Expert Review of Neurotherapeutics

ISSN: 1473-7175 (Print) 1744-8360 (Online) Journal homepage: informahealthcare.com/journals/iern20

# Issues and controversies surrounding the diagnosis and treatment of social anxiety disorder

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**To cite this article:** Kristy L Dalrymple (2012) Issues and controversies surrounding the diagnosis and treatment of social anxiety disorder, Expert Review of Neurotherapeutics, 12:8, 993-1009, DOI: 10.1586/ern.12.81

**To link to this article:** https://doi.org/10.1586/ern.12.81

EXPERT REVIEWS

# Issues and controversies surrounding the diagnosis and treatment of social anxiety disorder

*Expert Rev. Neurother.* 12(8), 993–1009 (2012)

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Although much has been learned about social anxiety disorder (SAD) in recent decades, many questions and controversies surrounding its diagnosis and treatment have remained. Similar to the state of affairs with other psychiatric disorders, no clear pathophysiology has been identified for SAD, and the question of where to draw the line between shyness, SAD and even avoidant personality disorder continues to be debated. Much of the evidence to date suggests that among persons with SAD, it is under-recognized and undertreated; however, other researchers contend that it may be overdiagnosed in some individuals. Questions also remain as to how best treat these individuals, such as with pharmacotherapy, psychotherapy or a combination of the two. The aim of this review is to provide an overview of the controversies related to the diagnosis and treatment of SAD. In addition, suggestions for future research are provided that could perhaps clarify these remaining questions, such as maximizing treatment efficacy by targeting broader outcomes such as quality of life and addressing common comorbidities that occur with SAD.

**Keywords:** cognitive behavior therapy • comorbidity • diagnosis • etiology • pharmacotherapy • social phobia • treatment



 10.1586/ERN.12.81  ISSN 1473-7175 

**Financial & competing interests disclosure**

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*Publisher, Future Science Group, London, UK.*

*Disclosure: Elisa Manzotti has disclosed no relevant financial relationships.*

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*Disclosure: Laurie Barclay, MD, has disclosed no relevant financial relationships.*

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*Disclosure: Kristy L. Dalrymple, PhD, has disclosed no relevant financial relationships.*

Social anxiety disorder (SAD) currently is defined as a significant fear of embarrassment or humiliation in social situations to the point that these situations often are avoided or endured with a significant amount of distress [1]. Typical situations feared or avoided by individuals with SAD include performance situations (e.g., giving a speech to an audience) or interpersonal situations (e.g., initiating conversations with individuals at a party). Currently, two subtypes are distinguished: generalized and specific. Definitions of the subtypes vary, but the DSM-IV defines the generalized subtype as fear in 'most' social situations, while the specific subtype is fear in only a few situations [1]. Epidemiological studies have indicated a lifetime prevalence rate of SAD of approximately 7–13% in Western countries, and it is the fourth most common mental disorder in the USA [2,3]. Studies within clinical samples have found a prevalence rate as high as 30% [4]. In addition, SAD has a cumulative incidence rate of 11% in the first three decades of life, with peak incidence generally occurring between 10 and 19 years of age [5]. SAD largely follows a chronic course, with patient populations reporting an average duration of SAD between 10 and 24 years [6] and recovery rates of approximately one-third after 8 years [7]. A more recent prospective community study of German females also found a full recovery rate of 36% after 1.5 years [6].

It has been well documented that SAD is highly comorbid with other psychiatric disorders, including mood, other anxiety and substance use disorders [8]. For example, Acarturk *et al*. found that 66% of individuals with SAD in a population-based sample met criteria for at least one other psychiatric disorder [9]. Among Axis II disorders, avoidant personality disorder (AVPD) is most commonly associated with SAD, occurring in up to 89% of individuals with the generalized subtype of SAD [10]. SAD is associated with lower levels of educational attainment, single marital status and unemployment [11], as well as fewer days worked and reduced work productivity [12]. Individuals with SAD also report poor quality of life [12,13] and high levels of service utilization [12,14], although not always for SAD specifically (see below). As a result, SAD is associated with substantial economic costs [15]. Despite all that has been discovered about SAD in the past three decades, controversies and questions remain about its etiology, diagnosis and treatment.

## Controversies & issues in the diagnosis of SAD

The aim of this review is to provide an overview of the various questions and controversies related to the diagnosis and treatment of SAD, in an effort to stimulate future research efforts to better identify and treat it. A summary of these issues or controversies is presented in TABLE 1, along with suggestions for future research that could potentially address these remaining questions.

### *Which etiological model best describes the phenomenon?*

Although it is acknowledged that it is not necessary for a definitive etiology to be known in order to diagnose a mental disorder according to DSM-IV, increasingly there is an assumption that just as other medical diseases are assumed to have clear underlying pathophysiologies, so too should mental disorders, and determining underlying biological mechanisms for mental disorders will aid in tailoring treatments for individuals [16]. For example, the DSM-IV defined a mental disorder in part as 'a manifestation of a behavioral, psychological or biological dysfunction' [1]. However, for DSM-5, it has been proposed to alter that criterion to read: 'that reflects an underlying psychobiological dysfunction' [17]. There is an increasing acknowledgment that biological factors do not 'map' onto current DSM-IV diagnostic categories with sufficient specificity, thus spurring interest in determining whether these biological factors 'map' onto neurobehavioral domains (as reflected in the aims of the Research Domain Criteria Project from the National Institute of Mental Health [201]). Specific to SAD, the past few decades have seen a rise in studies examining potential biological (e.g., brain activation patterns, neurotransmitters, genes, autonomic nervous system [ANS] abnormalities, hypothalamic–pituitary–adrenal axis abnormalities and neuropeptides) and environmental (developmental, behaviorally inhibited temperament, fear conditioning and extinction) etiological factors. Although a comprehensive review of all of the potential etiological factors related to SAD is beyond the scope of the current paper, a brief summary of the most relevant or recently studied factors is presented below.

#### Neuroimaging studies

With respect to brain activation patterns, some studies have reported that greater activation in the amygdala, amydala–hippocampal

**Table 1. Unanswered questions and suggestions for future research about the diagnosis and treatment of social anxiety disorder.**

| Remaining issues/questions | Suggestions for future research |
|---|---|
| *Diagnosis/etiology* | |
| Specificity of biological factors in the etiology of SAD | Studies comparing SAD to other anxiety disorders; studies using medication-naive samples |
| SAD as a dimensional versus categorical experience | Replication of taxometric studies in clinical samples |
| SAD and AVPD as separate versus the same disorder | Larger studies comparing clinical characteristics between SAD alone, AVPD alone and SAD + AVPD; considering revisions to the criteria |
| Under-recognition of SAD | Additional mental health literacy studies to examine knowledge of SAD among potential treatment seekers and healthcare providers; improved use of cost-effective screening measures |
| *Treatment* | |
| Undertreatment of SAD | Additional studies to examine individual and societal factors related to the underutilization of treatment |
| Pharmacotherapy versus psychotherapy | More long-term outcome studies; measurement of broader outcomes such as quality of life and functional impairment |
| Combined treatment (medication/other compounds plus psychotherapy) | More long-term outcome studies; measurement of broader outcomes such as quality of life and functional impairment; effects of withdrawing medication on psychotherapy response; more frequent measurement of symptoms other than pre–post (e.g., weekly measurements) to examine potential interactions between the two approaches |
| Alternative treatment strategies | Additional studies on various forms of self-help/guided self-help, particularly for subthreshold levels of SAD; cost–effectiveness studies |
| Impact of comorbidity | Additional studies to test treatments that address other comorbidities in addition to SAD; assess the effect of comorbidities in standard treatments for SAD |

AVPD: Avoidant personality disorder; SAD: Social anxiety disorder.

region and frontolimbic system is associated with exposure to fearful stimuli in individuals with SAD [18–20]. A recent review of neuroimaging studies [21] summarized studies showing increased activity in limbic and paralimbic regions in individuals with SAD compared with healthy controls, especially in response to facial expressions [22], aversive conditioning [23] and public speaking tasks [24], although findings in these areas have been somewhat mixed [25]. Other studies have found reduced amygdala response to public speaking tasks after treatment [26] but mixed findings with respect to activity in the insula (e.g., [23,27], as reviewed by [21]).

However, one limitation with such studies is that they only have the ability to identify correlates of SAD-related behaviors in the brain but not necessarily their causes. Although findings from these studies are interesting and shed some light on biological processes that may be associated with SAD, it is difficult to generalize findings due to the small sample sizes and lack of consistent replication. Furthermore, only some of these studies have included another anxiety disorder comparison group [28]. Therefore, it is difficult to determine whether the effects found are associated with SAD specifically. Freitas-Ferrari *et al.* noted other limitations in their review, such as the wide variation in methodology across studies, the presence of comorbid conditions in some studies, the lack of clarity as to whether differences in activation patterns represent pathological manifestations or compensatory responses, and age and/or gender differences across studies [21]. Replication with studies examining hypothalamic–pituitary–adrenal axis and ANS dysfunction has also been difficult to achieve, with some studies finding greater cortisol reponse [29] and ANS reactivity [30] following stress challenges in participants with SAD compared with controls, while other studies have found no such differences [31,32].

### Neurotransmitters

Other studies have examined neurotransmitters as potential etiological factors, particularly dopamine and serotonin [33,34]. For example, Schneier *et al.* [33] found a significantly lower dopamine $D_2$-binding potential in ten participants with SAD compared with ten healthy controls, and Warwick *et al.* [34] found decreased regional cerebral blood flow following treatment with citalopram. However, this latter study also found decreased regional cerebral blood flow following treatment with moclobemide, suggesting this effect was not specific to citalopram. Many of the studies examining the role of neurotransmitters have based findings on treatment outcome using serotonin

reuptake inhibitors (SSRIs) or other medications [34], which introduces the problem of inferring etiology from treatment. In other words, just because an individual with SAD experiences a decrease in symptoms after taking medication, this does not necessarily indicate that SAD was caused by factors affected by the medications. Furthermore, other studies have included patients who at the time were receiving medication treatment for SAD or who had previously taken medication [33,35], or were receiving concomitant medication such as benzodiazepines [34]. Therefore, these studies have not taken into account the effects of the psychoactive medication themselves, which are known to affect neurotransmitter activity.

### Genetic & family studies

Studies also have been conducted to attempt to identify specific genes related to SAD, particularly the human serotonin transporter gene (*5-HTT*). However, these findings have been unable to be consistently replicated and possess weaknesses due to the lack of a control group or small sample sizes [36,37]. Family and twin studies have shown more promise, especially as these studies have included larger sample sizes. For example, prospective family studies of SAD have shown that parental SAD was associated with increased risk for SAD in offspring, both independently and in combination with parental rearing practices such as overprotection [38]. In addition, Kendler *et al*. conducted a twin study using 700 pairs of monozygotic twins and 500 pairs of dizygotic male twins, across five different phobias [39]. Genetic factors accounted for significant variance in the lifetime prevalence of anxiety disorders in general and specific anxiety disorders, but so too did person-specific environmental factors. Twin studies also have found heritability rates of SAD ranging from 20 to 50% [39,40], although these researchers have acknowledged that genetic factors do not play an 'overwhelming' role in the etiology of phobic disorders. Other twin studies have been unable to support heritability for specific anxiety disorders [41]. By contrast, the study of environmental factors associated with SAD has produced fairly consistent results, showing support for various factors such as parental overprotection and rejection [42]. To summarize, results from family studies suggest familial transmission that may be specific to SAD, whereas the mixed findings from the twin studies suggest a predisposition toward anxiety more generally rather than to specific anxiety disorders. In a review, Hudson and Rapee concluded that given the greater specificity obtained in family studies (which measure both genetic and environmental factors) compared with twin studies, the family environment likely plays a more instrumental role in the transmission of SAD [43]. Therefore, although genetic factors may play a part, current evidence suggests that its role as an etiological factor is largely dependent upon more proximal environmental factors.

## *Are we overpathologizing shyness?*

Questions of the etiological basis of SAD naturally lead to the question of where to draw the line – what distinguishes someone who simply is 'shy' from someone who would be considered to have a disorder? The diagnosis of SAD first appeared in DSM-III in 1980, and soon after its appearance, it was considered to be 'among the most neglected of the major anxiety disorders' [44]. Since that time, a large research base has accumulated, and results from an epidemiological study indicated that lifetime prevalence rates of SAD have increased in recent cohorts [45]. Owing to the changing diagnostic criteria and the subsequent increase in the prevalence of SAD in recent cohorts, some researchers have questioned whether the experience of social anxiety is being overpathologized [46]. Conversely, other researchers have argued that SAD previously was underdiagnosed in epidemiological studies using DSM-III or DSM-III-R criteria (e.g., the Epidemiological Catchment Area Study [ECA]; [47]) compared with later epidemiological studies (e.g., the National Comorbidity Survey-Replication [NCS-R]; [3]), based on the way that SAD was defined [48,49].

Wakefield *et al.* have argued that many cases of currently defined SAD are not individuals who demonstrate disordered shy behaviors; rather, these individuals demonstrate behaviors that lie along a continuum of normal temperamental variants [46]. In addition, they argued that social anxiety may have evolutionary functions, such as preventing the pursuit of social relationships that may pose certain risks. Although Wakefield *et al.* stated that most cases of SAD would not meet the threshold of 'disorder', they acknowledged that many of these individuals still could benefit from treatment. In response to Wakefield *et al.*, Campbell-Sills and Stein argued that when high levels of social anxiety are present, mental/physical mechanisms that generate social anxiety (e.g., negative cognitions about performance, physiological symptoms) become overactive; this overactivity therefore suggests the presence of a disorder [50]. For instance, they noted that when these social anxiety mechanisms are overactive (i.e., 'malfunctioning'), these individuals are not minimizing risk in social relationships and at the same time allowing for a normative range of social interactions. Therefore, Campbell-Sills and Stein argued that high levels of social anxiety do not have evolutionary benefits (especially in modern society) because it may lead them to be 'shunned' by others (e.g., they may be perceived as being 'standoffish' by others when demonstrating shy behaviors). This, in turn, could lead to consequences related to work and social functioning.

Much of the research to date suggests that SAD is underdiagnosed, particularly in clinical samples in which other psychiatric disorders are present [51,52]. However, others have noted that there is no 'gold standard' in the DSM-IV for determining sufficient impairment or distress to warrant a diagnosis. Thus, it is a subjective and somewhat arbitrary distinction between normal variation and pathology [2,48]. Researchers have examined the degree to which prevalence rates change in epidemiological samples when altering the definition of significant impairment or distress, finding that the rates range from as low as 1.9% to as high as 22.6% [48,53–55]. Studies in countries such as Mexico [56] and Australia [57] have indicated lower prevalence rates of SAD when using ICD-10 diagnostic criteria. Narrow *et al*. applied a clinical significance criterion in an attempt to reconcile the different prevalence rates of disorders in the ECA and NCS studies [58]. The past-year

prevalence rate of SAD in the NCS study decreased from 7.4% to 3.7%, but remained at 1.7% in the ECA study after the criterion was applied. One potential explanation for these findings is that the ECA study originally applied a more stringent clinical significance criterion, by assessing clinical significance for each symptom in the anxiety disorders. By contrast, the NCS study assessed clinical significance only for the disorder overall. The controversy remains as to how liberally or conservatively to apply the criterion of significant distress or impairment; nonetheless, it is important to note how prevalence rates can change when the definition of clinically significant impairment and distress is modified.

Inherent in this discussion is the relationship between shyness and SAD; for example, is SAD just a more severe form of shyness, or are they distinct categories? Historically, shyness has been defined as a temperament that is transitory in nature [59], while SAD is conceptualized as being chronic and unremitting [60]. Heiser *et al*. compared prevalence rates and characteristics of individuals classified as shy versus having SAD in an undergraduate sample and examined the overlap between the two [61]. They found that nearly half of their sample was classified as shy, while approximately 10% met criteria for SAD (with the majority having the generalized subtype). In addition, they found that only 17% of those classified as shy met criteria for SAD, whereas 85% with SAD also were classified as being shy. More severe shyness was associated with a greater likelihood of having SAD, and the characteristics related to shyness versus nonshyness were similar to the clinical presentation of individuals with SAD (e.g., comorbidity with anxiety, mood and substance use disorders, as well as AVPD). The authors concluded that based on these results, shyness and SAD are related but SAD is not simply a more severe form of shyness.

However, the evidence taken together is still suggestive of the dimensional nature of the experience of social anxiety. For example, a study of a Brazilian college student sample found that students with SAD had greater severity and impairment compared with students with subthreshold SAD and compared with controls; students with subthreshold SAD also demonstrated greater severity and impairment compared with controls [62]. Two studies used a taxometric method to compare a dimensional versus categorical system of SAD, with results from both studies favoring a dimensional system [63,64]. Ruscio also demonstrated superior predictive validity of the dimensional system on some clinical indicators, such as subsequent onset of suicidal ideation and mood disorder [64]. While these results support a continuum of SAD, Filho *et al*. [62] and Ruscio [64] acknowledged the concern that switching to a dimensional system may overexpand an already somewhat controversial diagnosis. The Anxiety Disorders Work Group for DSM-5 also has concluded that the evidence to date suggests that social anxiety exists on a continuum, based on studies showing that a greater number of social fears indicates greater severity [65]. As a result, they are considering eliminating the 'generalized' subtype specifier, as this current categorization does not reflect the continuous nature of social anxiety. Instead, they suggest inclusion of a specifier for a 'performance only: if the fear is restricted to speaking or performing in public', given the evidence suggesting that those individuals with only performance anxiety are qualitatively distinct from other individuals with SAD [65].

### *Are SAD & AVPD the same disorder or distinct categories?*

In addition to the question of the relationship between shyness and SAD, another controversy in the diagnosis of SAD relates to its overlap with AVPD. AVPD is generally defined by the DSM-IV as: 'a pattern of social inhibition, feelings of inadequacy and hypersensitivity to negative evaluation' [1]. Many of the diagnostic criteria appear to overlap with SAD, such as avoidance of occupational, interpersonal or unfamiliar situations due to fears of inadequacy and being embarrassed, and a preoccupation with being criticized or rejected by others in social situations [1]. Given the considerable overlap between SAD and AVPD, debate continues as to whether they represent distinct disorders or a continuum of severity. Many researchers have concluded that AVPD is merely a more severe form of SAD [65] based on symptom profiles [66] and family studies [67]. However, other researchers have argued that AVPD and SAD are two distinct disorders based on studies that have indicated a sufficient percentage of individuals diagnosed with AVPD without SAD [68], and on studies that have associated AVPD with schizophrenia spectrum disorders [69], although findings in this area have been mixed [65]. There continues to be no definitive answer to this debate; therefore, at this time, they continue to be viewed as different disorders and both can be diagnosed concurrently under the current nomenclature [1].

### *Is SAD being under-recognized in clinical settings?*

Although SAD may be overdiagnosed in some cases as indicated above, it also is possible that among those who experience significantly impairing SAD, it may be under-recognized in clinical settings. For example, a study in a general outpatient psychiatry setting found that when a semi-structured diagnostic interview was used, SAD was nine times more likely to be diagnosed compared with when an unstructured clinical interview was administered [51]. A similar trend was found in a primary care sample, where only 24% of individuals diagnosed with SAD by a structured interview were identified by the physician as having SAD [52]. Although studies have shown that individuals with SAD show greater rates of healthcare utilization compared with individuals without SAD [12,14], these findings tend to be misleading because many of these individuals are not seeking treatment for SAD [9,70]. Rather, these individuals most often are seeking treatment for more acute problems, such as depression or other acute anxiety disorders [52]. Therefore, they may perceive that their other symptoms (e.g., symptoms of depression) are more significant at the time of presentation, and their social anxiety symptoms do not meet a threshold level to warrant attention. In line with this, a recent study by Dalrymple and Zimmerman in an outpatient psychiatric sample examined predictors of those seeking treatment principally for SAD versus those who had SAD as a comorbid diagnosis [70]. Results indicated that those who endorsed a greater number of social fears and who experienced less severe forms of depression (i.e., a diagnosis of depressive disorder not otherwise

specified compared with major depression) were more likely to have been seeking treatment primarily for SAD.

It also has been hypothesized by Dalrymple and Zimmerman that one reason for the under-recognition of SAD may be that historically it has received less media attention compared with depression or other anxiety disorders [71]. By contrast, a study by Coles and Coleman examined mental health literacy of SAD compared with generalized anxiety disorder (GAD), obsessive–compulsive disorder (OCD), panic disorder (PD) and depression, by presenting college students with vignettes of each of these disorders [72]. Results indicated recognition rates of approximately 80% for SAD and OCD, similar to that of depression; conversely, less than half of the participants recognized PD and GAD. It is interesting that SAD was recognized at a similar rate as OCD, given that OCD has received a large amount of media attention via television shows (e.g., The OCD Project). Another possible reason for under-recognition of SAD may be related to the nature of the disorder. Because individuals with SAD experience fear that others may judge them, they may feel embarrassed about seeking help. In fact, a study by Olfson *et al*. indicated that one reason for not seeking treatment cited by individuals with SAD was a concern of what others might think or say about them [73]. Furthermore, due to the nature of the disorder, such individuals may wish to continue to avoid the anxiety rather than face it. As a result, these individuals may be less likely to seek treatment at all or less likely to voice these concerns to health professionals, thereby decreasing the likelihood that SAD would be detected [73]. Owing to the early onset and chronicity of SAD, many individuals may also believe that these symptoms are part of their personality and therefore cannot be changed [74]. Regardless of the potential reasons for why individuals may not seek treatment, the evidence to date suggests that a significant number of individuals who suffer from significantly impairing SAD do not have this problem adequately identified [51,52].

### *Conclusions on diagnosis & etiology*

Research has indicated that SAD is often a chronic and significantly impairing problem. As is the case with other psychiatric disorders, no clear and definitive biological etiology has been identified for SAD despite decades of research. Biological factors such as activation in particular brain regions and neurotransmitters have been studied as possible factors, but at best, these studies have shown these factors to be correlates of social anxiety and not necessarily causes of the disorder. Furthermore, family and twin studies have not been able to parse out the distal effects of genetic factors from the more proximal effects of environment, and no specific set of genetic factors has been identified reliably. Therefore, the evidence to date suggests that multiple factors likely are involved in the development of SAD, including more proximal environmental factors. Questions remain as to where to draw the line between a normal variant of a shy temperament and clinical disorder [2,46], and the most recent research suggests that SAD is best captured as a dimensional experience rather than a categorical distinction [63,64]. Future research should attempt to better discern at which point the experience of social anxiety becomes pathological, as this has important treatment implications. For example, to what degree are pathological levels of SAD being missed, therefore preventing such individuals from receiving necessary treatment? Conversely, to what degree are subthreshold levels of SAD being overdiagnosed, thus exposing such individuals to potentially costly and unnecessary treatment? Are there less costly treatments with fewer risks that could potentially benefit individuals with subthreshold (but still problematic) levels of SAD? The following section addresses the current state of affairs with respect to the treatment of SAD.

## Controversies & issues in the treatment of SAD

### *Is SAD undertreated?*

In addition to being under-recognized, SAD also seems to be undertreated. Population-based studies have indicated that many individuals with SAD do not receive treatment for it [75]. Data from the ECA study showed that approximately two-thirds of individuals with SAD reported that they had never received outpatient mental health treatment [76], and only drug and alcohol use disorders have lower rates of treatment [75]. A more recent study assessed individuals in a probability sample from the ECA follow-up study on need for and use of mental health services [77], finding that SAD was the disorder with the lowest proportion of met need for treatment (at 7.9%).

Research also has indicated that there is a significant delay between identification and treatment initiation for SAD relative to other anxiety disorders. For example, Wagner *et al*. found that individuals with SAD experienced a longer delay to seeking treatment compared with individuals with PD and GAD [78]. The time between symptom onset and first contact with a health professional was 46 months for individuals with SAD, compared with 14 and 10 months for PD and GAD, respectively. This delay significantly increased when considering specific anxiety treatment; there was a delay of 166 months between symptom onset and presentation to a specialty anxiety clinic for individuals with SAD compared with 79 and 84 months for PD and GAD, respectively. Interestingly, although individuals with SAD may take longer to initiate treatment or prioritize other symptoms for treatment, if directly asked they often express a desire for treatment of SAD [51]. Dalrymple and Zimmerman found that predictors of desire of treatment for comorbid SAD have included a greater number of social fears endorsed and poorer work functioning, even after controlling for depression and overall illness severity [71].

If left untreated, the presence of SAD may impact upon the clinical presentation of other comorbidities. The best example is with depression, as SAD is the most common comorbid anxiety disorder with depression in clinical samples [79,80]. The presence of SAD in individuals with depression is associated with greater severity of symptoms [80,81], an earlier onset of symptoms [5,70], greater functional impairment [12,80] and greater comorbidity (particularly with other anxiety disorders and substance use disorders) [80,82]. A recent study in a large community sample also found that compared with individuals with episodic depression, those with long-term depression were significantly more likely to have SAD and benzodiazepine abuse [83].

### *Pharmacotherapy versus psychotherapy: is one better than the other?*

The research described above suggests that for those who experience significantly impairing and/or distressing SAD, it often goes undertreated. Fortunately, there are several efficacious treatments for SAD. Cognitive behavior therapy (CBT) is the most studied psychotherapy for SAD; several studies have demonstrated its efficacy both in the short term and long term [84], with moderate-to-large effect sizes at post-treatment compared with control conditions [85]. Evidence also supports the use of cognitive therapy (CT) [86] and internet-delivered CBT for SAD [87]. Several medications have demonstrated short-term efficacy for SAD, including the selective serotonin/serotonin–norepinephrine reuptake inhibitors (SSRIs/SNRIs), monoamine oxidase inhibitors (MAOIs) and to a somewhat lesser degree, benzodiazepines [88]. For example, a meta-analysis by van der Linden *et al.* showed small to large effect sizes for sertraline, fluvoxamine and paroxetine compared with placebo; paroxetine, sertraline and venlafaxine also have received the US FDA indications for the treatment of SAD [89]. Another meta-analysis by Blanco *et al*. found that although the MAOI phenelzine demonstrated the largest effect sizes, it did not significantly differ from other classes of medications studied [90]. Due to the dietary restrictions and adverse side effects of MAOIs, SSRIs/SNRIs are considered to be the first-line pharmacological treatment for SAD [88].

Some studies have directly compared pharmacotherapy and CBT for SAD, with mixed findings depending on whether short- or long-term results were examined and what types of outcome variables were studied. Regarding short-term results, Heimberg *et al*. found that both CBT and phenelzine were efficacious after 12 weeks of treatment, with phenelzine being slightly superior to CBT on independent assessor and self-report measures of symptom severity [91]. However, CBT and phenelzine did not differ from each other on self-report measures of social avoidance or self-rated performance satisfaction during a behavioral test, and response rates were nearly identical (77% for phenelzine vs 75% for CBT). A meta-analysis by Gould *et al.* also showed that both medications and CBT were efficacious compared to control conditions but did not differ from each other [92]. However, a meta-analysis comparing several different classes of medications and different CBT approaches (e.g., exposure, cognitive restructuring, full-package CBT) found that the most consistently efficacious treatments in the short term were SSRIs and benzodiazepines [93]. A more recent study comparing fluoxetine, CBT and placebo also found that fluoxetine and CBT were both efficacious compared with placebo but did not differ from each other after 14 weeks of treatment [94]. Only one known study directly compared clonazepam with group CBT [95], finding that clonazepam did not demonstrate greater efficacy compared with CBT after 12 weeks of treatment on clinician-rated measures. Although some studies have demonstrated superiority of medication compared with CBT in the short term, placebo-controlled discontinuation studies have also consistently demonstrated a higher rate of relapse in medication compared with CBT conditions [96–98].

Few studies directly comparing medications and CBT for SAD have included long-term follow-up periods. However, one study by Liebowitz *et al*. (a follow-up to the study by Heimberg *et al*. [91]) compared phenelzine and CBT after a 6-month maintenance phase and then an additional 6-month follow-up phase [99]. Results showed that those who received phenelzine had a significantly greater rate of relapse during the follow-up phase compared with those who received CBT (50 vs 17%, respectively). Another more recent study comparing CT with fluoxetine plus self-exposure and placebo plus self-exposure found that CT was superior to the other two conditions at post-treatment and at 12-month follow-up [86].

Therefore, the question of 'which one is better' depends on whether short-term or long-term benefits are being considered. If considering acute outcomes, pharmacotherapy appears to provide symptom reduction more rapidly compared with CBT, although these differences dissipate over time. On the other hand, CBT appears to provide better long-term outcomes, as it has demonstrated lower relapse rates compared with patients who have received pharmacotherapy. Given the chronic nature of SAD and risk of relapse, treatments that can successfully change the course of the disorder in the long term are most important to implement. As a result of these findings, researchers have been interested in whether the rapid effects of medication and the long-term benefits of CBT could be combined.

### *Combined pharmacotherapy & psychotherapy: are two better than one?*

On the basis of the comparative studies reviewed above, it had been assumed that the efficacy of combined treatment (pharmacotherapy plus CBT) for SAD would be superior to the efficacy of either monotherapy. One study by Blomhoff *et al*. compared exposure alone, sertraline alone, exposure plus sertraline and placebo [100]. Although the addition of exposure to sertraline resulted in significant change in symptom severity earlier (by week 8), in general, the results showed few significant differences between the combined treatment and either monotherapy. In addition, a study by Davidson *et al*. compared fluoxetine, CBT, placebo, CBT plus fluoxetine and CBT plus placebo [94]. After 14 weeks of treatment, all active treatments were superior to placebo, but there was no significant advantage of combined treatment over either monotherapy. By contrast, a more recent study compared phenelzine, CBT, placebo and phenelzine plus CBT in an acute phase 12-week trial and an additional 12-week continuation phase [101]. The combined treatment was superior to either monotherapy after both the acute and continuation phases. The authors suggested that these positive findings, which differed from prior combined treatment studies using SSRIs, may have been due to the use of medications with greater efficacy compared with the mixed or poor findings of efficacy of the medications used in prior trials [101]. In addition, it is possible that these more positive short-term findings with phenelzine compared with SSRIs could be due to the sedating effects commonly associated with this medication [202]. Therefore, the effects of phenelzine may be similar to those of benzodiazepines, in which a rapid symptom reduction occurs followed

by high relapse rates once the medication is discontinued [99,102]. However, this study did not include a discontinuation follow-up phase in order to assess relapse rates in each of the conditions.

Unfortunately, very little research has been conducted on the long-term efficacy of combination treatment. However, one study by Haug *et al.* [103], a 1-year follow-up study from the trial by Blomhoff *et al.* [100], found that exposure alone continued to improve during the follow-up period but sertraline and sertraline plus exposure showed deterioration during the follow-up period. The authors concluded that perhaps one reason for this finding is that: 'exposure techniques applied in situations with low levels of anxiety achieved by medication may have less impact than exposure therapy applied in situations with a higher level of anxiety and may lead to a higher degree of relapse after end of treatment'. On the basis of the existing literature, there is not yet strong evidence for combination treatment over either monotherapy; however, due to the paucity of research in this area this should be studied further, with a particular focus on long-term outcomes.

### *Can certain medications interfere with or enhance the process of exposure?*

Given the increase in use of combined treatment approaches for SAD in recent years, it is important to consider how best these two types of treatment modalities can be combined. For example, are there certain medications that can better complement the goals of CBT compared with others, such as standing-dose (i.e., SSRIs/SNRIs) versus as-needed (i.e., benzodiazepines) medications? In order to sufficiently explore this question, a brief discussion of a behavioral model of SAD and how exposure is applied is necessary.

#### Exposure model

On the basis of traditional conditioning models (e.g., Mowrer's two-factor theory; [104]), it is theorized that the experience of anxiety in social interactions will produce a conditioned fear response that is triggered when the person encounters a similar situation in the future. Individuals then learn to avoid social situations in order to escape the short-term anxiety that is elicited from social cues. Although this avoidance decreases distress momentarily, it further reinforces avoidance in the long term via negative reinforcement; therefore, an expanding pattern of avoidance is developed, which can cause long-term problems in functioning. Thus, the goal of exposure therapy is to extinguish conditioned fears and break the avoidance pattern by having patients repeatedly expose themselves to social situations and the triggered anxiety. One theory on the mechanism of exposure is that anxiety decreases over time via the process of habituation [105]. In addition, skills related to coping with the anxiety effectively are learned and reinforced through repeated exposure. However, newer theories on the mechanisms of exposure suggest that acceptance of fear is more critical to extinction rather than reduction of fear and that inhibitory learning (i.e., the development of competing, nonthreatening associations across different contexts) also is central to the extinction process [106].

Therefore, the process of exposure requires that the individual experiences anxiety at a sufficient level of intensity, in order to learn how to effectively cope with that anxiety and develop new associations with the feared stimulus. It has been shown that patients who experience an initial 'spike' in anxiety at the beginning of treatment (meaning that their anxiety was sufficiently provoked) tend to have better outcomes overall compared with patients who do not experience that pattern [10]. Thus, the question becomes whether medications prescribed on an as-needed basis (i.e., benzodiazepines) could interfere with the exposure process, perhaps because they reduce anxiety and thereby prevent the 'spike' in anxiety. In other words, it has been hypothesized that the learning that occurs from the exposure process only occurs in the context of benzodiazepine use and may not generalize to other contexts in which benzodiazepines are not used [107].

#### Benzodiazepines & exposure

As described above, meta-analyses have indicated that benzodiazepines are effective in reducing anxiety in the short term [93]. However, most studies examining the efficacy of benzodiazepines assess only SAD symptom severity, rather than broader outcomes such as quality of life, functioning or even behavioral performance in social interactions. In particular, only short-term effects of these medications are studied. For example, the meta-analysis by Fedoroff and Taylor indicated that only three of the nine medication studies used in their meta-analysis had follow-up data, with the mean duration of the five benzodiazepine trials being approximately 11 weeks [93]. One study [108] examined 2-year follow-up data from a prior trial comparing clonazepam to placebo [109], finding no significant differences between clonazepam and placebo. Unfortunately, little research has been done to examine the potential effects (both short term and long term) of benzodiazepine prescribing on exposure therapy for SAD. However, one naturalistic study examined predictors related to long-term outcome (up to 10 years) of exposure therapy for SAD [102]. At the beginning of the study, 84% of patients were taking a benzodiazepine; no new benzodiazepines were prescribed during the course of exposure therapy. Results showed that concurrent use of benzodiazepines was a significant risk factor for relapse, with those who were still taking benzodiazepines at the end of exposure therapy having a poorer outcome compared with those who did not take benzodiazepines. This issue has been studied more frequently within PD, with benzodiazepine use and discontinuation being associated with poorer therapy outcomes [107].

Concerns also exist regarding withdrawal symptoms after discontinuation of benzodiazepines [88,109,110] and the potential for abuse/physical dependence [93]. It is believed that slow discontinuation of benzodiazepines eliminates or significantly reduces the occurrence of withdrawal symptoms and the risk of relapse [111]. However, some evidence suggests that this may not be the case. For example, Connor *et al.* examined double-blind randomization to continuation of clonazepam versus discontinuation with placebo matching after 24 weeks of open-label treatment [112]. Although sample size was likely too small to compare relapse rates as a categorical variable (intent-to-treat sample size of 36),

a Kaplan–Meier analysis showed that patients in the discontinuation condition exhibited a significantly shorter time to relapse compared with patients in the continuation condition. Presence of withdrawal symptoms appeared to increase in the discontinuation condition during the taper period (from 6.7 to 27.8%), and there was a trend toward the discontinuation condition showing a greater number of withdrawal symptoms compared with the continuation condition at the midpoint of the taper period. Connor *et al.* acknowledged that a limitation of their study was assessing withdrawal symptoms only 1 week after both groups had fully discontinued clonazepam [112]. It is important to assess this for a longer period of time, as a recent prospective study showed four different patterns of withdrawal symptoms (one of which included an increase in withdrawal symptoms 4 weeks after tapering was completed [113]). It also has been argued that the decision on the length of the taper period, historically, has been based more so on clinical experience rather than strong empirical evidence [114,115].

Owing to these concerns, the use of benzodiazepines is considered to be a second-line treatment [88,116,117]. Interestingly, a study by Seedat and Stein found that clonazepam also did not show any incremental benefit when used as an augmentation agent to the SSRI paroxetine [118]. Despite its status as a second-line treatment, benzodiazepines still are commonly prescribed. For example, Mojtabai and Olfson examined national trends in psychotropic medication prescribing in the USA from 1996 to 2006, finding that sedative hypnotics were the second most commonly prescribed psychotropic medications behind antidepressants [119]. Furthermore, the prescription of two or more sedative hypnotics increased significantly over time, particularly for those who were female, had a diagnosis of an anxiety disorder and had diagnostic comorbidity. This trend may have fatal consequences, as overdose deaths related to anxiolytics such as diazepam and alprazolam tripled between the years of 2000 and 2008 [120]. In addition, benzodiazepines were one of the two most frequently reported prescription medications in emergency department drug abuse-related cases, and in 2002, they accounted for 100,784 emergency department mentions that were classified as drug abuse cases [121]. A review by Lader [114] also indicated that although benzodiazepines are recommended for short-term use (e.g., 2–4 weeks), they often are used for much longer periods, ranging from 6 months [122] to as long as 12 years [123].

### SSRIs/SNRIs & exposure

Although SSRIs/SNRIs do not have the same acute effect on anxiety as benzodiazepines, do they also have the potential to interfere with exposure therapy? As discussed, prior research has shown that relapse rates are high once medication is discontinued [97,124,125]. One naturalistic treatment study showed that pre-existing antidepressant use neither enhanced nor detracted from the effectiveness of group CBT for SAD [126]. By contrast, a 1-year follow-up study of a randomized controlled trial showed that patients who had received combination treatment of sertraline and exposure deteriorated during the follow-up period, while those who had received exposure therapy alone experienced further improvement during the follow-up period [103]. Additional research needs to be conducted in this area to determine what long-term impact medication in general may have on exposure therapy for SAD.

### Other compounds & exposure

More recently, researchers are examining ways in which medications or other compounds may be able to facilitate the process of extinction that occurs during exposure therapy. One such compound is D-cycloserine (DCS), a partial NMDA agonist that first was shown in animal models to facilitate the extinction of learned fear when administered immediately prior to or shortly after extinction training [127]. Two studies have examined its potential enhancement of five sessions of exposure therapy in SAD [128,129], both of them finding that patients receiving DCS with exposure reported significantly less social anxiety compared with patients receiving placebo with exposure. However, limitations of these studies include small sample sizes, a lack of consistent findings across self-report and clinician-rated measures in the study by Hofmann *et al.* [128], use of only self-report measures in the study by Guastella *et al.* [129], and a short follow-up period in both studies (1 month). Findings from studies on other anxiety disorders using DCS or other compounds have been mixed, with the most recent studies showing negative results in PD [130,131]. No studies have compared the use of DCS to another active compound for SAD. Other general limitations noted with DCS include that it may not work on re-extinction, and it shows tolerance effects (as reviewed by Davis [132]). Researchers now have begun to examine ways that fluoxetine may enhance extinction learning in mice and suggested that fluoxetine increases plasticity in neurons associated with the processes of fear conditioning and extinction [133]. However, prior studies using animal models to test antidepressant effects have tended to show either poor reliability across studies, poor predictive validity (e.g., drug action in the animal model does not correspond to drug action in the clinic) or high false-positive rates (see review by Bourin *et al.* [134]). Furthermore, it has been argued that using animal models to study anxiety is an oversimplification of complex human processes (e.g., cognition and language, important aspects of the learning process) that will never be accessible in animals (see review by Steimer [135]).

In summary, the existing literature suggests that although benzodiazepines effectively reduce anxiety symptoms in the short term, at this time, there is not sufficient evidence to determine the long-term benefits of these medications. Furthermore, there is some evidence showing that individuals who receive a combination of benzodiazepines and exposure treatment may be at greater risk for relapse compared with individuals receiving exposure alone. Coupled with concerns of a high risk of abuse, benzodiazepines may negatively impact the effects of exposure therapy, although additional research in this area is needed. With respect to standing-dose medications such as SSRIs, some prior research has suggested that pre-existing antidepressant use does not negatively impact CBT, while another study has suggested the opposite. Hypotheses as to how medications may interfere with exposure include that these medications

(especially benzodiazepines) can function as 'safety behaviors' (i.e., maladaptive coping behaviors) in social situations similar to use of alcohol or other substances [107,136], or that a patient who is starting to improve may attribute that improvement to the medication, which then may decrease their motivation for learning coping skills in psychotherapy [85]. However, more research in this area is needed to better determine how medications and CBT function together (both in the short term and the long term).

### *Alternative treatment strategies: self-help & guided self-help*

As described in the section on diagnosis, individuals may experience symptoms of SAD that do not meet 'threshold' levels for a diagnosis, yet these symptoms are problematic enough in which treatment may be beneficial. A cost–benefit analysis should be applied in this case when determining appropriate treatment options, such as evaluating whether the available treatments would provide sufficient benefit while keeping costs and risks low. Treatments that may be appropriate using these criteria include self-help and guided self-help. Recent studies have demonstrated the efficacy of internet-based self-help or guided self-help [87] and bibliotherapy self-help or guided self-help for individuals diagnosed with SAD [137,138]. No known studies have conducted cost–effectiveness analyses using these treatments for SAD specifically. One study conducted a secondary cost–effectiveness analysis on a prior trial comparing relaxation, a computer-based self-exposure program, and clinician-led exposure for panic and phobic disorders [139], finding that computer-aided self-exposure was more cost effective compared with clinician-led exposure even when it was supplemented with clinician input. Findings from these studies suggest that further research on these treatment approaches, especially for those with subthreshold levels of SAD, is warranted.

### *Does comorbidity impact treatment efficacy for SAD?*

Much of the existing literature suggests that the presence of comorbid depression has a detrimental effect on the treatment of SAD. For example, a study by Ledley *et al*. found that depression symptoms were associated with more severe SAD symptoms overall, less change in symptoms over the course of CBT for SAD and nonresponse to treatment [140]. However, one limitation is that they excluded patients who met full criteria for major depressive disorder (MDD). Another recent study by Marom *et al*. found that SAD patients with comorbid MDD improved at similar rates during the acute phase compared to SAD patients without MDD [141]. However, SAD patients with MDD experienced an increase in SAD severity during a 1-year follow-up period after treatment termination, while SAD patients without comorbid MDD experienced a further improvement in SAD symptoms during this same follow-up period. An older study by Erwin *et al.* found that although patients with SAD plus comorbid mood disorders improved at a similar rate during treatment compared with those with SAD alone and those with SAD plus other comorbid anxiety disorders, they experienced greater severity of SAD symptoms at pre- and post-treatment [142]. Contrary to Erwin *et al*. [142], Joorman *et al*. [143] found that patients with SAD and comorbid MDD did not differ from those without MDD on pre- or post-treatment SAD severity; however, they experienced elevated rates of depression at pre-treatment and throughout the course of treatment. A 12-week open trial of citalopram for comorbid SAD and MDD also found that while significant improvement occurred on symptoms of both disorders, improvement in SAD symptoms lagged behind improvement in MDD symptoms [144].

Some studies have examined the effect of other comorbidities on the treatment outcome of CBT for SAD, such as GAD [145] and AVPD [146]. However, most of these studies have found that individuals with and without these comorbid disorders improve at similar rates. In addition, there is some evidence to suggest the detrimental effect of comorbid substance use disorders on the treatment of SAD [147], although this needs to be studied further. Therefore, the presence of comorbid depression appears to be one of the most consistent challenges in the treatment of SAD.

### *Conclusions on treatment*

Controversies and issues in the treatment of SAD indicate that it is important to define the time frame that is being considered (i.e., short-term or long-term efficacy). In addition, efficacy also depends on the outcomes examined; for example, is symptom severity the only factor being considered or are other variables also being assessed, such as behavioral performance in social situations, quality of life and overall functioning? The research to date suggests that pharmacotherapy is effective in reducing severity of SAD symptoms in the short term, yet CBT is more effective in reducing symptoms and changing avoidance behaviors in the long term. As of yet there is not sufficient evidence to suggest that combined treatment is more efficacious than either monotherapy in general, especially in the long term. In addition, there is some evidence to suggest that medications could negatively impact the efficacy of CBT, at least in certain situations. The accumulation of data to date on benzodiazepines in particular suggests that the potential benefits of temporary decreases in symptoms do not outweigh the risks of abuse or relapse. More recent research efforts have been devoted to examining the efficacy of other compounds (e.g., DCS), in enhancing the exposure process. However, only a few studies have examined this in SAD specifically, and more recent studies in other anxiety disorders have produced mixed findings. Alternative treatment approaches such as self-help and guided self-help have received little attention thus far and should continue to be explored as potential treatment options, especially for individuals with subthreshold or less severe forms of SAD. Finally, evidence has shown that comorbid conditions (especially depression) can negatively impact the efficacy of treatment for SAD. Therefore, future research efforts should be directed toward determining better ways of treating SAD in the presence of comorbidity, and what types of treatments will be most efficacious in the long term.

## Expert commentary

Prior research has indicated that despite the increased awareness of SAD over recent decades and a potential for the overdiagnosis of normal shyness, the disorder also can go under-recognized in clinical populations relative to other mental health problems. Steps can be taken to improve the detection of SAD in those who are significantly affected by it through the use of structured or semi-structured diagnostic interviews (e.g., the Anxiety Disorders Interview Schedule [148]). However, it is understandable that these methods can be time consuming in routine clinical settings. Therefore, a viable alternative is to screen for SAD using empirically validated self-report measures, of which many are available (e.g., the Mini-Social Phobia Inventory [149]). With respect to the treatment of SAD, clearly more research needs to be conducted on treatments that will maximize efficacy. The mixed findings on combined treatment suggest that if this treatment approach is to be optimized, research needs to be devoted to examining better ways of integrating these two treatment modalities. Further research also needs to be directed toward treatments that have a focus of long-term improvement, target broader outcomes such as quality of life and functioning rather than symptom reduction alone, and are better able to address comorbidities that commonly occur with SAD. For example, transdiagnostic CBT approaches [150] may be helpful, as well as newer acceptance- and mindfulness-based approaches [151] that are designed to address underlying maladaptive processes (e.g., behavioral and emotional avoidance) and emphasize improving quality of life.

## Five-year view

It is hoped that the detection of SAD in clinical settings will improve, and advances will be made in facilitating initiation of treatment for these individuals, while balancing it with sensitivity toward not overdiagnosing it in individuals who display a more normal variation of social anxiety. With respect to treatment, there has been a 400% increase in prescription of antidepressant medication over recent years [152]. This raises concerns that there is too large of an emphasis being placed on pharmacological treatments that address symptoms in the short term and that important long-term psychosocial factors (e.g., functioning and quality of life) are being underappreciated. Therefore, it is hoped that in the future psychotherapeutic interventions (e.g., acceptance-based behavioral treatments) will continue to be adapted to place a greater emphasis on improving functioning in the long term rather than simply reducing symptoms in the short term, and to address common comorbidities that may decrease the efficacy of treatment for SAD.

## Key issues

- Social anxiety disorder (SAD) is defined as experiencing significant anxiety and fear of embarrassment or humiliation in social or performance situations, to the point at which it causes significant impairment in functioning or distress.
- Despite decades of research no clear biological etiology of SAD has been identified, just as with other psychiatric disorders. On the basis of the current evidence, it is likely that its etiology is based on a combination of dispositional and environmental factors, in which a genetic predisposition may be a contributing factor, but its expression is heavily dictated by person-specific environmental factors.
- Although much of the research to date suggests that SAD is underdiagnosed, controversy remains as to the degree to which it is being overdiagnosed and the temperament of shyness is being overpathologized. Nevertheless, even researchers who believe that it may be overdiagnosed agree that at least a percentage of individuals have significantly impairing SAD, and those with subthreshold levels of social anxiety could still benefit from treatment.
- Researchers also continue to disagree as to whether SAD and avoidant personality disorder (AVPD) are two distinct disorders or two points along a broader continuum (with AVPD hypothesized as being a more severe variant of SAD).
- Another challenge in the diagnosis of SAD includes its under-recognition, possibly due to lack of proper assessment; the presence of comorbidity that may be more acute and therefore more of a focus of treatment; and the nature of the disorder (e.g., embarrassment about discussing anxiety problems or believing that it is a personality characteristic that cannot be changed).
- Due at least in part to the under-recognition of SAD, evidence suggests that it also is being undertreated. This undertreatment may be due to the presence of other comorbidities that often take precedence in clinical settings (e.g., depression).
- The question of which type of treatment is best for SAD (pharmacotherapy vs psychotherapy, particularly cognitive behavior therapy [CBT]) continues to be examined. The current literature suggests that both the types of treatment are efficacious in the short term, but CBT tends to show greater benefits in the long term.
- It has been assumed that combined treatment (pharmacotherapy plus CBT) would produce the best outcomes for SAD. However, findings thus far have been mixed, with some studies showing that patients receiving combined treatment fared worse in the long term compared with those who had received CBT alone.
- Benzodiazepines continue to be commonly prescribed for individuals with SAD, despite recommendations discouraging their use. There is some evidence to suggest that benzodiazepines may interfere with the exposure process in the treatment of SAD, but more research is needed in this area. It also has been well documented that these medications have a high risk for abuse, as well as a high risk for relapse once they are discontinued.
- There is a need for future research to continue to examine: how recognition of the disorder can be improved; how (or if) combination treatment of pharmacotherapy plus CBT can be delivered in a more complementary fashion; and how psychotherapeutic interventions can be adapted to address the common comorbidities that occur with SAD and impact treatment efficacy.

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Medscape EDUCATION

# Issues and controversies surrounding the diagnosis and treatment of social anxiety disorder

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**Activity Evaluation**
Where 1 is strongly disagree and 5 is strongly agree

| | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| 1. The activity supported the learning objectives. | | | | | |
| 2. The material was organized clearly for learning to occur. | | | | | |
| 3. The content learned from this activity will impact my practice. | | | | | |
| 4. The activity was presented objectively and free of commercial bias. | | | | | |

1. Your patient is a 15-year-old male with shyness thought possibly to be a manifestation of social anxiety disorder (SAD). Based on the review by Dr. Dalrymple, which of the following statements about the clinical characteristics and epidemiology of SAD is **most likely** correct?

- ☐ **A** Situations feared or avoided by individuals with SAD include only performance situations
- ☐ **B** Patients with SAD always avoid all triggering situations
- ☐ **C** There are 2 subtypes of SAD: generalized and specific
- ☐ **D** SAD is a rare disorder

2. Based on the review by Dr. Dalrymple, which of the following statements about challenges in the diagnosis of SAD regarding the patient described in question 1 is **most likely** correct?

- ☐ **A** Patients with SAD typically have no other comorbidities
- ☐ **B** The underlying pathophysiology of SAD is clear, which facilitates diagnosis
- ☐ **C** SAD and avoidant personality disorder (AVPD) have been definitively proven to be 2 distinct disorders
- ☐ **D** Much evidence suggests that SAD is underdiagnosed, but the degree to which it is being overdiagnosed and the temperament of shyness is being over-pathologized remain controversial

3. Based on the review by Dr. Dalrymple, which of the following statements about challenges in the management of SAD would **most likely** be correct?

- ☐ **A** Pharmacotherapy is more effective than cognitive-behavioral therapy (CBT) in the long term
- ☐ **B** SAD tends to be overtreated
- ☐ **C** It has been proven that pharmacotherapy plus CBT produces the best outcomes
- ☐ **D** Benzodiazepines are often prescribed for individuals with SAD, despite recommendations discouraging their use