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The Latent Structure of Social Anxiety Disorder: Consequences of Shifting to a Dimensional Diagnosis

Ayelet Meron Ruscio

Department of Psychology, University of Pennsylvania.

Abstract

Despite longstanding debate over the nature of the boundary between social anxiety disorder (SAD) and less severe social anxiety, no study has tested directly whether the defining features of the disorder correspond to a latent category or dimension. The present study examined this question using data from the National Comorbidity Survey Replication (NCS-R), a nationally representative survey of the U.S. household population. Indicators representing the *DSM-IV* criteria for SAD were submitted to taxometric analyses in a subsample of adults ($n = 2,166$) who reported excessive social fear in their lifetime. Multiple taxometric procedures and consistency tests converged on a dimensional solution, suggesting that SAD is continuous with milder social anxiety. In follow-up analyses, a dimensional SAD diagnosis outperformed the *DSM-IV* diagnosis in predicting the subsequent onset of a range of clinically important outcomes. Large differences in associations with comorbid mood disorders, suicidality, and treatment-seeking in particular favored the prognostic value of dimensional over categorical diagnosis. These findings support the validity and potential utility of a dimensional conceptualization of SAD that may inform efforts to revise the diagnosis for *DSM-V*.

Keywords

social anxiety disorder; social phobia; taxometrics; dimensional diagnosis; diagnostic utility

Social anxiety has long been recognized as a common, even normative, human experience (Gilbert, 2001; McNeil, 2001; Widiger, 2005). Population surveys find that upwards of 60% of adults report elevated levels of social fear (Stein, Walker, & Forde, 1994) and that roughly one-quarter to one-third of adults report intense anxiety or avoidance of social situations at some time in their lives (Kessler, Stein, & Berglund, 1998; A. M. Ruscio et al., 2008). The pervasiveness of social anxiety has led to spirited debate over how best to distinguish normal social fear from social anxiety disorder (SAD), or social phobia. The *Diagnostic and Statistical Manual of Mental Disorders, 4th edition (DSM-IV; APA, 1994)* makes this distinction by requiring social fear to be persistent, excessive, accompanied by avoidance or intense anxiety, and associated with significant distress or impairment in order for SAD to be diagnosed. Notably, only a minority of people who experience strong fear in social situations meet the diagnostic criteria for SAD (Furmark, 2002; Merikangas, Avenevoli, Acharyya, & Angst, 2002; Wittchen & Fehm, 2003).

Correspondence concerning this article should be addressed to Ayelet Meron Ruscio, Department of Psychology, University of Pennsylvania, 3720 Walnut Street, Philadelphia, PA 19104. ruscio@psych.upenn.edu.

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SAD is represented by *DSM-IV* as a discrete condition and consequently is studied, assessed, and treated categorically in most settings. Many theorists have wondered, however, whether SAD may be better understood as the upper extreme of a continuum (Furmark, 2002) or spectrum (Merikangas et al., 2002; Schneier, Blanco, Antia, & Liebowitz, 2002) of social anxiety. A number of research findings seem consistent with this alternate conceptualization. First, functional impairment and treatment-seeking increase monotonically with increasing numbers of social fears and with more stringent definitions of SAD (Merikangas et al., 2002; A. M. Ruscio et al., 2008; Stein, Torgrud, & Walker, 2000). Second, even minor adjustments to the diagnostic criteria, particularly to the level of impairment required for a diagnosis, have a large impact on the prevalence of SAD (Davidson, Hughes, George, & Blazer, 1994; Pélassolo, André, Moutard-Martin, Wittchen, & Lépine, 2000; Stein et al., 1994; Wittchen, Nelson, & Lachner, 1998). Third, although social anxiety is highly persistent, this may be accounted for less by the stability of the diagnosis than by shifts between the full syndrome and subthreshold presentations over time (Merikangas et al., 2002). Finally, subthreshold SAD is far more similar to diagnosed SAD than to normal controls with respect to family history of SAD (Merikangas et al., 2002) and to indices of functioning such as educational attainment, income, social support, and health services utilization (Davidson et al., 1994). Importantly, subthreshold cases have reported comparable disability due to social anxiety as those with SAD (Wittchen, Fuetsch, Sonntag, Muller, & Liebowitz, 2000) and in one study accounted for 61% of those seeking treatment for social anxiety (Merikangas et al., 2002).

Results like these have led some authors to suggest that the diagnostic threshold for SAD is arbitrary (Stein et al., 2000) and that, rather than representing a discrete disorder, SAD exists along a severity continuum comprising subclinical social fears, nongeneralized and generalized SAD, and perhaps avoidant personality disorder as well (Hofmann & Roth, 1996; Herbert, Hope, & Bellack, 1992; McNeil, 2001; Rapee & Heimberg, 1997; Rettew, 2000; Turner, Beidel, & Townsley, 1992). However, because apparent continuity at the level of measured scores need not correspond to continuity at the latent level (Murphy, 1964; J. Ruscio, Haslam, & Ruscio, 2006), the possibility remains that SAD differs qualitatively from milder social fears. Adjudicating between these possibilities requires evaluation of the underlying structure of SAD using statistical approaches appropriate for distinguishing latent groups from dimensions. Of the existing approaches, Meehl's taxometric method (Meehl, 1995, 1999; Waller & Meehl, 1998) has arguably the strongest evidentiary base for making this distinction (see J. Ruscio et al., 2006, for a review). Responding to calls to test empirically, on a disorder-by-disorder basis, the latent structure of mental disorders (Meehl, 1995), researchers are increasingly using the taxometric method to distinguish categories from continua in psychopathology (see Haslam, 2007, for a review).

One published study to date used the taxometric method to evaluate the latent structure of social anxiety (Kollman, Brown, Liverant, & Hofmann, 2006). More than 2,000 outpatients with anxiety and mood disorders were asked to rate, on interview and questionnaire measures, the amount of fear that they experience in different social situations involving assertiveness, dating, public speaking, authority interaction, and social interaction. Kollman and colleagues submitted these ratings to taxometric analyses and arrived at a dimensional conclusion. Several notable strengths of the study bolster this conclusion, including the size and clinical characteristics of the sample, the quality of the measures, and the range of analyses performed. However, the focus of analysis on the situations in which fear is experienced, rather than on the diagnostic features of SAD, suggests that the results reflect the structure of social fear rather than the much narrower construct of SAD. Evaluating the structure of SAD requires that social fear be analyzed together with additional defining features of the disorder, including fear of negative evaluation, reactions when exposed to social situations, behavioral avoidance, and associated distress and impairment. Taxometric

analysis of such variables represents a logical extension of past work and an important step in informing the forthcoming revision of the SAD diagnosis for *DSM-V*.

Evaluation of latent structure may not be enough, however, to motivate diagnostic revision. While the *DSM* serves many purposes, its foremost purpose is to function as a useful clinical tool (APA, 2000). The adoption of a proposed diagnostic modification, especially one that deviates markedly from current practice, consequently may hinge on demonstrating that clinical utility would improve if the diagnosis were revised. For example, if SAD is dimensional in nature, shifting to a dimensional diagnosis that distinguishes among differing levels of social anxiety could improve prediction of clinically significant outcomes (cf. Cohen, 1983). In contrast, there may be little improvement if the current categorical diagnosis offers a reasonable shorthand measure of the underlying dimension. Prediction may even worsen if SAD is categorical in nature, as dimensional assessment may simply add measurement error when cases are distinguished only by group divisions (J. Ruscio & Ruscio, 2002). As dimensional and categorical models are favored under different conditions (DeCoster, Iselin, & Gallucci, 2009; Grove, 1991; J. Ruscio & Ruscio, 2002), the benefit of a given diagnostic change is an empirical question that may best be resolved through side-by-side comparisons of competing diagnostic models (Ruscio, 2008). A compelling argument for change could be made if a diagnosis reflecting latent structure improves substantially on the current diagnosis in predicting outcomes important for clinical planning and care.

The present study examined the latent structure of SAD using the taxometric method. Data from a nationally representative household survey of U.S. adults were submitted to multiple taxometric procedures and consistency tests to arrive at a structural solution. Follow-up analyses compared a diagnosis based on this structural solution with the *DSM-IV* diagnosis on the ability to predict a range of clinically relevant outcomes.

Method

Data Source and Measure

Data were drawn from the National Comorbidity Survey Replication (NCS-R), a nationally representative mental health survey of the U.S. household population (Kessler & Merikangas, 2004). The survey was fielded from February 2001 to December 2003 and had a response rate of 70.9%. Adults ages 18 years and older were selected to participate using a multi-stage clustered area probability sampling design. They were mailed a letter and study fact brochure, then visited by a professional lay interviewer who obtained verbal informed consent and conducted the face-to-face interview. Respondents received \$50 for their participation.

SAD was assessed by Version 3.0 of the Composite International Diagnostic Interview (CIDI 3.0; Kessler & Üstün, 2004), a fully structured interview. A sizable literature supports the reliability and validity of diagnostic decisions yielded by the CIDI (see Andrews & Peters, 1998; Haro et al., 2006; Wittchen, 1994). Participants were assessed for SAD if they reported on diagnostic stem questions that, at some time in their lives, they had (a) felt very afraid or shy with people or when having to do something in front of a group, (b) thought the fear was excessive, and (c) either avoided or experienced substantial distress in the feared situation. The CIDI Social Phobia section asked about lifetime experiences of shyness, fear, or discomfort in each of 14 performance and interactional situations. Subsequent questions assessed for lifetime and 12-month *DSM-IV* SAD.

Of the initial sample of 9,832 participants, 2,166 (22%) endorsed the stem questions described above and completed the SAD assessment. This subsample was predominantly

female (58%) and Caucasian (73%) and ranged in age from 18 to 94 ($M = 42.36$, $SD = 15.33$). Most participants were employed (71%) and married or cohabitating with a partner (55%) at the time of the survey. Close to half (51%) of the subsample met *DSM-IV* criteria for lifetime SAD, suggesting a moderate base rate of a putative SAD taxon that was well-suited for taxometric analysis (J. Ruscio et al., 2006). As participants with milder social fears were excluded from this subsample, the latent structure of less severe social anxiety was not examined here.

Indicator Selection and Construction

In keeping with dominant conceptualizations of SAD as a lifetime-persistent condition with early temperamental precursors, an early age of onset, and an unremitting course (APA, 2000; Kagan, 1997; A. M. Ruscio et al., 2008; Wittchen & Fehm, 2003), indicators were constructed to measure lifetime SAD. Candidate items were selected to achieve (a) good coverage of the SAD construct; (b) high correlations with the *DSM-IV* SAD diagnosis, to maximize indicator validity; and (c) higher correlations among items within indicators than across indicators, to minimize indicator redundancy and nuisance covariance. Items assessing theoretically related facets of SAD were standardized and then combined into five composite indicators to enhance reliability and measurement variability.

Indicator 1 reflected the pervasiveness of social anxiety, operationalized by the number of social situations feared out of the 14 situations assessed. Consistent with Criterion A for SAD, these situations encompassed a wide range of interactional and performance fears involving exposure to unfamiliar people or to possible scrutiny by others (the complete list of situations appears at www.hcp.med.harvard.edu/ncs). Several studies have found the number of social fears to be an excellent indicator of SAD severity and a strong predictor of impairment among those with the disorder (Furmark, Tillfors, Stattin, Ekselius, & Fredrikson, 2000; A. M. Ruscio et al., 2008; Stein et al., 2000). *Indicator 2* represented fear of negative evaluation. Constituent items focused on thoughts experienced in feared social situations, including fears of doing something embarrassing or humiliating; fears that others might look at, talk about, or think negative things about the respondent; and fears of being the focus of attention. *Indicator 3* assessed bodily sensations and related concerns experienced in feared social situations. These included physical symptoms associated with social anxiety (e.g., blushing or shaking, feared loss of control over bowels or bladder), panic attacks or panic symptoms experienced in social situations, and fears of becoming trapped or unable to escape the social situation. *Indicator 4* reflected the impact of social fear on the respondent's life. It included avoidance of social situations, distress caused by social fear or avoidance, and impairment caused by social fear or avoidance in work, social life, or personal relationships. *Indicator 5* assessed the persistence of social anxiety over time, operationalized as the recency of strong social fear or avoidance. Persistence was included in the indicator set to capture the Criterion A requirement of persistent fear and to reflect theoretical and empirical depictions of SAD as a chronic disorder (e.g., Keller, 2003; Wittchen & Fehm, 2003). Together, the five indicators provided good coverage of four of the five *DSM-IV* criteria for SAD. The fifth criterion (Criterion C) of excessive or unreasonable fear was required for entry into the CIDI Social Phobia section and consequently was met by all participants included in the analyses.

Indicator score distributions ranged from a low of four discrete values (Indicator 2) to a high of 20 values (Indicator 4). Using the base rate classification method (J. Ruscio, 2009a), groups were formed to estimate indicator parameters prior to analysis. The average nuisance covariance ($r = .08$ and $.04$ in the taxon and complement, respectively) and indicator skew (-0.33) were highly favorable for taxometric analysis, whereas the average indicator validity ($d = 1.20$) was rather low. To facilitate accurate conclusions, sample-specific

comparison data were generated and analyzed alongside the research data in each taxometric analysis (see *Taxometric Analyses* below).

Outcome Variables

Eleven variables drawn from three domains—comorbid disorders, suicidality, and treatment-seeking—served as outcomes in predictive analyses. Comorbid disorders included any lifetime *DSM-IV* anxiety disorder (panic disorder, agoraphobia without panic, specific phobia, generalized anxiety disorder, posttraumatic stress disorder, separation anxiety disorder), mood disorder (major depressive disorder, dysthymic disorder, bipolar spectrum), impulse-control disorder (attention-deficit/hyperactivity disorder, oppositional-defiant disorder, conduct disorder, intermittent explosive disorder), or substance use disorder (alcohol and illicit drug abuse and dependence), as well as any lifetime comorbid disorder out of the 17 listed above. Diagnoses were based on CIDI 3.0 assessments and have been shown to have generally good concordance with diagnoses made by blind clinical interviewers (Haro et al., 2006) using the Structured Clinical Interview for *DSM-IV* (SCID; First et al., 2002), with the exception of impulse-control disorders which were not validated due to the unavailability of a gold standard clinical assessment for adults.

Suicidality was represented by two dichotomous variables, suicidal ideation (operationalized as ever thinking seriously about committing suicide) and suicide attempt. Treatment-seeking included use of psychotherapy or counseling services, use of psychotropic medications, any mental health treatment by a professional, and any treatment sought specifically for social anxiety.

Taxometric Analyses

The five indicator variables were submitted to taxometric analyses to test whether SAD is taxonic (categorical) or dimensional in nature. The taxometric method is noteworthy for its emphasis on consistency testing rather than significance testing, with confidence in a structural solution increasing as findings converge across analyses. In the present study, consistency was evaluated across multiple taxometric procedures: MAMBAC (Mean Above Minus Below a Cut; Meehl & Yonce, 1994), MAXEIG (MAXimum EIGenvaue; Waller & Meehl, 1998), and L-Mode (Latent Mode; Waller & Meehl, 1998). MAMBAC and MAXEIG were also performed multiple times, varying the combination and position of the indicators in the analyses, to provide further checks of consistency. Detailed descriptions of these taxometric procedures are available elsewhere (Meehl & Yonce, 1994; J. Ruscio et al., 2006; Waller & Meehl, 1998).

The primary output of taxometric analyses are graphed curves that are visually inspected for characteristic signs of taxonic and dimensional structure. Because various data parameters and analytic decisions influence the appearance of taxometric curves, accurate interpretation may be facilitated by generating taxonic and dimensional comparison data for parallel analysis. J. Ruscio and colleagues (J. Ruscio, 2007; J. Ruscio & Kaczetow, 2009; J. Ruscio & Marcus, 2007; J. Ruscio, Ruscio, & Meron, 2007) have demonstrated the utility of simulating comparison data of known latent structure that reproduce the distributional and correlational features of one's research data. Research data and comparison data are submitted to identical analyses, and results are compared to judge whether the output yielded by the research data more closely resembles that of the taxonic or dimensional comparison data. Comparison is aided by a comparison curve fit index (CCFI; J. Ruscio et al., 2007; J. Ruscio & Kaczetow, 2009; J. Ruscio & Walters, in press) which calculates the fit of the research curve to the average curve yielded by the simulated taxonic and dimensional data, respectively. The CCFI ranges from 0 to 1, with a value of .50 representing equally good fit of taxonic and dimensional structure and progressively more

extreme values providing increasingly stronger support for dimensional (closer to 0) or taxonic (closer to 1) structure. To generate taxonic comparison data in the present study, cases were assigned to putative groups via the base rate classification method (J. Ruscio, 2009a) using the mean base rate estimated from taxometric analyses of the research data.

Taxometric analyses and data simulations were carried out using J. Ruscio's (2009b) suite of taxometric programs, which includes the comparison data generation program developed by J. Ruscio and Kaczetow (2008). The analysis proceeded in four steps. First, taxometric analyses were performed on the research data to obtain base rate estimates of the putative taxon. Second, the mean base rate estimate yielded by the research data was used to generate populations of taxonic and dimensional comparison data from which 10 samples of each structure were drawn for each analysis. Third, taxometric analyses were performed on the comparison data sets, and implementation settings were identified which most powerfully differentiated the results yielded by taxonic and dimensional data. Fourth, the research data were reanalyzed using these final implementation settings. Results of the research data were compared with those of the comparison data to facilitate accurate interpretation.

Predictive Analyses

Follow-up analyses were performed to evaluate the practical implications of structural results yielded by taxometric analyses. Analyses were carried out using the SAS Proc Genmod procedure, which adjusted for the weighting and clustering inherent in the NCS-R sample design. These analyses consequently were restricted to the 1,925 of the 2,166 participants for whom a final sample weight was available.

A structure-based dimensional diagnosis of SAD, constructed by standardizing and summing the five indicators, was compared with *DSM-IV* SAD in its ability to predict a range of subsequent functional outcomes. All models included age to adjust for possible age cohort effects. For each outcome, the two diagnoses were compared using the seemingly unrelated regression procedure (SUR; e.g., Rochon, 1996; Zeller, 1962) in the context of discrete-time survival logistic models (e.g., Efron, 1988) fitted with generalized estimating equations. Person-year was the unit of analysis, and all variables were treated as time-varying. Associations with the onset of each outcome were estimated separately for *DSM-IV* and dimensional SAD. Temporally primary SAD was used to predict the onset of temporally secondary outcomes, with temporal order determined from retrospective age-of-onset reports. To permit direct comparison between diagnoses, survival coefficients were transformed to standardized beta coefficients and interpreted as log odds ratios. The difference between coefficients was then estimated and tested for statistical significance. To evaluate the size of the difference between coefficients for the two diagnoses, Cohen's *d* was calculated for each pair of log odds ratios. This calculation was based on the logistic latent variable underlying the categorical SAD diagnosis following the work of Mackinnon & Dwyer (1993) and was interpreted using Cohen's (1992) conventions for small (.20), medium (.50), and large (.80) effects.

Results

Taxometric Analyses

MAMBAC was performed using each variable in turn as the output indicator and the sum of the four remaining variables as the input indicator. For each indicator configuration, 50 equally spaced cuts were made along the input starting 25 cases from either end, and 10 internal replications (J. Ruscio et al., 2006) were performed to reduce the effects of arbitrarily cutting between cases with identical scores. All five MAMBAC curves had the characteristic concave shape associated with dimensional latent structure. The average of

these curves appears in the top panel of Figure 1, alongside the curves generated by parallel analyses performed in taxonic and dimensional comparison data sets. The research curve closely resembles the dimensional curves, and both are readily distinguished from the taxonic curves. The closer fit of the research data to the dimensional than the taxonic comparison data was reflected in the CCFI of .323.

MAXEIG was performed using standardized variables, with two variables at a time serving as output indicators and the remaining variables summed to form the input indicator. Analyses were performed using 35 windows that overlapped 90%, as this configuration best discriminated the taxonic and dimensional comparison curves and produced sufficient curve stability while still including enough points to reveal the full shape of the curve. To further stabilize the curves, 10 internal replications were performed in each analysis. None of the 10 MAXEIG curves yielded by the research data evidenced the peak indicative of taxonic structure (see Figure 1, middle panel). Instead, the average research curve closely matched the comparison dimensional curves and was easily distinguished from the comparison taxonic curves. The CCFI of .160 provided further, strong evidence of dimensional structure.

An L-Mode analysis performed on the five indicators yielded a unimodal curve, consistent with dimensional latent structure (see Figure 1, bottom panel). Unimodal curves were also yielded by analysis of dimensional comparison data, in contrast to the bimodal curves that emerged in analysis of taxonic comparison data. A dimensional interpretation was additionally supported by the CCFI of .209. For all three taxometric procedures, sensitivity analyses restricted to the three most valid indicator variables¹ and to past-year cases of social anxiety² upheld this dimensional interpretation.

Predictive Analyses

Discrete-time survival analyses were used to compare the predictive validity of the *DSM-IV* SAD diagnosis with a structure-based, dimensional diagnosis reflecting severity of SAD. The dimensional diagnosis shared a larger association with 10 of the 11 outcomes considered here, significantly so for six of these outcomes (see Table 1). Compared with *DSM-IV* SAD, dimensional SAD was a better predictor of the subsequent onset of suicidal ideation (Wald $\chi^2(1) = 18.92, p < .001$) and suicide attempt (Wald $\chi^2(1) = 16.76, p < .001$). It also more powerfully predicted the subsequent onset of a mood disorder (Wald $\chi^2(1) = 19.02, p < .001$), but not a non-mood disorder (all Wald $\chi^2 < 0.86$, all $p > .356$), than the *DSM-IV* diagnosis. Finally, dimensional SAD was more strongly associated with subsequent

¹Although taxometric analyses consistently pointed to a dimensional conclusion, this conclusion was tempered by lower-than-desirable correlations and validity estimates for some indicators. Importantly, the finding that taxonic and dimensional comparison data, simulated to closely match the research data, produced clearly distinguishable taxometric results supported the adequacy of the indicators to distinguish these competing structures. To further test whether the data were sufficiently powerful to detect a taxon, had one existed, a sensitivity analysis was performed by replicating all analyses using the three most valid indicators (detailed results available on request). Indicators 2 and 3 were combined into a single composite with higher estimated validity than either indicator alone. Indicator 5 was excluded given its less central role in the diagnosis of SAD than the other indicators. The three resulting indicators (range of social situations feared, intensity of cognitive/somatic fear reaction in social situations, life impact of social fears) were submitted to MAMBAC, MAXEIG, and L-Mode analyses performed as described above. All analyses yielded clear evidence for dimensional structure, including CCFI values in the range .233–.323, increasing confidence in this structural solution.

²To determine whether similar results would be obtained with data that were less susceptible to retrospective recall bias, taxometric analyses were performed within the subsample ($n = 1,006$) of respondents who reported social anxiety during the past year. Notably, approximately half ($n = 542$) of this subsample met *DSM-IV* criteria for 12-month SAD. Two new indicators were constructed focusing specifically on past-year symptoms, one assessing social fear and avoidance and the other assessing functional impairment due to social anxiety. Two additional indicators were drawn from the lifetime assessment to characterize the fears of the 12-month subsample and to increase the range of taxometric procedures that could be performed; they assessed the number of feared social situations and the intensity of the cognitive and somatic fear reaction in social situations. Taxometric analyses were performed in the 12-month subsample with all four indicators and with just the two indicators that asked specifically about past-year symptoms (detailed results available on request). All results, including the CCFI values of .228–.426, were consistent with the earlier dimensional conclusion.

treatment-seeking, both pharmacological (Wald $\chi^2(1) = 44.28, p < .001$) and psychotherapeutic (Wald $\chi^2(1) = 13.83, p < .001$), than was *DSM-IV* SAD. Importantly, the association held for all professional mental health treatment (Wald $\chi^2(1) = 20.14, p < .001$) rather than for treatment specifically for social anxiety (Wald $\chi^2(1) = 0.20, p = .656$).

Cohen's d provides a metric for quantifying the amount of improvement in prediction that would be achieved by using the dimensional diagnosis in place of the *DSM-IV* diagnosis. Differences between the log odds ratios for the two diagnoses varied widely for the outcomes considered here, from a low of $d = .01$ for a subsequent anxiety disorder to a high of $d = 4.71$ for a subsequent suicide attempt. There was a large difference, though, for six of these outcomes, with the dimensional diagnosis a far stronger predictor than the *DSM-IV* diagnosis of comorbid mood pathology, suicidality, and treatment-seeking following the onset of social anxiety.

Discussion

The present study evaluated the latent structure of SAD in a general population sample. Indicators characterizing the defining features of the *DSM-IV* disorder were analyzed in a subsample of respondents who reported lifetime social anxiety. Multiple taxometric procedures and parallel analysis of simulated comparison data converged on a dimensional solution, suggesting that SAD exists on a continuum with less severe social anxiety. In follow-up analyses evaluating the practical implications of these findings, a dimensional SAD diagnosis outperformed the categorical *DSM-IV* diagnosis in predicting a range of clinically important outcomes, with several improvements in prediction corresponding to differences that were large as well as statistically significant.

These results are consistent with prior studies in which increasingly severe social anxiety was found to be associated with increasingly severe impairments in functioning, with no apparent threshold between syndromal and subsyndromal SAD at the manifest level. The absence of such a threshold at the latent level provides further evidence that continuity is not merely an artifact of the measurement scales or analytic approaches investigators chose to use, but an intrinsic property of SAD as it is currently defined. This suggests that adopting a dimensional conceptualization of SAD would advance efforts to understand the origins of the disorder by narrowing the range of plausible etiological models to those involving additive, graded causes (Haslam, 1997; J. Ruscio et al., 2006) rather than discrete, all-or-none causal processes. Along these lines, recent taxometric research has shown that fear of evaluation, a core cognitive feature and presumed risk factor for SAD (Bögels & Stein, 2009), has a dimensional latent structure (Weeks, Norton, & Heimberg, 2009). Future research would profit from considering how a range of potential causal factors that are themselves distributed along gradients of severity combine to produce the symptom profile characteristic of SAD.

The present findings also underscore the conceptual importance of subsyndromal social anxiety. Together with prior findings of widespread movement into and out of the SAD diagnosis over time (Merikangas et al., 2002) and of substantial disability and treatment-seeking among subsyndromal cases (Davidson et al., 1994; Merikangas et al., 2002; Wittchen et al., 2000), they suggest that important information is missed when individuals falling below the diagnostic threshold are ignored. Clinically, subsyndromal symptoms may be significant because they identify prodromal or residual cases that require monitoring to prevent emergence of the full syndrome, because they influence the clinical course or approach to treatment for other disorders, or because they are distressing or disabling in their own right. For any of these reasons, attending to subthreshold symptoms may promote accurate prognosis and effective treatment planning. To the extent that the dimensional

findings observed here apply to all levels of social anxiety, even levels falling well below the threshold of clinical significance may have considerable value for efforts to understand social anxiety. Research that includes not only diagnosed cases but also subclinical cases evidencing traits that are hypothesized to fall on the same spectrum as SAD (e.g., shyness, behavioral inhibition; McNeil, 2001; Schneier et al., 2002) may allow a more sensitive search for genes that contribute to the disorder (Stein, Jang, & Livesley, 2002; see also Dick et al., 2008) as well as for environmental and gene-by-environment interaction effects (Saudino, 2001). Assessing the full social anxiety continuum may additionally increase power to detect causal relationships and to account for temporal fluctuations in symptoms (cf. Cohen, 1983). In contrast, parsing the continuum into “present” and “absent” categories may distort the course of social anxiety and mask potentially important differences between individuals that account for variance in outcomes.

The dimensional solution reported here is bolstered by its detection in a large, national sample containing a moderate base rate of the putative taxon; by indicators drawn from face-to-face interviews that provided good coverage of the SAD construct; by parallel analysis of simulated comparison data that aided in interpretation of results; and by consistency testing that spanned multiple taxometric procedures. Nevertheless, several limitations should be borne in mind when considering these taxometric results. First, interviews were fully structured and administered by lay interviewers. Although interviewers underwent extensive training and supervision (Kessler et al., 2004) and the reliability and validity of CIDI diagnoses are well-established (see Andrews & Peters, 1998; Wittchen, 1994), semi-structured clinical interviews remain the gold standard assessment for SAD, and replication using clinical ratings would increase confidence in the dimensional conclusion. Second, while the focus on lifetime symptoms is arguably appropriate for a disorder known to have a characteristically early onset and chronic, unremitting course, analysis of current symptoms might have yielded different results. Lifetime symptom assessment also raises the possibility of retrospective recall bias, a possibility only partially mitigated by safeguards in CIDI 3.0 to minimize recall bias (Kessler & Üstün, 2004) and by the replication of results among respondents with past-year symptoms. Research focusing on current symptoms is needed to address these concerns. Third, the CIDI assessment of SAD follows closely the *DSM-IV* criteria for the disorder. A valuable extension of this work would involve other measures of SAD that provide a more detailed assessment of core symptoms, assess additional hypothesized features (e.g., distorted self-appraisals, selective processing of social threat cues), and incorporate other modes of assessment (e.g., behavioral, physiological). Fourth, all participants in the analysis sample screened positive for a lifetime history of excessive social anxiety involving distress or avoidance. While only about half qualified for a lifetime diagnosis of SAD, differences between the putative taxon and complement groups on the indicators (and, in turn, the corresponding indicator validities) were smaller than if the complement had included people with milder social fears. Importantly, analysis of comparison data generated to match these research data revealed clear differences between taxonic and dimensional structures, suggesting that the indicators were capable of detecting a taxon had one existed in this sample. Nonetheless, the exclusion of individuals with low social anxiety from the sample left open the possibility of a taxonic boundary at less severe levels of anxiety than those included here. The present findings await replication in other, complementary data sets that encompass a broader range of social anxiety and include indicators that are clinically assessed, focused on current symptoms, and sensitive to differences at the lower end of the severity distribution.

Bearing these limitations in mind, the apparent dimensionality of SAD hinted that shifting from a categorical to a dimensional classification of cases would improve the predictive validity of the diagnosis. Results of follow-up analyses were consistent with this hypothesis. Compared with *DSM-IV* SAD, a dimensional diagnosis representing symptom severity was

more strongly associated with 10 of the 11 outcomes examined here, with six differences reaching statistical significance. Notably, these outcomes were constrained to occurrences following the onset of SAD, and so may be taken as support for the greater prognostic value of the dimensional diagnosis. The large gains in prediction observed for several of these clinically relevant outcomes suggest that a diagnosis reflecting the latent structure of SAD may be not only more valid, but also more useful, than the *DSM-IV* diagnosis.

In fact, the predictive validity of a dimensional SAD diagnosis is likely underestimated here. This is because the dimensional measure was restricted to interview items used in making the *DSM-IV* diagnosis, thereby allowing comparison of the diagnostic models (rather than the quality or quantity of information included in the models) but leaving less unique variance for the dimensional diagnosis to share with the outcomes. A dimensional measure encompassing a wider array of clinical features, weighting them according to their informational value, and combining them in a more sophisticated fashion (such as through item response theory; e.g., Embretson, 1996) presumably would have performed better. Developing such a measure represents a worthwhile next step for future work, not only to capture meaningful differences between cases that could aid research and practice, but to inform emergent discussions of how dimensions should be incorporated into *DSM-V* (see Krueger, Watson, & Barlow, 2005; Regier, 2007). Of course, the psychometric advantages of a more complex measure will need to be weighed against the logistical advantages of a simpler measure that can be implemented more readily in routine practice settings. A further consideration will be where along the dimension thresholds should be drawn to guide categorical decisions that invariably arise in practice, as well as which disorder characteristics (e.g., severity of avoidance), associated concerns (e.g., social skills deficits), and functional consequences (e.g., social and occupational impairments) should be used to draw thresholds that will optimize these decisions (A. M. Ruscio, in press).

Ultimately, the widespread adoption of a dimensional SAD measure will depend substantially on its utility. The predictive analyses reported here offer one way of measuring the utility of a nosological change. This approach allowed competing diagnostic models to be compared directly on key outcomes, and differential prediction of these outcomes provides a compelling rationale for revising the SAD diagnosis. However, several outcomes of particular interest to clinicians were not available for this community sample, such as those pertaining to treatment selection (dose needed to achieve response, differential response to different treatments), treatment outcome (size and speed of response, rate of relapse), and clinical management (length of treatment, need for ancillary treatments). The analyses reported here should consequently be viewed as a first step requiring extension to other samples and outcomes. Whether the improved prediction that is demonstrated for any revision is clinically significant and worth the costs of change (First, 2005) is to some extent a value question that cannot be answered by data alone. However, given the far-reaching impact of the diagnostic nomenclature, this is a question that can and should be informed by data, with preference given to proposed revisions that enhance both the validity and utility of diagnosis.

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MAMBAC

MAXEIG

L-Mode

Figure 1.

Panels of MAMBAC, MAXEIG, and L-Mode curves for the SAD indicators. In each panel, the curve shown for the research data is the average of all individual curves for that taxometric procedure. The corresponding taxonic and dimensional comparison data plots include curves for each of 10 simulated data sets (dotted lines) as well as their average (solid line).

Table 1
Associations of *DSM-IV* and Dimensional Diagnoses of SAD with the Onset of Subsequent Clinical Outcomes

Outcome	Diagnosis		Wald χ^2	p	d
	<i>DSM-IV</i>	Dimensional			
Comorbid Disorders					
Anxiety disorder	2.03	2.05	0.00	.947	.01
Mood disorder	3.29	5.02	19.02	< .001	.95
Impulse-control disorder	3.38	3.26	0.07	.788	.07
Substance use disorder	3.04	3.71	0.85	.357	.37
Any comorbid disorder	1.23	1.37	0.56	.455	.08
Suicidality					
Suicidal ideation	5.71	9.09	18.92	< .001	1.86
Suicide attempt	11.18	19.72	16.76	< .001	4.71
Treatment-Seeking					
Psychotherapy/counseling	2.88	4.49	13.83	< .001	.89
Psychotropic medications	3.53	7.46	44.28	< .001	2.17
Any professional treatment ^a	2.30	3.86	20.14	< .001	.86
Social anxiety-specific treatment ^b	15.73	16.32	0.20	.656	.32

Note. Results are based on the seemingly unrelated regression procedure performed in the context of discrete-time survival models with person-year as the unit of analysis. The models adjusted for age (including linear, quadratic, cubic, and quartic effects). The associations of each social anxiety disorder (SAD) diagnosis with the onset of temporally secondary lifetime outcomes are expressed here as standardized beta coefficients and interpreted as log odds ratios. A 1 degree of freedom Wald χ^2 test compares the *DSM-IV* and dimensional SAD diagnoses; the corresponding *p* value indicates whether the two betas differ significantly from one another. The size of the difference between the betas is expressed as Cohen's *d* and interpreted using standard conventions for small (.20), medium (.50), and large (.80) effect sizes.

^aIncludes treatment by a professional for any mental health problem.

^bIncludes treatment sought specifically for social anxiety.