Published in final edited form as:

Psychiatry Res. 2015 July 30; 228(1): 65–71. doi:10.1016/j.psychres.2015.04.014.

Clinical Presentation and Pharmacotherapy Response in Social Anxiety Disorder: The Effect of Etiological Beliefs

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Abstract

Therapies for social anxiety disorder (SAD) leave many patients symptomatic at the end of treatment and little is known about predictors of treatment response. This study investigated the predictive relationship of patients' etiological attributions to initial clinical features and response to pharmacotherapy.

Methods—One hundred thirty-seven individuals seeking treatment for SAD received 12 weeks of open treatment with paroxetine. Participants completed the Attributions for the Etiology of

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Contributors

Jonah N. Cohen: I was the primary writer of the manuscript and central in idea generation.

Carrie M. Potter: I contributed to the manuscript by running the statistics and writing the results.

Deborah A. Drabick: I contributed by helping to analyze the results and overall statistical consultation.

Carlos Blanco: I contributed to the generation of the study idea, study design, and reviewed the manuscript and provided comment and input.

Frank R. Schneier: I contributed to the generation of the study idea, study design, and reviewed the manuscript and provided comment and input.

Michael R. Liebowitz: I contributed to the generation of the study idea, study design, and reviewed the manuscript and provided comment and input.

Richard G. Heimberg: I contributed to the generation of the study idea, study design, manuscript writing, and editorial review.

Conflicts of Interest

Mr. Cohen, Ms. Potter, Dr. Drabick, Dr. Blanco, and Dr. Heimberg have no financial disclosures nor conflicts of interest to report.

Dr. Liebowitz reports the following: (1) equity ownership: Pherin Pharmaceutical, Liebowitz Social Anxiety Scale, ChiMatrix LLC (terminated 2011); (2) consulting: Astra Zeneca, Pfizer, Takeda, Pherin, Otsuka, Eisai, Avera, Tikvah, Wyeth, Lilly, Concert, J and J, Forest, Sunovion; (3) licensing software: GSK, Pfizer, Avera, Tikvah, Endo, Lilly, Indevus, Servier; (4) speaking or presentation fees: Wyeth, Pfizer, Pherin, Forest; (5) recent or current clinical trial contracts: Pfizer, GSK, Astra Zeneca, Forest, Tikvah, Avera, Eli Lilly, Novartis, Sepracor, Horizon, Johnson and Johnson, Allergan, PGX Health, Abbott, Jazz, MAP, Takeda, Wyeth, Cephalon, Indevus, Endo, Ortho-McNeil, Grunenthal, Purdue Pharma, Lundbeck, Otsuka, Naurex, Shionogi, Teva, Cerecor, MSI, Pherin, Alkermes, Sunovion, Roche Genentech; and (6) recent or current investigator-initiated trial support: Pfizer, Forest.

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Social Anxiety Scale at baseline in addition to measures of social anxiety and depression at baseline and over the course of treatment.

Results—A latent class analysis suggested four profiles of etiological beliefs about one's SAD that may be characterized as: *Familial Factors*, *Need to be Liked*, *Bad Social Experiences*, and *Diffuse Beliefs*. Patients in the more psychosocially-driven classes, *Need to be Liked* and *Bad Social Experiences*, had the most severe social anxiety and depression at baseline. Patients in the *Familial Factors* class, who attributed their SAD to genetic, biological, and early life experiences, had the most rapid response to paroxetine.

Conclusions—These results highlight the effect of biological and genetically-oriented etiological beliefs on pharmacological intervention, have implications for person-specific treatment selection, and identify potential points of intervention to augment treatment response.

Keywords

paroxetine; social anxiety disorder; social phobia; treatment-matching; latent class analysis; personalized medicine

1. Introduction

Social anxiety disorder (SAD) is highly prevalent, with up to 13% of the U.S. population experiencing SAD at some point during their lives (Ruscio et al., 2008). SAD is characterized by a marked and/or persistent fear of one or more social situations and is associated with significant functional impairment (Aderka et al., 2012; American Psychiatric Association, 2013; Schneier et al., 1994). Individuals with SAD have higher rates of alcohol and drug dependence, depression, suicide, and use of medical resources, as well as diminished vocational and educational attainment (Acarturk et al., 2009; Katzelnick et al., 2001; Van Ameringen et al., 2003).

Although there are well-validated treatments for SAD (Heimberg and Magee, 2014; Schneier et al., 2014; Wong et al., 2012), response rates for even the best empirically supported treatments suggest that many treated patients remain symptomatic. For instance, in one study, 35% of patients receiving the monoamine oxidase inhibitor phenelzine and 42% of patients receiving group cognitive behavioral therapy (CBT) were classified as non-responders (Heimberg et al., 1998). Moreover, trials of serotonin-norepinephrine reuptake inhibitors and selective serotonin reuptake inhibitors (SSRIs) suggest similar rates of non-response. For example, non-response rates ranged from 41% with venlafaxine (Liebowitz et al., 2005) to 45% with paroxetine (Stein et al., 1998) to 47% with sertraline (Van Ameringen et al., 2001).

In an effort to augment treatment response for mental disorders, the National Institute of Mental Health (NIMH) called for the study of elements of personalized mental health care in its Strategic Plan (NIMH, 2008). Personalized medicine seeks to identify variables related to both patient and treatment modality that optimize treatment outcomes. The scope of possible avenues of research is wide and include pharmacogenetics, pharmacotherapy dosing schedules, and predictors of treatment outcome that inform patient-treatment matching (Arch and Ayers, 2013).

Only a handful of studies have examined variables that impact treatment outcome in SAD. Having an expectation of benefiting from group CBT predicts enhanced treatment response (Chambless et al., 1997; Safren et al., 1997). Among patients with SAD, certain cognitive characteristics (e.g., cognitive reappraisal self-efficacy, negative cognitive appraisal) may mediate response to CBT (Goldin et al., 2012; Hofmann, 2000). However, only a few published studies have examined predictors of response to pharmacotherapy for SAD (Bruce et al., 2012). To our knowledge, there are no published studies investigating whether and to what extent patients' cognitive characteristics impact response to pharmacotherapy for SAD, information potentially relevant to tailoring to personalizing therapeutic intervention.

1.1 Attributions as a potential predictor of pharmacotherapy response in SAD

Etiological attributions (i.e., causal explanations of the etiology of one's disorder) are one type of cognitive characteristic that may impact treatment response. Causal attributions have been associated with both the etiology and maintenance of SAD (Hope et al., 1989). For example, individuals with SAD tend to attribute positive outcomes to external factors and negative outcomes to internal factors. This attributional bias strengthens as social anxiety intensifies (Coles et al., 2001). Though this literature is not specifically focused on attributions about etiology, attributions about causality play an important role in SAD.

In the context of psychopathology more generally, the attributions that individuals make about their disorder may influence the steps they take in the pursuit of treatment (Roth and Eng, 2002). Although there is currently no research that investigates whether etiological beliefs directly influence treatment response, research suggests that etiological beliefs influence *perceived* efficacy of treatment (Furnham, 1995). Thus, matching treatments to patients' etiological beliefs may lead to better treatment response based on expectancy effects, which as noted above, have been associated with response to CBT for SAD (Chambless et al., 1997; Safren et al., 1997). Thus, given the relevance of causal attributions to SAD, investigating the clinical effect of etiological attributions is warranted.

1.2 Present study

Aligned with the NIMH's call for the personalization of mental health care, we sought to identify baseline patient characteristics differentially related to clinical presentation and response to pharmacotherapy, information which may inform ways to augment treatment response. This work has the potential to add to an emerging body of literature on personalization of treatment for SAD (e.g., Craske et al., 2014) and to extend this research by investigating personalization within the context of etiological attributions and pharmacotherapy, two previously unexplored domains.

Thus, we examined whether individuals' attributions about the etiology of their SAD are related to their the initial severity of their symptoms and predictive of their response to pharmacotherapy with paroxetine, an SSRI demonstrated to be efficacious in the treatment of SAD (Allgulander, 1999; Baldwin et al., 1999; Stein et al., 1998). We hypothesized that individuals would differ both in the types of attributions that they endorse (e.g., genetics, family environment, stressful social experiences), as well as frequency of attributions (e.g., moderate vs. high levels of genetic attributions). We used latent class analysis (LCA) to

identify distinct profiles of SAD-related etiological attributions. We expected that classes of individuals whose profiles are characterized by genetic/biological attributions would be associated with a more severe clinical presentation at baseline. Additionally, we expected classes of individuals with profiles emphasizing genetic/biological attributions to exhibit better response to paroxetine, as these types of etiological attributions would best match the treatment modality and, in line with previous research (Furnham, 1995), lead to higher expectancy of treatment efficacy.

2. Methods

2.1 Participants

Data for this study were obtained from treatment-seeking outpatients with a principal diagnosis of generalized SAD. Individuals were recruited to participate in an open trial of the treatment of SAD with paroxetine, followed by randomization to augmentation with CBT or continuation of paroxetine. Due to a substantially smaller sample size after randomization, only the open treatment portion of the trial is considered here. Forty-six patients from the Adult Anxiety Clinic of Temple University (AACT) and 92 patients from the Anxiety Disorders Clinic of the New York State Psychiatric Institute (NYSPI) participated. One patient with missing data on the measure of etiological attributions was excluded from analyses; therefore, the analysis sample was 137. Individuals were excluded from this study based on various criteria including current psychotic symptoms, current or past diagnosis of bipolar disorder or major depressive disorder, significant suicidal ideation, past adequate trials of an SSRI or CBT for SAD, unwillingness to discontinue other psychotropic medications, current psychotherapeutic intervention or inability to give consent (Heimberg et al., unpublished results).

2.2 Procedure

Individuals who met inclusion criteria after preliminary screening underwent a structured diagnostic interview. Those patients meeting DSM-IV criteria for generalized SAD and not meeting any exclusionary criteria underwent a comprehensive medical evaluation and then consented to treatment. All procedures were approved by relevant Institutional Review Boards. Patients met with a psychiatrist weekly for the first 6 weeks during titration and then every other week, for a total of 9 visits over 12 weeks. Patients started at 10mg of paroxetine per day and were increased to a therapeutic level on an individual basis (from 20–60mg). The psychiatrist offered general encouragement and support while monitoring clinical progress and medication effects. The psychiatrist instructed patients to expose themselves to feared situations to help overcome avoidance behaviors and explained that the role of paroxetine was to make such exposure easier. However, systematic exposure instructions were not offered. Psychiatrists performed pill counts and asked patients whether they were taking their medication as prescribed. Assessments were conducted at Weeks 0 (baseline), 4, 8, and 12 (end of open treatment).

2.3 Measures

2.3.1. Demographic Characteristics—Demographic characteristics, including age, sex, race, ethnicity, employment status, marital status, and religion, were assessed at baseline.

2.3.2. Diagnostics—At the NYSPI, individuals were administered the *Structured Clinical Interview for DSM-IV, Patient Edition with Psychotic Screen* (SCID-I/P; First et al., 2002) at baseline. At the AACT, individuals were administered the *Anxiety Disorders Interview Schedule for the DSM-IV: Lifetime Version* (ADIS-IV-L; Di Nardo et al., 1994). At NYSPI, because the reliability of SAD is lower when based on the SCID-I/P (Zanarini and Frzenburg, 2001; Zanarini et al., 2000) than on the ADIS-IV-L, the social phobia module of the ADIS was also administered.

2.3.3. Social anxiety—The Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987) is a 24-item clinician-administered scale that assesses social anxiety and avoidance in performance and social interaction situations. The LSAS has been successfully used in studies that assess change in response to psychotropic medication (Heimberg et al., 1998) and has strong convergent validity (Heimberg et al., 1999). The LSAS was administered to patients at Weeks 0, 4, 8, and 12, and the total score was used to assess social anxiety severity at each time point. Cronbach's α for the LSAS ranged from .93 to .96 across assessment points in the current study.

2.3.4. Depression—Participants' depressive symptoms were assessed with the Beck Depression Inventory–II (BDI-II; 36), a 21-item self-report measure that assesses symptoms of depression on a scale that ranges from 0 (e.g., “I am not discouraged about my future”) to 3 (e.g., “I feel my future is hopeless and will only get worse”). The BDI-II has demonstrated strong test-retest reliability and convergent and divergent validity (Beck et al., 1996). The BDI-II was only administered at Week 0 ($\alpha = .93$) and Week 12 ($\alpha = .92$).

2.3.5. Etiological attributions of social anxiety—The Attributions for the Etiology of Social Anxiety Scale (AESAS)¹ was created by a panel of experts in SAD for the purposes of this study. Although other measures of etiological beliefs exist for various kinds of psychopathology, no known measure existed for SAD prior to the creation of this scale. Thus we elected to generate our own SAD-specific scale which resembled early measures of the etiology of specific phobia (e.g., McNally and Steketee, 1985; Öst and Hugdahl, 1981). The AESAS attempted to assess two causes of social anxiety: a genetic/biological dimension and a psychosocial dimension, but the final item content was that generated by the panel. The final version of the AESAS consisted of 17 items rated on a 5-point Likert-type scale ranging from 0 (*not at all important as a cause of my social anxiety*) to 4 (*one of the most important as a cause of my social anxiety*). Items assess a range of attributions from “something inherited from my parents/family genetics” and “something wrong with the way my body works that makes me a nervous person” to a “general tendency to be sensitive to criticism” (several AESAS items appear in Figure 1). Patients completed the AESAS at Week 0 ($\alpha = .84$).

2.4 Statistical analyses

All statistical analyses were performed with MPlus Version 7.11 (Muthén and Muthén, 1998–2014) and SPSS Version 21.0 (International Business Machines Corporation, 2012).

¹The AESAS is available upon request from the corresponding author.

LCA was used to identify groups of patients based on etiological attributions of SAD. As opposed to *variable*-centered approaches (e.g., factor analysis, structural equation modeling), LCA considers relations among *individuals* to classify them into homogeneous “classes” that differ in terms of the frequency, severity, and/or quality of their etiological attributions (Muthén and Muthén, 2000). LCA does not require pre-determined assumptions about the structure of the etiological attribution profiles (Beauchaine, 2003). In the present LCA, the 17 AESA items, treated as continuous variables, were entered as predictors of class membership. MPlus uses Full Information Maximum Likelihood estimation to handle missing data, and all participants were included in all analyses.

Unlike other person-centered techniques such as cluster analysis, which is “structure seeking” and identifies the number of classes specified by the researcher, LCA uses a step-wise procedure and a variety of fit indices to determine whether the addition of classes improves the fit to the data (Beauchaine, 2003). Thus, there are no “gold standard” guidelines regarding the number of participants and power for a proposed LCA. Instead, a variety of statistical indices and conceptual considerations are taken into account to determine which model best fits the data (Nylund et al., 2007a, 2007b). The LCA procedure starts with a one-class (unconditional) model, and the number of classes is increased until there was no additional improvements to model fit (Nylund et al., 2007a, Nylund et al., 2007b). In terms of fit indices, first, the Akaike Information Criterion (AIC; Akaike, 1987), Bayesian Information Criterion (BIC; Schwartz, 1978), and sample-size adjusted BIC (ABIC; Sclove, 1987) are examined. The model that produces the smallest values on these indices is considered to have the best fit. Second, the Bootstrap Likelihood Ratio Test (BLRT) is examined to determine whether a model with k classes significantly improved model fit over a model with $k - 1$ classes (Nylund et al., 2007a, 2007b). Third, models are examined to determine if they were theoretically sound and clinically meaningful. Fourth, the size of the smallest class is considered. Any classes composed of less than 5% of the total sample would suggest over-fitting of the data and that the classes might be difficult to replicate in other samples. Monte Carlo simulation studies using a variety of sample sizes suggest that the BIC and BLRT are the most robust indicators of classes (Nylund et al., 2007a). If we are under-powered to perform these analyses or if a 1-class model provides the best fit to the data (suggesting that a person-centered approach is not necessary), fit statistics would converge to suggest that the 1-class (unconditional) model should be selected.

Following the identification of latent classes, tests of equality of means across classes were used to examine the external validity of the identified classes (Potter et al., 2014). First, we evaluated whether classes differed on demographic characteristics, social anxiety and depressive symptoms at baseline. Second, we computed residualized change scores for social anxiety and depressive symptoms to examine whether the classes differed in symptom reduction by Weeks 4, 8, or 12 of treatment. Residualized change scores were computed by regressing LSAS Week 4, 8, and 12 scores on baseline LSAS scores and BDI-II Week 12 scores on baseline BDI scores (the BDI-II was not administered at weeks 4 or 8). The test of equality of means holds class membership constant and provides χ^2 statistics for omnibus and pairwise comparisons across latent classes. Pairwise comparisons were interpreted if the omnibus tests were significant ($p < .05$).

3. Results

3.1 Characteristics of sample

The sample had a mean age of 32.82 ($SD = 11.34$), ranged from 18 to 62 years, and was 37.9% female. The mean years of education were 15.29 years. Sixty-five participants (47.4%) self-identified as Caucasian, 18 (13.1%) as Asian or Pacific Islander, 29 (21.2%) as Black, and 25 (18.2%) as “Other.” At baseline, the sample was mildly depressed (BDI-II; $M = 16.91$; $SD = 12.42$) and displayed high levels of social anxiety (LSAS; $M = 75.98$; $SD = 21.04$).

3.2 Latent class analysis

LCA models were first conducted with all 17 AESAS items as indicators of class membership. However, only the one-class model fit the data; the two-class model could not be replicated because of extreme scores or lack of variability in responses. Specifically, one class was composed of individuals who rated the items “highly perfectionistic parents” and “unstable or abusive family life” as high in importance, suggesting that lack of variance for these items was leading to difficulties replicating the model solution. Thus, we removed these items as predictors of class membership.

The final set of LCA models was conducted using the remaining 15 AESAS items as predictors of class membership. As indicated in Table 1, the lowest BIC was associated with the three-class model, and the lowest AIC and ABIC were associated with the four-class model. However, the BLRT indicated that the four-class model better fit the data than the three-class model, suggesting that the four-class model had the best overall fit. The five-class model produced two small classes, one of which ($n = 7$) was only 5% of the sample. This small class size suggests that the five-class model was unlikely to be replicated; therefore, it was not considered further.

Mean AESAS item scores across the four classes are displayed in Figure 1. Consistent with LCA conventions, we named the classes based on the frequency and quality of the profiles of responses regarding individuals’ beliefs about the etiology of their SAD. The *Familial Factors* class ($n = 12$) exhibited the greatest endorsement of family-related etiological factors, both biological (e.g., “something inherited from my parents/family genetics”) and early environmental (e.g., “very critical parents”). The *Need to be Liked* class ($n = 53$) exhibited lower endorsement of familial factors and relatively stronger endorsement of feelings of low self-esteem and a strong desire to make a good impression or to be liked by others. The *Bad Social Experiences* class ($n = 48$) exhibited greater endorsement than did all other classes of the belief that “having been teased as a child or rejected by peers” and having “one or more bad experiences in social situations that resulted in embarrassment or humiliation” played an important role in the development of their SAD. They also endorsed feelings of low self-esteem, sensitivity to criticism, and a tendency to think pessimistically. The *Diffuse Beliefs* class ($n = 24$) rated all of the AESAS items as having just below slight to moderate importance regarding the development of their SAD.

3.3 Comparisons of external validators among classes

3.3.1. Demographic characteristics—Omnibus χ^2 analyses indicated that the classes did not differ on age, sex, race, ethnicity, employment status, marital status, or religion (χ^2 s < 2.02 , $ps = .383$).

3.3.2. Baseline symptom severity—Omnibus χ^2 analyses revealed significant between-class differences for baseline social anxiety and depressive symptoms (see Table 2). Pairwise comparisons indicated that the *Need to be Liked* and *Bad Social Experiences* classes exhibited greater social anxiety and depressive symptoms at baseline than the other two classes (χ^2 s > 4.33 , $ps = .037$).

3.3.3. Treatment response—Omnibus χ^2 analyses revealed significant between-class differences for residualized social anxiety change scores² by Week 4 of treatment (see Table 2). Pairwise comparisons indicated that the *Familial Factors* class exhibited greater reductions in social anxiety by Week 4 ($M [SD] = -0.61 [0.26]$) than did the *Need to be Liked* ($M [SD] = 0.10 [0.15]$) and *Bad Social Experiences* classes ($M [SD] = 0.20 [0.16]$; see Figure 2). The *Diffuse Beliefs* class did not differ from any of the other classes. There were no between-class differences in social anxiety change at Weeks 8 or 12. Although depressive symptoms were not assessed at Weeks 4 or 8, there were no observed differences in depressive symptom change at end of treatment (Week 12). Results suggest that there are four distinct profiles of beliefs about the causes of SAD and these profiles were differentially associated with baseline severity of symptoms and initial response to pharmacotherapy.

4. Discussion

In line with the movement towards personalized medicine, this was the first study to examine the association between etiological attributions, clinical presentation, and pharmacotherapy response in SAD. It was also the first study to create a measure to assess etiological attributions for SAD.

4.1. Class profiles of etiological attributions for SAD

The LCA produced four distinct profiles of beliefs about the causes of SAD. The *Familial Factors* class endorsed beliefs that their SAD was caused by factors related to their family genetics and the family environment in which they grew up. Individuals in the *Need to be Liked* class believed that their SAD was caused by a lack of self-esteem and a desire to be liked by others. Item loadings suggest that patients in this class did not believe that these concerns were due to genetic/biological/familial factors. The *Bad Social Experiences* class endorsed the belief that negative, peer-oriented social experiences (e.g., bullying) contributed to their SAD. Finally, the *Diffuse Beliefs* class rated all AESAS items as having below moderate importance, suggesting that these individuals lacked strong opinions about

²The current report focuses on the LSAS total score as an external validator indexing social anxiety severity. Between-class comparisons of the LSAS fear and avoidance subscales revealed that the fear subscale followed the same pattern as the LSAS total score, as did the avoidance subscale, but the pattern for the avoidance subscale was only marginally significant. For a more complete account of the analyses of the LSAS subscales, please contact the corresponding author.

the etiology of their social anxiety. Against expectation, we did not find a distinct class focused solely on genetic/biological factors. Instead, we found a class characterized by both the genetic and environmental etiological factors related to one's family.

4.2. Baseline severity of symptoms

The *Need to be Liked* and *Bad Social Experiences* classes were both associated with greater baseline social anxiety and depressive symptoms than the *Diffuse Beliefs* and *Family Factors* classes. These results, which differed from our hypothesis, suggest that having etiological attributions for SAD that are centered on extra-familial psychosocial factors is associated with greater baseline severity. More than individuals with biological/genetic/familial attributions or those who do not have strong attributions, individuals with predominately psychosocial attributions presented with more severe symptoms at baseline.

Interestingly, this result differs from findings in other disorders that suggest that genetic/biological attributions are associated with beliefs that the disorder is more serious than if it were a result of psychosocial factors (Cunningham et al., 2008; Lam and Salkovskis, 2008). Although this body of research is small, the aforementioned studies investigated depression and panic disorder. This discrepancy may suggest a different effect of etiological attribution for SAD than these other disorders. More purely psychosocial etiological attributions may be related to severe pathology among individuals with SAD because negative social experiences are a substantial risk factor for developing the disorder (Rapee and Spence, 2004).

4.3. Response to pharmacotherapy

The *Familial Factors* class achieved the most immediate response to pharmacotherapy, somewhat consistent with our hypothesis regarding superior response to paroxetine for genetic/biological profiles. Notably, however, this class was not solely characterized by attributions regarding family genetics or one's biology, but also about family environmental factors, suggesting that individuals who demonstrated both genetic and environmental family-related attributions achieved the fastest response to pharmacotherapy. Individuals in this class exhibited greater reductions in severity of social anxiety symptoms by Week 4 than individuals with other profiles of etiological attributions. However, contrary to our hypothesis, the classes displayed no significant differences in social anxiety change at Weeks 8 or 12.

These differential effects of etiological attributions may be, in part, explained by patients' belief that their disease or disorder is only treatable by mechanisms analogous to which they attribute the etiology of their illness (Brickman et al., 1987; Weiner, 1993). Speculatively, patients in the *Familial Factors* class, who more strongly endorsed inherited factors as causes of their SAD than patients in the other classes, may have thought to a greater degree that the pharmacological treatment would most effectively treat a "biological" disorder (Wright et al., 2012). Indeed, attributing etiology to family-based genetic and environmental factors is consistent with an epigenetic view of mental health, perhaps represented by the "family factors" class. Therefore, the closer match between treatment modality and attribution may have increased their belief in treatment efficacy. Treatment expectancy is a

well-documented predictor in social anxiety response to CBT (Boettcher et al., 2013; Chambless et al., 1997; Hedman et al., 2012; Safren et al., 1997), although this has not been documented within the context of pharmacotherapy for SAD.

Treatment expectancy may trigger a number of candidate mechanisms which may explain the difference in initial response to paroxetine. For example, individuals may have been more optimistic or hopeful about the treatment's efficacy which, studies suggest, increases treatment response (Westra et al., 2002). Indeed, expectancy is one of the strongest triggers of the placebo effect (Rutherford and Roose, 2013). Moreover, our pattern of findings is consistent with research suggesting that placebo response is often associated with rapid clinical improvement but that such gains are less persistent (Quitkin et al., 1993, 1996). These patients, perhaps feeling optimistic or protected by the paroxetine, may have experienced less anticipatory anxiety, permitting them to expose themselves to anxiety-provoking situations earlier, leading to quicker anxiety reduction. Patients in other classes may have only begun to expose themselves to anxiety-provoking situations around weeks 6–8, when paroxetine typically reaches therapeutic levels (Liebowitz, 2000), consistent with the lack of differences in amount of change among groups at weeks 8 and 12.

Notably, receiver operating characteristic analysis of the LSAS (Mennin et al., 2002) indicate that a score of 60 or above on the LSAS is a sensitive and specific cutoff score for generalized SAD. All classes had means on the LSAS above 60 at baseline, but only the *Familial Factors* and the *Diffuse Beliefs* classes had means below 60 (38.59 and 47.82, respectively) by Week 4. Moreover, the *Diffuse Beliefs* class demonstrated markedly less change from baseline than the *Familial Factors* class. Importantly, this effect was present *before* paroxetine had the time to be fully therapeutic (Liebowitz, 2000).

4.4. Limitations

This is a novel study of the predictive relationship of etiological attributions about SAD to clinical presentation and response to pharmacotherapy, but several limitations should be acknowledged. First, although this study suggests that patterns of etiological attributions are differentially associated with response to pharmacotherapy, we were not able to examine whether this might be the case in combined CBT and pharmacotherapy as well because of sample size restrictions. Attaining the goals of personalized medicine and treatment matching will require investigating whether groups such as those identified in this study respond differently to varying treatments in prospective studies. For example, a study evaluating whether the *Need to be Liked* and *Bad Social Experiences* groups respond more favorably to psychotherapy than the *Familial Factors* group would be a logical next step.

Secondly, the AESAS was designed in an *a priori* attempt to probe genetic/biological and psychosocial etiological attributions. The results of the LCA did not fully track this prediction as individuals who endorsed early family factors as causes of their SAD were included in the same class as those who endorsed genetics and biological factors. Interestingly, however, parenting behaviors are substantially influenced by genetic factors (Kendler, 1996), and whether this is reflected in one's attributions for etiology is an avenue of future research. Additionally, the number of items in each domain was uneven, with 14 psychosocial items but only three genetic/biological ones. Future research may benefit from

creating a scale with a more even item distribution between domains or expanding the range of potential factors.

Finally, the BDI-II was not administered at weeks 4 and 8. Thus, it was not possible to assess whether changes in depressive symptoms were similar to changes in social anxiety over time. Although it has been demonstrated that changes in depression follow changes in social anxiety among patients with SAD receiving CBT (Moscovitch et al., 2005), future research should evaluate these patterns more fully in pharmacotherapy patients. Finally, no overt measure of expectancy effects was included in this study. Although we conjectured that an attribution-to-treatment match would facilitate a belief in the effectiveness of the treatment thereby increasing response, we were unable to evaluate this supposition directly.

4.5. Implications and conclusions

The finding that a psychosocial etiological attribution may be associated with a more severe clinical presentation at baseline is a marked departure from extant research and may uniquely differentiate SAD from other disorders. Additionally, the influence of etiological attributions on initial response to pharmacotherapy suggests possibilities in augmenting treatment effectiveness. To these ends, the findings highlight the possible significance of treatment modification and selection on a person-specific basis. Psychoeducation that helps to facilitate a shift in patients' etiological attributions to better align with the chosen intervention (e.g., to explain genetic contributions to SAD if the treatment is pharmacological) may increase patient expectancies of treatment, thereby improving outcome. Finally, although this study lacked a comparison group, a reasonable hypothesis for future research is that matching type of treatment with its corresponding causal attribution may improve response. By initially assessing causal attributions, providers may obtain helpful information to aid in treatment selection, perhaps making treatment more effective.

Acknowledgments

Role of Funding Source

Portions of this study were supported by grants from the National Institute of Mental Health to Richard G. Heimberg (R01MH064481) and to Michael R. Liebowitz (R01MH040121) as well as grants to each of these investigators from GlaxoSmithKline Pharmaceuticals, Inc. The funding sources had no involvement in study design, collection, analysis, data interpretation, writing of the report, or the decision to submit the article for publication.

Dr. Schneier reports receiving research funding from Forest Labs.

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Highlights

- Therapies for social anxiety disorder (SAD) leave many individuals symptomatic
- We examined patients' etiological attributions (EAs) for their SAD
- Psychosocial EAs were associated with more severe baseline presentation
- Biological/familial EAs related to quicker response to pharmacotherapy
- Findings have implications for personalized medicine and treatment augmentation

Figure 1.

Mean Attributions for the Etiology of Social Anxiety Scale item scores for each class in the four-class model. Possible item ratings range from 0 (*not at all important as a cause of my social anxiety*) to 4 (*one of the most important as a cause of my social anxiety*). Items 7, “highly perfectionistic parents” and 15, “unstable or abusive family life” are not included in the figure because they were removed from the latent class analysis.

Figure 2.
Mean Liebowitz Social Anxiety Scale (LSAS) scores for each class in the four-class model over the course of treatment with paroxetine.

Table 1

Fit indices for latent class analysis models with 1–4 classes

Number of classes	Number of free parameters	Log likelihood	AIC	BIC	ABIC	BLRT
1	30	−3464.40	6988.81	7076.41	6981.50	N/A
2	46	−3346.00	6783.99	6918.31	6772.79	<.001
3	62	−3308.78	6741.57	6922.60 ^a	6726.46	<.001
4	78	−3272.47	6700.94 ^a	6928.70	6681.94 ^a	<.001

Note. AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion; ABIC = sample-size Adjusted BIC; BLRT = Bootstrap likelihood ratio test. A five-class model produced a class composed of only 5% of the sample and is therefore not reported.

^a Indicates best fitting model according to each index.

Table 2

Comparisons of external validators in the four-class model

	Class 1: Diffuse Beliefs (<i>n</i> = 24)		Class 2: Familial Factors (<i>n</i> = 12)		Class 3: Need to be Liked (<i>n</i> = 53)		Class 4: Bad Social Experiences (<i>n</i> = 48)		Omnibus χ^2 test		Pairwise Comparisons ^a
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	χ^2	<i>p</i>	
Social Anxiety Symptoms: Week 0 (Baseline)											
LSAS – Total score	63.28	4.71	67.34	4.04	77.46	2.67	83.13	3.25	15.43	0.001	Classes 3,4>1,2
Depressive Symptoms: Week 0 (Baseline)											
BDI-II – Total score	6.98	1.71	11.21	2.08	17.53	1.79	21.68	1.83	30.11	<0.001	Classes 3,4>1,2
Social Anxiety Symptoms: Week 4											
LSAS – Total score	47.82	6.66	38.52	4.28	60.80	3.68	67.12	3.93	8.25 ^b	0.041	Class 3,4>2

Note. LSAS = Liebowitz Social Anxiety Scale; BDI-II = Beck Depression Inventory – II. Comparisons on change in social anxiety symptoms by Weeks 8 and 12 and change in depressive symptoms by Week 12 are not reported here; however, they were conducted and no significant between-class differences were found.

^a All pairwise comparisons are significant at the $p < .05$ level.

^b Between class comparisons for differences in LSAS – Total score by Week 4 were conducted using LSAS residualized change scores for each class (which were computed by regressing LSAS – Total at week 4 on LSAS – Total at week 0).