment: *Instituto de Psiquiatria, Universidade Federal do Rio de Janeiro (IPUB/UFRJ) Rio de Janeiro, Brazil.*
Paula Vigne
Employment: *Instituto de Psiquiatria, Universidade Federal do Rio de Janeiro (IPUB/UFRJ) Rio de Janeiro, Brazil.*
Leonardo F. Fontenelle
Employment: *Instituto de Psiquiatria, Universidade Federal do Rio de Janeiro (IPUB/UFRJ); Departmento de Psiquiatria e Saúde Mental, Instituto de Saúde da Comunidade, Universidade Federal Fluminense (MSM/UFF); Instituto D'Or de Pesquisa e Ensino (IDOR), Rio de Janeiro, Brazil.* **Research grant:** *Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq), Fundação Carlos Chagas Filho de Amparo à Pesquisa do Estado do Rio de Janeiro (FAPERJ), Brazil.* **Other research grant or medical continuous education:** *Instituto D'Or de Pesquisa e Ensino (IDOR), Rio de Janeiro, Brazil.* **Speaker's honorária:** *Lundbeck.*
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** Significant
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