J Psychopathol Behav Assess
DOI 10.1007/s10862-006-9033-x

ORIGINAL PAPER

Onset of Illness and Developmental Factors in Social Anxiety Disorder: Preliminary Findings from a Retrospective Interview

Kristy L. Dalrymple · James D. Herbert · Brandon A. Gaudiano

© Springer Science+Business Media, Inc. 2006

Abstract Although many advances have been made in the treatment of Social Anxiety Disorder (SAD), less is known about its onset and factors related to its course and severity. The current study aimed to investigate developmental factors (e.g., onset of illness, behavioral inhibition, socially traumatic experiences) that research has suggested are related to the course and severity of SAD in a sample of adults diagnosed with generalized SAD. Results showed behavioral inhibition to be the only consistent predictor of current severity. Results for age of onset were consistent with previous studies suggesting an early childhood and later adolescent pattern. In addition, an earlier age of onset negatively impacted improvement in cognitive behavior therapy for SAD, but no other developmental factors were related to treatment outcome. Future research using longitudinal designs and multiple informants is needed to confirm findings from retrospective reports.

Keywords Social phobia · Onset of illness · Etiology · Development · Behavioral inhibition · Retrospective report

K. L. Dalrymple
Brown Medical School & Rhode Island Hospital,
Rhode Island, USA

J. D. Herbert
Drexel University,
Philadelphia, PA, USA

B. A. Gaudiano
Brown Medical School & Butler Hospital,
Rhode Island, USA

K. L. Dalrymple (✉)
Department of Psychiatry, Rhode Island Hospital,
235 Plain St., Suite 501, Providence, RI 02905, USA
e-mail: kristy_dalrymple@brown.edu.

Social Anxiety Disorder (SAD) is an excessive fear of social or performance situations in which embarrassment or humiliation may occur (American Psychiatric Association, 2000). SAD is the third most common psychiatric disorder in the United States, following major depression and alcohol dependence (Kessler, Berglund, Demler, Jin, & Walters, 2005), and typically follows a chronic and unremitting course without treatment (Juster & Heimberg, 1995; Reich et al., 1994). Some research indicates that the most frequent age of onset of SAD is in mid-adolescence (Schneier et al., 1992). However, recent research also suggests a bimodal pattern of onset, with some individuals reporting an onset before the age of 5 and others reporting onset in mid-adolescence (Juster, Brown, & Heimberg, 1996; Juster & Heimberg, 1995; Stein, Chavira, & Jang, 2001). This bimodal pattern may be a reflection of the two subtypes of SAD, with persons with generalized SAD (i.e., fear and avoidance of most social situations) tending to report an earlier onset associated with greater severity, and those with the specific subtype (i.e., fear and avoidance of one or two discrete social situations) reporting a later onset. The evidence is unclear as to whether differences exist between earlier and later onset groups on factors such as symptom severity and response to treatment (Stein et al., 2001).

In addition to age of onset, researchers have begun to examine other developmental factors related to the etiology and course of SAD. A frequently studied theoretical construct is behavioral inhibition (BI), defined as a temperamental style characterized by the tendency for children to display fear, avoidance, or quiet restraint in unfamiliar situations (Kagan, Reznick, & Snidman, 1988). Research has documented a relationship between BI and anxiety disorders in general (Turner, Beidel, & Wolff, 1996), as well as an association between BI and SAD specifically (Biederman et al. 2001; Schwartz, Snidman, & Kagan, 1999).

UNCORRECTED PROOF

Research also has examined the association between family variables and the etiology of SAD, such as parent sociability. Several retrospective studies have shown that individuals with SAD perceive their parents as having isolated them from social experiences and as being more avoidant of social situations themselves (Bruch & Heimberg, 1994; Bruch, Heimberg, Berger, & Collins, 1989; Rapee & Melville, 1997). Several studies also have examined the possible role of socially traumatic events in the development of SAD (e.g., Stemberger, Turner, Beidel, & Calhoun, 1995). A socially traumatic event refers to a social rejection experience, such as being humiliated during a class presentation. One study found that 58% of the sample recalled a socially traumatic event as having been related to the development of their social anxiety (Öst & Hughdahl, 1981). Some research suggests that these socially traumatic events are more clearly linked to the specific subtype of SAD (Stemberger et al., 1995).

Most conceptual models of SAD focus on proximal factors that maintain the disorder, such as various cognitive processes (Clark & Wells, 1995; Rapee & Heimberg, 1997). There is only one known conceptual model of developmental factors related to SAD. Morris (2001) describes possible pathways and entry points among factors such as temperament, family processes, peer relationships, performance inhibition, and social skills deficits. The model begins with a BI child who, because of inhibition, has a poor quality of interaction with parents that leads to poor attachment. The child thus has difficulty forming peer relationships, which leaves him/her with few opportunities to interact with others and leads to social skills deficits. This in turn increases the child's discomfort and inhibition in social situations, which results in further isolation, thereby establishing a vicious cycle.

These developmental factors have most often been studied independently, and few previous studies have examined their relative predictive power (Morris, 2001). Stemberger et al. (1995) conducted one of the few published studies that systematically examined the association between developmental and personality factors and SAD. Sixty-eight adults with specific or generalized SAD were compared with 25 non-clinical controls on family history of illness, childhood shyness, socially traumatic experiences, neuroticism, and extraversion. Results showed that socially traumatic experiences were associated with the specific subtype of SAD, whereas childhood shyness and lower extraversion were associated with the generalized subtype.

The current study attempted to expand on the Stemberger et al. (1995) study by examining developmental factors such as socially traumatic experiences and childhood shyness as they relate to severity of illness in SAD. Given the consistent differences between SAD subtypes in terms of severity and impairment, the current study examined these developmental variables within a homogeneous sample of adults diagnosed with generalized SAD. Furthermore, the current study included other potentially relevant developmental factors (e.g., BI, parent sociability) that research has suggested are associated with SAD and that are described in current conceptual models of SAD development (Morris, 2001). Finally, the current study expanded on Stemberger et al. by examining the relationship between these developmental factors and treatment outcome in a subsample of participants receiving cognitive behavior therapy (CBT) for SAD.

Therefore, the specific aims of the study were as follows: (1) To conduct an exploratory investigation of the relationship between earlier versus later onset of illness, BI, childhood shyness, socially traumatic experiences, parent sociability, and adult social anxiety severity; and (2) To examine the relationship between these developmental variables and treatment outcome following 12 sessions of CBT for SAD. Results from this study may help to inform a comprehensive, empirically-based developmental model of SAD. In addition, examination of the relationship between these developmental variables and treatment outcome may help in the refinement of existing treatments for SAD.

Method

Participants

Participants were 102 adults (54% female) recruited via community advertisements and professional referrals to participate in treatment outcome research. The sample ranged in age from 18 to 60 ($M = 34$, $SD = 11.5$), and was mostly Caucasian (62%). The majority of the sample had some college education or higher (44%), was employed full time (51%), and was single (67%). All participants met criteria for a primary diagnosis of SAD, generalized subtype. The generalized subtype was operationally defined as fear and avoidance of three or more social situations (Herbert et al., 2005).

Measures

Structured clinical interview for DSM-IV axis I disorders (SCID-I/P)

The SCID-I/P (First, Spitzer, Gibbon, & Williams, 1996) is a widely used semi-structured diagnostic interview for the major Axis I disorders and is based on *DSM-IV* (APA, 1994) criteria. Several studies have found that the SCID-I/P has moderate to high inter-rater reliability for most of the major mental disorders (Segal, Hersen, & Van Hasselt, 1994).

UNCORRECTED PROOF

Social Phobia and anxiety inventory (SPAI)

The SPAI (Turner, Beidel, Dancu, & Stanley, 1989) is a 45-item self-report measure that assesses clinical symptoms of SAD. The 32-item Social Phobia subscale (SPAI-SP) was used as it has been found to be a better index of social anxiety symptoms than the difference subscale score (Herbert, Bellack, & Hope, 1991). Psychometric research on the SPAI has indicated good test-retest reliability, internal consistency, and discriminant, concurrent, and external validity (Beidel, Bordon, Turner, & Jacob, 1989; Beidel, Turner, Stanley, & Dancu, 1989).

Beck depression inventory (BDI)

The BDI (Beck & Steer, 1987) is a 21-item self-report inventory assessing severity of depression symptoms. The BDI is one of the most widely used depression measures. Numerous studies have indicated that it possesses good reliability and validity in clinical and non-clinical samples (Beck, Steer, & Garbin, 1988).

Developmental social anxiety interview (D-SAI)

The D-SAI (Herbert, Goldstein, & Dalrymple, 2004) is a structured interview designed to assess relevant developmental factors that may be associated with social anxiety symptoms, as well as to track retrospectively symptom severity at various age points. The interview was created for this study as no validated assessment devices exist that assess the specific developmental variables of interest. Question content was formulated based on a review of the developmental literature in this area. The interview consists of 54 Likert-scale (range = 0–4) and 28 open-ended questions. Developmental factors and severity of social anxiety symptoms are assessed separately at the following age points: infancy (1st year of life), toddlerhood (1 to 3 years old), younger childhood (4 to 6), older childhood (7 to 11), younger teenager (12 to 15), older teenager (16 to 19), and currently.

Open-ended questions were coded by two raters into appropriate categories for data analysis. Categories were generated from a random sample of participant responses for these questions (based on the most common and frequent responses). The categories were reviewed by the second author (J.D.H.), and modifications were made to the categories based on his feedback. Inter-rater reliability was high in the coding of these open-ended questions ($\kappa = .90$).

The introductory section of the interview assesses demographic factors relevant to development (e.g. number of siblings), as well as an open-ended question assessing the individual's perception of the age of onset of SAD symptoms. In addition, participants are asked to rate the severity of their social anxiety symptoms on a scale from 0 to 100 for each of the age points described above, similar in concept to the Subjective Units of Distress Scale (SUDS; Wolpe & Lazarus, 1966). The interview consists of subsections of questions pertaining to each of the aforementioned age points. Each of these age-specific subsections includes a mixture of Likert-scale questions and open-ended questions. Some questions appear in nearly all age subsections (e.g., "As a (toddler, young child, etc.) I was shy"), whereas other questions are formulated to be appropriate for a particular age subsection (e.g., "As a young child, I was anxious during my first day of kindergarten"). The interview concludes with open-ended questions assessing parental characteristics (e.g., rearing practices, parent sociability), sibling relationships, family history of SAD, and perceived cause of SAD.

Goldstein et al. (1997) presented preliminary data derived from the interview from 15 adults diagnosed with generalized SAD according to the SCID-I/P. Results showed heterogeneity in symptom onset, with 50% reporting an onset in childhood and 50% in adolescence. In addition, results from the pilot study were consistent with previous research suggesting the relevance of developmental factors and SAD severity (e.g., Arrindell et al., 1989; Stemberger et al., 1995).

Procedure

All procedures were approved by the local Institutional Review Board. After an initial brief phone screening, individuals interested in participating in the larger treatment study were invited to the clinic for an evaluation by a diagnostician using the SCID-I/P. Diagnosticians were advanced doctoral students in clinical psychology trained to proficiency and reliability in the assessments. All diagnosticians were extensively trained by didactic materials, direct observation of assessments, and practice ratings of patient videotapes until reliability was obtained. Tapes of the diagnostic interviews were reviewed periodically to ensure diagnostic accuracy. New SCID-I/P assessments were reviewed weekly by the second author (J.D.H.), who has extensive experience in the assessment and treatment of SAD.

Epidemiological data indicate that SAD has high comorbidity with other Axis I disorders (Kessler et al., 2005). Therefore, participants in this study with comorbid diagnoses were included as long as their social anxiety was judged to be primary to and of greater severity than other Axis I diagnoses. Primacy of SAD was demonstrated by an earlier reported age of onset compared to other Axis I diagnoses, and severity was determined by the level of symptoms and the degree of impairment due to SAD compared to other co-occurring diagnoses. Inclusion criteria required participants to be between the ages of 18 and 60 and to have a primary diagnosis of generalized SAD. Exclusion criteria included a history of substance dependence within the past 6 months, mental retardation, pervasive

UNCORRECTED PROOF

developmental disorder, organic mental disorder, acute suicide potential, or previous participation in behavioral or cognitive behavioral therapy for SAD (as the current study was part of a larger treatment study).

After obtaining informed consent and administering diagnostic assessments, participants were interviewed using the D-SAI. Participants interested in pursuing treatment were then assigned to 12 sessions of cognitive-behavior therapy (CBT), either in group or individual format, and completed questionnaires at post-treatment. Detailed procedures used in the treatment studies are described in other publications (Herbert, Rheingold, et al., 2004; Herbert et al., 2005).

Data reduction and analytic strategies

Developing a new clinical measure was not the purpose of the current study. However, because the D-SAI was created specifically for this study in order to assess all developmental variables of interest, preliminary reliability and validity of the D-SAI severity scores were assessed by computing Cronbach's alpha coefficients and correlations with other validated symptom measures. As severity of SAD symptoms were of interest, the D-SAI was compared to the SPAI-SP, a well-validated measure of social anxiety severity (Heimberg & Becker, 2002). Internal consistency was analyzed as each of the age subsections were composed of multiple items. The relationship between the various developmental factors and SAD severity was investigated to determine their clinical relevance. A mean severity score was calculated for each age subsection as these sections contained a different total number of questions. For example, a mean score was calculated for 12 items in the older child age point. Examples of types of questions include: "As an older child I had friends come to my house to play" and "As an older child I was [not, slightly, moderately, very, or extremely] anxious while playing with friends." A repeated measures analysis of variance (ANOVA) was conducted on the age point severity scores to examine differences between those who reported an earlier onset (in childhood) of social anxiety symptoms compared to a later onset (in adolescence or adulthood). To increase confidence in the reliability of results derived from the D-SAI severity scores, a similar ANOVA was conducted between earlier and later onset for SUDS ratings.

In addition, multiple regression analyses were computed based on variables identified from the pilot study (Goldstein et al. (1997)) and other studies (Stemberger et al., 1995) that have examined developmental factors related to social anxiety. A stepwise approach (George & Mallery, 1999) was used to determine whether prediction of social anxiety severity could be improved by combining various developmental variables. Two primary regression analyses were conducted. The first regression used the D-SAI Current Severity Score as the criterion variable. However, to increase confidence in the reliability of results, participants' pre-treatment SPAI-SP scores were used in the second regression analysis. The SPAI-SP was chosen as it is one of the most well-validated and psychometrically sound measures of social anxiety severity (Heimberg & Becker, 2002) and because it was the primary outcome assessed in the clinical trials upon which the current study is based (Herbert, Rheingold, et al., 2004; Herbert et al., 2005).

Finally, analyses were conducted to examine the relationship between treatment outcome and the developmental factors. These analyses were conducted on the smaller subsample ($n = 41$) who completed CBT for SAD. Therefore, this analysis excluded those who dropped out of treatment, never started treatment, decided to pursue non-study treatments, failed to complete post-treatment assessments, etc. Repeated measures ANOVAs were computed for the categorical variables (socially traumatic experience, parent sociability, and onset) on pre- to post-treatment SPAI-SP scores. A Pearson correlation was computed between SPAI-SP change scores and the continuous variable BI. It was deemed statistically inappropriate to examine these variables in one combined analysis due to insufficient power because of the lower sample size for outcome analyses (Pedhazur, 1997). Sample sizes vary in some analyses where noted due to incomplete data.

Results

Preliminary reliability and validity of the D-SAI scores

Cronbach's alpha coefficients were calculated to determine internal consistency of the items for each of the age subsections: infancy, toddlerhood, younger childhood, older childhood, younger teenager, older teenager, and current age. Results indicated that reliability ranged from.69 to.86, with.76 the average across the age subsections. Coefficients of.60 or higher are considered adequate for research purposes (Nunnally, 1978).

A Pearson correlation was conducted between the D-SAI Current Severity Score and the SPAI-SP ($n = 85$) to evaluate convergent validity. Results revealed a significant, positive, and moderately strong association between the SPAI-SP and the D-SAI Current Severity Score ($r = .66$, $p < .01$). Discriminant validity was evaluated by comparing the D-SAI Current Severity Score and the BDI. The D-SAI Current Severity Score was only moderately correlated with the total BDI score ($r = .33$, $p < .01$). The magnitude of correlation between the D-SAI and the SPAI-SP was significantly greater than with the BDI (Fisher's $z = 3.74$, $p < .05$), supporting discriminant validity.

UNCORRECTED PROOF

Preliminary analyses

Participants were asked to report the age of onset of their social anxiety. This item was examined categorically, rather than continuously, as many participants could not report a specific age of onset. Responses to this open-ended question were coded into five categories: younger child (37%), older child (21%), adolescent (28%), adult (11%), and "don't know" (3%). Based on the above responses, participants were classified into earlier (in childhood; $n = 57$) or later onset (in adolescence or adulthood; $n = 39$) categories and analyses were conducted to examine differences between these groups. Adolescent and adult onsets were combined in the later onset category because so few participants reported an onset in adulthood.

Analyses indicated no significant differences between the onset groups on age, gender, race, education, employment, or marital status (all ps $> .05$). Preliminary analyses were conducted between the onset groups on the BDI and other developmental variables used in the analyses below: BI (infant to age 3), childhood shyness (ages 4–11), socially traumatic experiences, and parent sociability.[1] There were no significant differences between earlier and later onset for the BDI, socially traumatic experiences, and parent sociability (all ps $> .05$). However, results revealed a significant difference between the onset groups for BI ($t_{85} = 2.27$, $p < .05$) and childhood shyness ($t_{95} = 3.16$, $p < .01$), with the earlier onset group reporting greater BI and childhood shyness than the later onset group (see Table 1 for descriptive statistics).

Relationship between developmental variables and social anxiety symptoms

Course of illness

A 2 [earlier ($n = 52$) vs. later ($n = 38$) onset] by 6 (age points) repeated measures ANOVA on the D-SAI severity scores revealed a significant main effect for age ($F_{5,88} = 18.47$, $p < .001$), a significant main effect for group ($F_{1,88} = 13.61$, $p < .001$), but no significant interaction. There was a general increase across the age points in the severity scores, and those who reported an earlier onset also reported significantly greater symptom severity compared to the later onset group (see Fig. 1).

An ANOVA also was conducted on the SUDS Severity ratings (0–100) to examine reliability of the results obtained from the D-SAI severity scores. Results were similar, with SUDS scores increasing across the age points ($F_{6,53} = 69.48$, $p < .01$), and the earlier onset group reporting greater severity compared to the later onset group ($F_{1,53} = 21.82$, $p < .01$), but no significant interaction.

Current severity

A stepwise multiple regression analysis was conducted to examine developmental variables (earlier versus later onset, socially traumatic experiences, parent sociability, childhood shyness, and BI) associated with current severity, based on the D-SAI Current Severity Score ($n = 86$). Results showed that only BI ($\beta = .37$, $p < .01$) was associated with current severity of social anxiety symptoms based on the D-SAI ($F_{1,\ 82} = 12.95$, $p < .01$). Greater BI as a toddler was related to greater current social anxiety severity. This model accounted for 13.8% of the variance in current severity scores.

In an attempt to replicate results from the D-SAI Current Severity Score, a similar analysis was conducted using the same developmental variables to examine their association with current severity based on the SPAI-SP ($n = 70$). This regression also revealed only BI ($\beta = .27$, $p < .05$) as significantly associated with current severity based on the SPAI-SP ($F_{1,\ 66} = 4.99$, $p < .05$), such that greater BI as a toddler was related to greater current social anxiety severity. This model accounted for 7.1% of the variance in current severity scores (see Table 2 for regression statistics).

Relationship between developmental variables and treatment outcome

Analyses were conducted to examine the relationship between the developmental variables and treatment outcome.

[1] The childhood shyness variable was the sum of four Likert-scale questions assessing the degree to which the individual was shy at various points during childhood (from ages 4–11): 1) "When I was a younger child (ages 4–6), I was shy" (0-strongly disagree to 4-strongly agree); 2) "Compared to my peers, I was (0-much less to 4-much more) shy as other young children"; 3) "When I was an older child (ages 7–11), I was shy" (0-strongly disagree to 4-strongly agree); 4) "Compared to my peers, I was (0-much less to 4-much more) shy as other children my age." The socially traumatic experience subscale included one question: "At any point during your life did something ever happen to you that embarrassed you or humiliated you in front of people?" This item was coded into two categories, "yes" or "no." Based on the description of the event provided by the participant, raters determined whether the event qualified as a social rejection experience (e.g., others laughed at him/her during a class presentation). The parent sociability subscale consisted of one question: "Did your parents socialize a lot with friends or other family members, or did they mainly keep to themselves?" Responses were categorized into "yes, socialized with family or friends or both" and "no, did not socialize with family or friends." Only these two categories were used, as nearly all participants (86%) said that they either did or did not socialize with both friends and family. Finally, the behavioral inhibition variable was the sum of three Likert-scale questions taken from infancy and toddlerhood age points (up to 3 years old): 1) "I was a slow-to-warm-up baby, one who cried often but was easily soothed" (0-strongly disagree to 4-strongly agree); 2) "As a toddler I was very quiet and socially withdrawn around strangers" (0-strongly disagree to 4-strongly agree); 3) "As a toddler I was shy" (0-strongly disagree to 4-strongly agree).

UNCORRECTED PROOF

Table 1 Descriptive statistics for study variables

Study variable	Total sample *M* (*SD*)	Earlier onset ($n = 57$) *M* (*SD*)	Later onset ($n = 39$) *M* (*SD*)	Test statistic (*t* or *F*)	*p*
SPAI-SP (pre-treatment)	137.3 (29.7)	148.8 (22.9)	133.1 (31.2)	1.49	.14
BDI	12.7 (9.2)	12.7 (9.3)	13.3 (9.5)	−.33	.74
Childhood shyness	2.7 (.9)	2.9 (.8)	2.3 (.9)	3.16	.002
Behavioral inhibition	1.9 (.9)	2.0 (.9)	1.6 (.8)	2.27	.03
D-SAI severity scores					
Group main effect				13.61	<.001
Age points main effect				18.47	<.01
Interaction effect				0.59	.71
Toddlerhood	2.3 (1.0)	2.4 (1.0)	2.1 (.9)		
Younger child	2.0 (.7)	2.2 (.7)	1.8 (.7)		
Older child	2.2 (.7)	2.4 (.8)	2.0 (.6)		
Younger teenager	2.6 (.6)	2.8 (.6)	2.4 (.6)		
Older teenager	2.5 (.7)	2.7 (.7)	2.3 (.6)		
Currently	2.6 (.6)	2.7 (.6)	2.5 (.6)		
	n (%)	*n* (%)	*n* (%)	χ^2	
Parent sociability	74 (71.8)	44 (78.6)	27 (71.1)	.69	.41
Socially traumatic experience	78 (75.7)	46 (82.1)	27 (73.0)	1.11	.29

Note. BDI: Beck Depression Inventory; SPAI-SP: Social Phobia and Anxiety Inventory-Social Phobia Subscale; D-SAI: Developmental Social Anxiety Inventory. Parent Sociability: frequency and percentage of participants reporting that parents socialized with friends and family. Socially Traumatic Experience: frequency and percentage of participants reporting presence of a socially traumatic experience (e.g., humiliated during a class presentation). Test statistics were: *t* for the SPAI-SP, BDI, Childhood Shyness, and Behavioral Inhibition; *F* for the D-SAI Severity Scores; and χ^2 for Parent Sociability and Socially Traumatic Experience.

UNCORRECTED PROOF

Repeated measures ANOVAs were conducted on pre- and post-treatment SPAI-SP scores for each of the dichotomous variables (socially traumatic experience, parent sociability, and onset). Between-group results for the socially traumatic experience ($yes = 44$, $no = 12$) and parent sociability ($yes = 40$, $no = 16$) variables were not significant ($ps > .05$). However, there were main effects for time for both ANOVAs, with SPAI-SP scores decreasing significantly from pre- to post-treatment ($ps < .05$).

The ANOVA between earlier ($n = 26$) and later ($n = 15$) onset groups showed a significant effect for time ($F_{1,39} = 69.40$, $p < .001$), a significant effect for group ($F_{1,39} = 4.11$, $p = .05$), but no significant interaction. Tukey post hoc tests showed that SPAI-SP scores differed between onset groups at post-treatment, but not at pre-treatment (see Table 3). In other words, those reporting an earlier onset were more severe in their social anxiety symptoms at post-treatment, but not pre-treatment, compared to those reporting a later onset.

Finally, the Pearson correlation between BI and the SPAI-SP change score ($n = 43$) was not significant ($r = .11$, $p > .05$), suggesting no significant relationship between BI and treatment-related improvement in social anxiety symptoms.

Discussion

Results from the current study were consistent with previous research on age of onset in SAD. Over half of the current sample of adults with generalized SAD reported an onset in childhood (59%), with the remaining reporting onset in adolescence/early adulthood. Some have argued that the earlier versus later onset distinction may be an artifact of the SAD subtypes (Stein et al., 2001). However, the current study found a similar pattern of onset within a sample of participants diagnosed with the generalized subtype. Therefore, current results suggest that age of onset does not appear to be associated with SAD subtype per se.

Although both onset groups reported overall increased severity of social anxiety across the age points, the earlier onset group showed greater severity relative to the later onset group. It is not surprising that those reporting an earlier onset reported greater severity in childhood compared to those reporting a later onset. However, those with an earlier onset also reported greater severity at later time points compared to the later onset group, including during earlier and later adolescence, suggesting that timing of onset denotes a more severe course of illness. The lack of group differences

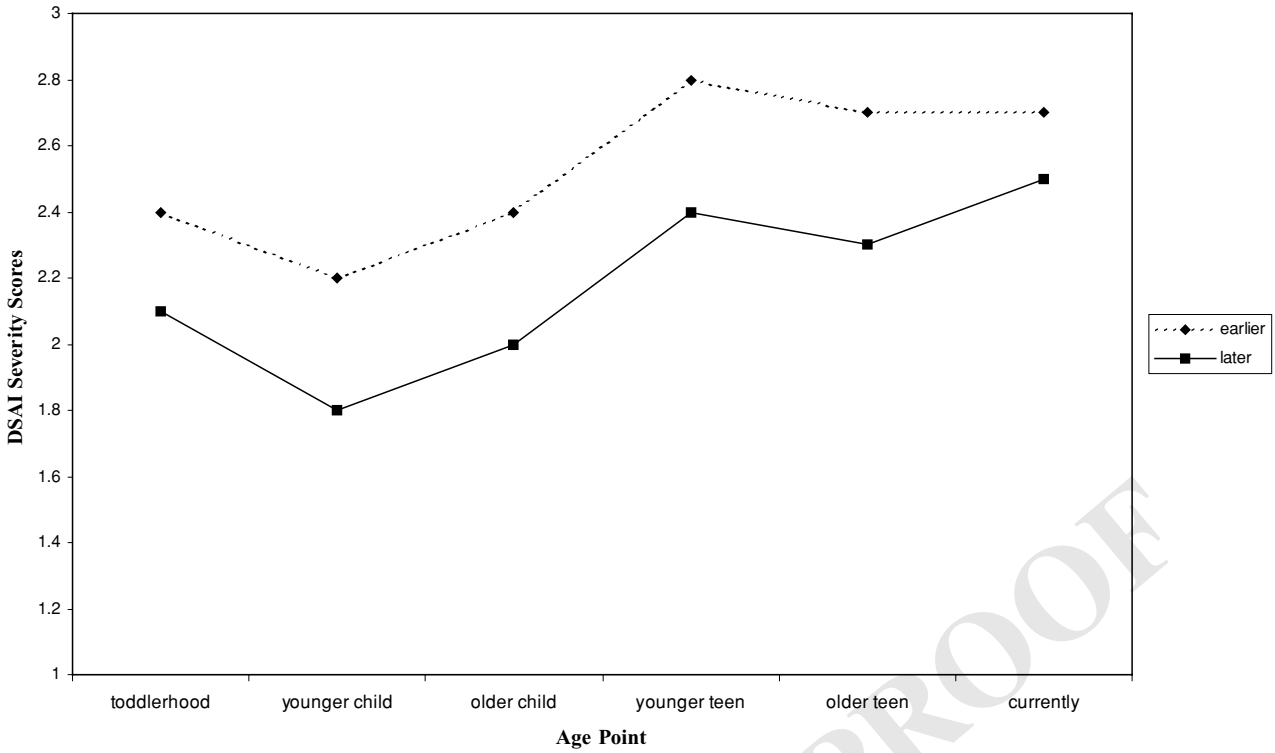

Fig. 1 D-SAI severity scores for earlier versus later onset of illness groups

in current severity may be due to the fact that this was a treatment-seeking sample; thus, scores were potentially elevated at the time of assessment. It is also important to note that although both groups improved significantly over the course of treatment, those with an earlier onset remained more severe at post-treatment compared to those with a later onset, even though pre-treatment severity was similar between the onset groups. This suggests that having an earlier onset may negatively impact the course of treatment.

Several studies have examined possible developmental factors related to SAD separately (see Morris, 2001, for a review), but the current investigation is one of the few to systematically examine multiple developmental factors as they relate to severity of SAD. Results were similar to those found by Stemberger et al. (1995), with both studies finding childhood shyness to be related to severity of adult SAD. However, socially traumatic experiences in the current study were not related to severity of generalized SAD. Perceived socially traumatic events may be more relevant for the specific subtype, as was suggested in the Stemberger et al. study.

Results of the current study extended previous research by finding that only BI was associated with current severity of SAD symptoms based on the D-SAI and SPAI-SP. Emerging evidence suggests that BI may not only be associated with the later development of anxiety disorders in general, but SAD specifically (Schwartz et al., 1999). In general, the current study did not support the predictive validity of the other developmental variables in relation to current symptom severity when in combination with BI. It is possible that the effects of these variables added little to the explained variance in the presence of more salient ones, such as BI. However, it also is possible that the restriction of range due to dichotomous coding of the socially traumatic experiences and parent sociability variables limited the ability of these two variables to demonstrate an effect. Future studies should examine these variables using continuous measures.

Table 2 Stepwise multiple regression results

	Criterion variables					
	D-SAI current severity			SPAI-SP severity		
Predictors	β	t	p	β	t	p
Behavioral inhibition	.37	3.60	.001	.27	2.23	.029
Childhood shyness	− .18	− 1.34	.183	− .05	.30	.764
Onset	− .10	− .97	.333	− .15	− 1.24	.219
Traumatic experience	.07	− .68	.499	− .04	− .33	.744
Parent sociability	.17	1.65	.103	.10	.83	.410

Note. D-SAI: Developmental Social Anxiety Interview; SPAI-SP: Social Phobia and Anxiety Inventory-Social Phobia Subscale.

UNCORRECTED PROOF

Table 3 Treatment outcome anova results

	Pre-treatment SPAI-SP *M* (*SD*)	Post-treatment SPAI-SP *M* (*SD*)	Between subjects *F* (*p*)	Within subjects *F* (*p*)	Interaction effect *F* (*p*)
Onset			4.11 (.049)	69.40 (<.01)	0.08 (.77)
Earlier	148.8 (22.9)	106.5 (33.4)			
Later	133.1 (31.2)	87.8 (37.3)			
Socially traumatic experience			1.43 (.239)	55.27 (<.01)	0.00 (.99)
Yes	140.3 (27.2)	97.4 (32.4)			
No	150.2 (24.2)	111.3 (42.9)			
Parent sociability			0.09 (.768)	64.93 (<.01)	0.16 (.69)
Yes	142.4 (30.2)	99.1 (39.1)			
No	141.5 (15.0)	105.4 (17.7)			

Note. SPAI-SP: Social Phobia and Anxiety Inventory-Social Phobia Subscale.

Although there were differences between earlier and later onset groups in treatment response, no other relationships were found between treatment outcome and developmental factors. Further, although BI significantly predicted current (pre-treatment) severity, it was not related to treatment outcome. One possible explanation is that BI is most closely related to illness severity, and previous research has not shown pre-treatment severity to be a consistent predictor of treatment outcome, particularly when examining symptom improvement instead of end-state functioning (Lincoln et al., 2005). Timing of onset may denote more than symptom severity, and those with an earlier onset may represent a qualitatively different group. For example, those with an earlier onset may be more likely to develop depression or other comorbid conditions compared to those with a later onset, leading to poorer outcomes. One of the few consistent predictors of poor treatment response in SAD has been comorbid conditions, and in particular, depression (Chambless, Tran, & Glass, 1997; Lincoln et al., 2005). Future studies with larger samples should longitudinally examine those with an earlier versus later onset to investigate whether they experience differences in their course of illness (e.g., the development of comorbid conditions) that may be related to poorer treatment response.

Potential limitations exist that should be considered when interpreting the findings. The differences in severity across age points could have been an artifact of measurement. In order to include items that were developmentally appropriate, the age subsections contained different numbers of questions. However, this was controlled for by computing an average score for each age point. In addition, examination of SUDS ratings at each of the age points showed the same pattern of results. Finally, past studies have found a similar onset pattern in epidemiological samples using different methodologies (Wittchen, Stein, & Kessler, 1999; Juster et al., 1996).

Another potential limitation of the current study was the lack of a non-clinical or non-SAD psychiatric control group. Therefore, the degree to which results are specific to SAD versus other clinical and non-treatment seeking samples remains a question for further study. Nevertheless, the current study obtained results similar to those found in Stemberger et al. (1995), which included a non-clinical comparison group.

Although results from the current study showed BI to be consistently associated with current social anxiety severity, this subscale may have more simply assessed shyness or social anxiety during toddlerhood, rather than a more complex construct of temperament. For example, the items used to form the BI subscale appear to most clearly assess the facet of BI related to social withdrawal in the presence of strangers. Hayward, Killen, Kraemer, and Taylor (1998) found that two components of BI, social avoidance and fearfulness, predicted a four times greater risk of development of social anxiety in adolescence. Furthermore, recent study findings for BI are consistent with a growing body of evidence showing more specific links between this temperamental style and SAD (Kagan, 2000; Schwartz et al., 1999).

As with any retrospective study, memory inaccuracies and cognitive biases could influence the recall of information. Longitudinal studies in both non-clinical and clinical samples have found evidence of compromised memory for details (Offer, Kaiz, Howard, & Bennett, 2000). However, a study by Masia et al. (2003) found that if a childhood disorder was recalled ten years later, then it was likely that some disorder had actually been present in childhood. As the current study utilized a treatment-seeking sample, current mood state could have influenced recall and interpretation. However, a review by Brewin, Andrews, and Gotlib (1993) concluded that there is little evidence for general memory deficits associated with anxiety, and recall of significant past events does not appear to be affected by mood state.

A final potential limitation is that separate ratings of mother and father sociability were not obtained. Different results may have been obtained with separate ratings, given

UNCORRECTED PROOF

that previous research has found interactions between parent and child gender (Neal & Edelmann, 2003). However, other studies examining mother and father sociability separately have found no differences, showing that lower sociability in both parents predicted severity of social anxiety (Bogels, van Oosten, Muris, & Smulders, 2001; Bruch & Heimberg, 1994).

Despite these potential limitations, the present findings provide support for certain childhood factors (BI, age of onset) that may be related to the course and severity of SAD and response to treatment. Current results were consistent with other studies indicating an earlier and later pattern of onset, even in those with generalized SAD, suggesting that onset is not merely related to diagnostic subtype. Future research using longitudinal designs is needed and should include the use of multiple informants to reduce the potential impact of memory bias in retrospective reports.

Acknowledgement We would like to thank Suzy Goldstein, Ph.D. for her assistance on this project. This research was supported in part by Grant No. R01 MH052232 from the National Institute of Mental Health.

References

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington, DC: Author.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Arrindell, W. A., Kwee, M. G. T., Methorst, G. J., Van Der Ende, J., Pol, E., & Moritz, B. J. M. (1989). Perceived parenting styles of agoraphobic and socially phobic in-patients. *British Journal of Psychiatry, 155*, 526–535.

Beck, A. T., Steer, R. A., & Garbin, R. A. (1988). Psychometric properties of the Beck depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8*, 77–100.

Beck, A. T., & Steer, R. A. (1987). *Manual for the Beck Depression Inventory*. San Antonio, TX: Psychological Corporation.

Beidel, D. C., Borden, J. W., Turner, S. M., & Jacob, R. G. (1989). The Social Phobia and Anxiety Inventory: Concurrent validity with a clinic sample. *Behaviour Research and Therapy, 27*, 573–576.

Beidel, D. C., Turner, S. M., Stanley, M. A., & Dancu, C. V. (1989). Assessment of social phobia: Reliability of an impromtu speech. *Journal of Anxiety Disorders, 3*, 149–158.

Biederman, J., Hirshfeld-Becker, D. R., Rosenbaum, J. F., Herot, C., Friedman, D., Snidman, N., et al. (2001). Further evidence of association between behavioral inhibition and social anxiety in children. *American Journal of Psychiatry, 158*, 1673–1679.

Bogels, S. M., van Oosten, A., Muris, P., & Smulders, D. (2001). Familial correlates of social anxiety in children and adolescents. *Behaviour Research and Therapy, 39*, 273–287.

Brewin, C. R., Andrews, B., and Gotlib, I. H. (1993). Psychopathology and early experience: A reappraisal of retrospective reports. *Psychological Bulletin, 113*, 82–98.

Bruch, M. A., & Heimberg, R. G. (1994). Differences in perceptions of parental and personal characteristics between generalized and nongeneralized social phobics. *Journal of Anxiety Disorders, 8*(2), 155–168.

Bruch, M. A., Heimberg, R. G., Berger, P., & Collins, T. M. (1989). Social phobia and perceptions of early parental and personal characteristics. *Anxiety Research, 2*, 57–65.

Chambless, D. L., Tran, G. Q., & Glass, C. R. (1997). Predictors of response to cognitive-behavioral group therapy for social phobia. *Journal of Anxiety Disorders, 11*, 221–240.

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social Phobia: Diagnosis, assessment, and treatment* (pp. 69–93). New York: Guilford.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured clinical interview for DSM-IV axis I disorders—patient edition* (SCID-I/P, Version 2.0). New York: Biometrics Research Department.

George, D., & Mallery, P. (1999). *SPSS for windows step by step: A simple guide and reference*. Boston: Allyn & Bacon.

Goldstein, S. G., Herbert, J. D., Hartfield, C., Christian, R., Kaplan, M., & Rheingold, A. (1997, November). *The etiology of social phobia: A developmental perspective*. Poster presented at the 31st Annual Convention of the Association for the Advancement of Behavior Therapy, Miami, FL.

Hayward, C., Killen, J. D., Kraemer, H. C., & Taylor, C. B. (1998). Linking self-reported childhood behavioral inhibition to adolescent social phobia. *Journal of the American Academy of Child and Adolescent Psychiatry, 37*, 1308–1316.

Heimberg, R. G., & Becker, R. E. (2002). *Cognitive-behavioral group therapy for Social Phobia: Basic mechanisms and clinical strategies*. New York: Guilford Press.

Herbert, J. D., Bellack, A. S., & Hope, D. A. (1991). Concurrent validity of the Social Phobia and anxiety inventory. *Journal of Psychopathology and Behavioral Assessment, 14*, 357–368.

Herbert, J. D., Gaudiano, B. A., Rheingold, A. A., Myers, V. H., Dalrymple, K., & Nolan, E. M. (2005). Social skills training augments the effectiveness of cognitive behavioral group therapy for social anxiety disorder. *Behavior Therapy, 36*, 125–138.

Herbert, J. D., Goldstein, S. G., & Dalrymple, K. L. (2004). *The developmental social anxiety interview (D-SAI)*. Unpublished manuscript. Drexel University, Philadelphia, PA.

Herbert, J. D., Rheingold, A. A., Gaudiano, B. A., & Myers, V. H. (2004). Standard versus extended cognitive behavior therapy for social anxiety disorder: A randomized-controlled trial. *Behavioural and Cognitive Psychotherapy, 32*, 131–147.

Juster, H. R., Brown, E. J., & Heimberg, R. G. (1996). Social phobia. In J. Margraf (Ed.), *Textbook of behavior therapy* (pp. 43–59). Berlin: Springer-Verlag.

Juster, H. R., & Heimberg, R. G. (1995). Social phobia: Longitudinal course and long-term outcome of cognitive-behavioral treatment. *The Psychiatric Clinics of North America, 18*, 821–842.

Kagan, J. (2000). Temperamental contributions to affective and behavioral profiles in childhood. In S. G. Hofmann & P. M. DiBartolo (Eds.), *From social anxiety to social phobia: Multiple perspectives* (pp. 216–234). Boston: Allyn & Bacon.

Kagan, J., Reznick, J. S., & Snidman, N. (1988). Biological basis of childhood shyness. *Science, 240*, 167–171.

Kessler, R. C., Berglund, P., Demler, O., Jin, R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*, 593–602.

Lincoln, T. M., Rief, W., Hahlweg, K., Frank, M., Von Witzleben, I., Schroeder, B., & Fiegenbaum, W. (2005). Who comes, who stays, who profits? Predicting refusal, dropout, success, and relapse in a short intervention for social phobia. *Psychotherapy Research, 15*, 210–225.

Masia, C. L., Storch, E. A., Dent, H. C., Adams, P., Verdeli, H., Davies, M., & Weissman, M. M. (2003). Recall of childhood psychopathol-

UNCORRECTED PROOF

ogy more than 10 years later. *Journal of the American Academy of Child and Adolescent Psychiatry, 42*(1), 6–12.

Morris, T. L. (2001). Social phobia. In M. W. Vasey, & M. R. Dadds (Eds.), *The developmental psychopathology of anxiety* (pp. 435–458). New York: Oxford University Press.

Neal, J. A., & Edelmann, R. J. (2003). The etiology of social phobia: Toward a developmental profile. *Clinical Psychology Review, 23*, 761–786.

Nunnally, J. C. (1978). *Psychometric theory* (2nd ed.). New York: McGraw-Hill.

Offer, D., Kaiz, M., Howard, K. I., & Bennett, E. S. (2000). The altering of reported experiences. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 735–742.

Ost, L.-G., & Hughdahl, K. (1981). Acquisition of phobias and anxiety response patterns in clinic patients. *Behaviour Research and Therapy, 16*, 439–447.

Pedhazur, E. J. (1997). *Multiple regression in behavioral research: Explanation and prediction*. Fort Worth, TX: Harcourt Brace.

Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research & Therapy, 35*, 741–765.

Rapee, R. M., & Melville, L. F. (1997). Recall of family factors in social phobia and panic disorder: Comparison of mother and offspring reports. *Depression and Anxiety, 5*, 7–11.

Reich, J., Goldenberg, I., Vasile, R., et al. (1994). A prospective follow-along study of the course of social phobia. *Psychiatry Research, 54*, 249–258.

Schneier, F. R., Johnson, J., Hornig, C. D., et al. (1992). Social phobia: Comorbidity and morbidity in an epidemiologic sample. *Archives of General Psychiatry, 49*, 282–288.

Schwartz, C. E., Snidman, N., & Kagan, J. (1999). Adolescent social anxiety as an outcome of inhibited temperament in childhood. *Journal of the American Academy of Child and Adolescent Psychiatry, 38*, 1008–1015.

Segal, D. L., Hersen, M., & Van Hasselt, V. B. (1994). Reliability of the Structured Clinical Interview for DSM-III-R: An evaluative review. *Comprehensive Psychiatry, 35*, 316–327.

Stein, M. B., Chavira, D. A., & Jang, K. L. (2001). Bringing up bashful baby: Developmental pathways to social phobia. *Psychiatric Clinics of North America, 24*, 661–676.

Stemberger, R. T., Turner, S. M., Beidel, D. C., & Calhoun, K. S. (1995). Social phobia: An analysis of possible developmental factors. *Journal of Abnormal Psychology, 104*, 526–531.

Turner, S. M., Beidel, D. C., Dancu, C. V., & Stanley, M. A. (1989). An empirically derived inventory to measure social fears and anxiety: The Social Phobia and Anxiety Inventory. *Psychological Assessment, 1*, 35–40.

Turner, S. M., Beidel, D. C., & Wolff, P. L. (1996). Is behavioral inhibition related to the anxiety disorders?. *Clinical Psychology Review, 16*, 157–172.

Wittchen, H. U., Stein, M. B., & Kessler, R. C. (1999). Social fears and social phobia in a community sample of adolescents and young adults: Prevalence, risk factors and comorbidity. *Psychological Medicine, 29*, 309–323.

Wolpe, J., & Lazarus, A. A. (1966). *Behavior therapy techniques*. New York: Pergamon.

UNCORRECTED PROOF