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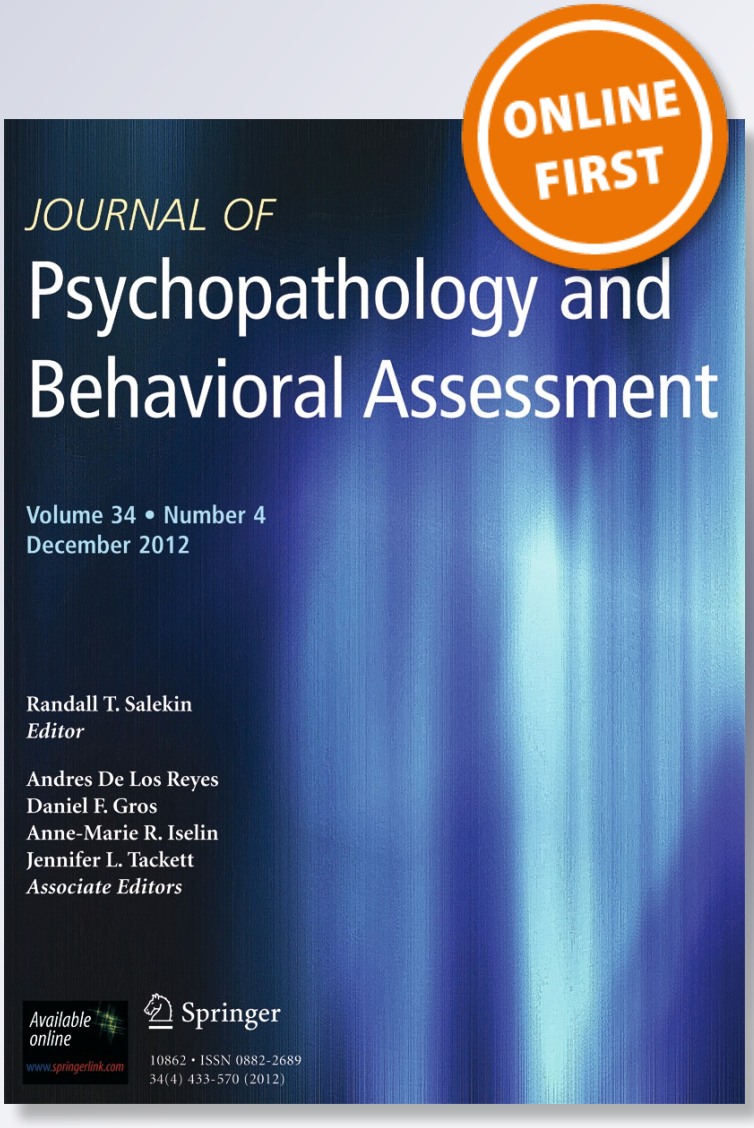
Journal of Psychopathology and Behavioral Assessment

ISSN 0882-2689

J Psychopathol Behav Assess
DOI 10.1007/s10862-013-9338-5



Springer

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The Relevance of Age of Onset to the Psychopathology of Social Phobia

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Abstract The present study aimed to examine the relevance of age of onset to the psychopathology of social phobia using a large clinical sample of 210 patients with social phobia. The two most common periods of onset were during adolescence (ages 14–17) and early childhood (prior to age 10). Structural regression modeling was used to test predictions that early onset social phobia would be associated with greater severity of the disorder, stronger current symptoms of depression and anxiety, greater functional impairment, and more pronounced levels of emotional disorder vulnerabilities (e.g., neuroticism/behavioral inhibition, extraversion, perceptions of control). Logistic regression was used to evaluate relationships between age of onset and the presence of acute and chronic stress at the time of onset. Results showed that earlier age of social phobia onset was associated with stronger current psychopathology, functional impairment, and emotional disorder vulnerabilities, and that later age of onset predicted the presence of an acutely stressful event around the time of disorder emergence. These results are discussed in regard to their clinical implications and congruence with prominent etiological models of the emotional disorders.

Keywords Social phobia · Age of onset · Vulnerability-stress · Impairment · Structural equation modeling

Social phobia is classified in the 4th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*) as an anxiety disorder characterized by an excessive fear of social or performance situations in which embarrassment or humiliation may occur (American Psychiatric Association 2000). Social phobia is the third most common psychiatric disorder in the United States following major depression and alcohol dependence (Kessler et al. 2005), and the most common lifetime social fears are public speaking (21.2 %) and speaking in meetings or classes (19.5 %; Ruscio et al. 2008). Prevalence estimates for lifetime and 12-month *DSM-IV* social phobia are 12.1 % and 7.1 %, respectively (Kessler et al. 2005; Ruscio et al. 2008).

Social phobia is associated with one of the earliest onsets of all the anxiety disorders, and without treatment, social phobia tends to follow a chronic and unremitting course (Brown et al. 2001a; Juster and Heimberg 1995; Reich et al. 1994). Research suggests a bimodal pattern of onset, with some reports of onset before age five, and other reports of onset in mid-adolescence (Juster and Heimberg 1995; Stein et al. 2001). Whereas onset during childhood and adolescence coincides with normative vulnerabilities to social embarrassment (Ollendick and Hirshfeld-Becker 2002), the development of social phobia after these peak periods is relatively uncommon and usually particular to cases that are occurring secondary to another mental disorder (i.e., depression, psychotic disorders, eating disorders; Wittchen and Felm 2003). In children, the anxiety response of social phobia may manifest in the form of crying, clinging to a familiar person, or wariness, whereas adolescents and adults may experience panic-like symptoms (e.g., autonomic arousal such as accelerated heart rate, sweating, or trembling; Ollendick and Hirshfeld-Becker 2002).

According to the National Comorbidity Survey-Replication, nearly two-thirds (62.9 %) of respondents with lifetime social phobia meet criteria for at least one other lifetime *DSM-IV* disorder, and the extent of comorbity with other disorders is positively associated with number of feared social situations (Ruscio et al. 2008). Because social phobia typically begins in childhood, its onset often precedes other disorders with which it is comorbid, and may be a direct or indirect risk factor for the development of other mental disorders (e.g., earlier onset may

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Published online: 16 January 2013

predict increased comorbidity). For example, social phobia has been shown to be a predictor of both later-onset depression (Stein et al. 2001) and substance use disorders (Zimmermann et al. 2003). Given such findings, it is not surprising that untreated social phobia is associated with a number of negative outcomes, including poor school and work performance, school dropout, and unemployment (Wittchen and Beloch 1996; Wittchen et al. 1999). Importantly, research has also found that individuals with social phobia and no comorbid psychopathology experience significant levels of functional impairment (Ruscio et al. 2008). Despite this literature suggesting that social phobia has an early age of onset (i.e., childhood and adolescence) and is associated with significant comorbidity and functional impairment, very few studies have evaluated the relevance of age of social phobia onset in predicting clinical outcomes (e.g., anxiety and depression severity, extent of comorbidity and impairment). Prior studies have found that compared to those with later onset, individuals with earlier onset social phobia report more severe anxiety, respond less well to treatment, and are more likely to experience earlier and more chronic depression (Dalrymple et al. 2007; Dalrymple and Zimmerman 2011; Wittchen et al. 1999).

Although conceptual models have outlined several developmental pathways to the onset of social phobia including genetics, temperament/personality (e.g., behavioral inhibition, neuroticism, extraversion), parental influences, conditioning events, and cognitive vulnerabilities (Barlow 2000; Ollendick and Hirshfeld-Becker 2002), few studies have examined the relationship between age of social phobia onset and such etiological (i.e., vulnerability-stress) factors. Barlow (2000) posits that genetically based dimensions of neuroticism, extraversion, behavioral inhibition, and behavioral activation may serve as important biological vulnerabilities to the development of anxiety and depressive disorders. Specifically, a combination of high neuroticism/behavioral inhibition and low extraversion/behavioral activation are believed to constitute important risk factors in the development of social anxiety. Barlow also discusses the psychological vulnerability of perceived controllability/uncontrollability (i.e., of stress and emotions), which may serve as a mediator between negative life events and the emergence of anxiety in early development.

In addition to personality/temperament vulnerabilities, conditioning events may also play an important role in the development of social phobia; many adults with social phobia (44–58 %) are able to identify a specific humiliating event associated with the onset of their disorder, and this is especially true for those with one or more specific performance fear(s) (Ollendick and Hirshfeld-Becker 2002; Öst 1985; Stemberger et al. 1995). In particular, conditioning experiences appear to result in social phobia among individuals with preexisting temperamental vulnerabilities (Mineka and Zinbarg 1995; Ollendick and Hirshfeld-Becker 2002). Unfortunately, prior research has yet to examine the relationships between age of social phobia onset and such environmental factors (e.g., conditioning events, chronic stress).

The current study attempted to extend our understanding of the role of age of onset in the psychopathology of social phobia by addressing several limitations of extant literature. Very few studies have evaluated the importance of social phobia age of onset, and the existing literature has yet to examine how onset predicts severity of depression, autonomic arousal, or comorbidity (e.g., number of comorbid diagnoses). Moreover, virtually no studies have examined the relationship between onset and personality/temperament vulnerabilities (e.g., neuroticism, extraversion). Collectively, the social phobia age of onset literature has failed to address the potentially confounding effects of duration of social phobia. Duration of social phobia may be associated with negative outcomes (e.g., longer duration predicting more severe anxiety and functional impairment), and might better account for the differences between early-onset and late-onset groups. Additionally, previous research on age of onset has usually treated onset as a dichotomous or three-category variable (e.g., Dalrymple et al. 2007) with patients divided into only two (e.g., early versus late onset) or three (early versus middle versus late onset) different onset groups. To gain additional information and reduce measurement error, age of onset was treated as a five-point ordinal variable for the analyses in this study. Finally, our study expands on the generalizability of the social phobia age of onset literature (e.g., Dalrymple and Zimmerman 2011) by examining a diverse sample of individuals with comorbid Axis I disorders.

Present Study

The present study evaluated the relevance of age of onset of social phobia by examining its relationships with anxiety and depression severity, comorbidity, impairment, emotional disorder vulnerabilities, and stress. Similar to other studies that have examined the effects of age of onset while controlling for duration of illness (i.e., in generalized anxiety disorder, see Campbell et al. 2003), a structural modeling approach was used in the present study. Latent variables representing the constructs of interest were used when multiple indicators were available. It was predicted that earlier onset social phobia would be associated with more severe psychopathology (i.e., social phobia severity, symptoms of anxiety and depression, lifetime comorbidity), more pronounced emotional disorder vulnerabilities (i.e., highly neurotic, introverted, low perceived emotional control), and greater functional impairment. Age of social phobia onset was also expected to be associated with the presence of stress at the time of onset; whereas earlier onset social phobia was expected to be associated with chronic stress, later onset social phobia was hypothesized to be associated with an acutely stressful event.

Material and Methods

Participants

The sample consisted of 210 patients who presented for assessment and treatment at the Center for Anxiety and Related Disorders (CARD) at Boston University. At the time of their initial assessment, individuals underwent a semi-structured interview and completed a packet of well-known self-report questionnaires. All individuals included in the sample carried a current diagnosis of social phobia. Women constituted the larger portion of the sample (54.8 %) and the average age was 32.48 (*SD*=12.60, range=17 to 74). The sample was predominantly Caucasian (90.5 %; African-American=3.4 %, Asian=4.3 %, Pacific Islander=.5 %, American-Indian/Alaskan=.5 %). Diagnoses were established using the Anxiety Disorders Interview Schedule for *DSM-IV*: Lifetime version (ADIS-IV-L; Di Nardo et al. 1994), a semi-structured interview designed to ascertain reliable diagnosis of the *DSM-IV* anxiety, mood, somatoform, and substance use disorders, and to screen for the presence of other conditions (e.g., psychotic disorders). This instrument was administered to patients by trained doctoral students or doctoral-level clinical psychologists upon presentation for assessment and treatment at CARD. The ADIS-IV-L assesses key and associated features of many disorders (e.g., major depressive disorder, dysthymia, social phobia, specific phobia, generalized anxiety disorder, and obsessive compulsive disorder) regardless of whether a diagnosis of that disorder is under consideration. A reliability study of a subset of patients seen at CARD (*n*=362) who had two independent administrations of the ADIS-IV-L indicated good to excellent inter-rater agreement in diagnosing current disorders (range of *k*s=.67 to .86, except dysthymia *k*=.31; Brown et al. 2001b). For each diagnosis, interviewers assign a 0–8 clinical severity rating (CSR) that indicates the degree of distress and impairment associated with the disorder (0 = "none" to 8 = "very severely disturbing/disabling"). In patients with two or more current diagnoses, the "principal" diagnosis is the one receiving the highest CSR. For current and lifetime disorders that meet or surpass the threshold for a formal *DSM-IV* diagnosis ("clinical" diagnoses), CSRs of 4 (definitely disturbing/disabling) or higher are assigned. Current clinical diagnoses not deemed to be the principal diagnosis are referred to as "additional" diagnoses. The rates of current clinical disorders (collapsing across principal and additional diagnoses) that frequently occurred in the sample were as follows: social phobia (100 %), panic disorder with or without agoraphobia (16.2 %), generalized anxiety disorder (40.5 %), specific phobia (9.0 %), obsessive–compulsive disorder (14.3 %), major depressive disorder (29 %), and dysthymic disorder (6.2 %). Fifty-one percent of the present sample (*n*=108) had a principal diagnosis of social phobia and 50 % (*n*=105) were diagnosed with the generalized subtype.

Measures

Social Phobia Severity As described above, the ADIS-IV-L is used to make ratings of key and associated features of social phobia. Dimensional ratings are made representing fear and avoidance of 13 different social situations (e.g., attending parties, formal speaking, initiating a conversation; range = 0 [none] to 8 [severe]). Using this same Likert-scale, additional dimensional ratings are also made for the five *DSM-IV* social phobia criteria. These *DSM-IV* criteria ratings differ from the ADIS-IV-L fear and avoidance ratings by also ascertaining excessiveness (criterion C) and interference (criterion E). A reliability study of a subset of patients seen at CARD (*n*=292) indicated good inter-rater agreement for these dimensional social phobia ratings (range of *r*s=.80 to .86, Brown et al. 2001b). A latent variable of social phobia severity was formed using composite scores of (a) the 13 ADIS-IV-L social phobia fear ratings, (b) the 13 ADIS-IV-L social phobia avoidance ratings, and (c) the 5 *DSM-IV* social phobia criteria ratings.

Depression Symptoms A latent variable of unipolar depression was formed using (a) the 9 ADIS-IV-L major depression criteria ratings (range = 0 [none] to 8 [severe]), (b) the Beck Depression Inventory-II (21-item BDI-II; Beck et al. 1996, see Steer et al. 1997 for psychometric properties) and, (c) the Depression scale of the 21-item version of the Depression Anxiety and Stress Scales (DASS-21; Lovibond and Lovibond 1995; see Brown et al. 1997 for psychometric properties). Although both the BDI-II and DASS-21 are composed of self-descriptive statements and individuals are asked to answer using a four-point Likert-type scale, responses choices for the BDI-II vary item-by-item, whereas DASS-21 ratings range from 0 (*did not apply to me at all*) to 3 (*applied to me very much, or most of the time*).

Autonomic Arousal A latent variable of anxiety symptoms (i.e., autonomic arousal) was constructed using two well-validated self-report questionnaires: (a) the Beck Anxiety Inventory (21-item BAI; Beck and Steer 1993) and (b) the Anxiety scale of the 21-item version of the DASS (Lovibond and Lovibond 1995; see Brown et al. 1997 for psychometric properties). The BAI consists of self-descriptive statements about anxiety symptoms that individual's rate using a four-point Likert-type scale ranging from 0 (*not at all*) to 3 (*severely, I could barely stand it).*

Neurotic and Extraverted Temperament Neuroticism and extraversion were assessed using three self-report measures: the NEO Five-Factor Inventory (NFFI; Costa and McCrae 1992), the Eysenck Personality Questionnaire (EPQ; Eysenck and Eysenck 1975), and the Behavioral Inhibition/Activation Scales (BIS/BAS; Carver and White 1994). The NFFI is a 60-

item self-report measure of the five-factor model of personality. Items are composed of self-descriptive statements rated on a five-point Likert-type scale ranging from 1 (*strongly disagree*) to 5 (*strongly agree*). The latent structure of the NFFI has been supported in clinical samples (Rosellini and Brown 2011), and each domain has been found to possess adequate internal consistency (Costa and McCrae 1992) and temporal stability (Robins et al. 2001). The EPQ is a measure of personality in which individuals respond "yes" or "no" to a variety of self-descriptive statements. Several studies have supported the reliability, validity, and long-term test-retest reliability of the EPQ (Eysenck and Eysenck 1975; Loo 1979). The 20-item short-form version of the EPQ was used in the present study (cf. Fanous et al. 2007). The BIS/BAS is 20-item self-report measure of behavioral inhibition and behavioral activation. Items are composed of self-descriptive statements and are rated on a four-point Likert-type scale ranging from 1 (*quite untrue*) to 4 (*quite true*). The latent structure of the BIS/BAS has been supported in clinical samples, and each domain has been found to possess adequate scale reliabilities (Campbell-Sills et al. 2004). Whereas the Neuroticism scales of the NFFI and EPQ, and the Behavioral Inhibition Scale of the BIS/BAS comprised the latent variable of Neurotic Temperament, the Extraversion scales of the NFFI and EPQ, and Behavioral Activation Scale of the BIS/BAS were used to represent the latent variable of Extraverted Temperament.

Perceived Control A latent variable representing perceived control was formed using the total score of the Anxiety Control Questionnaire-Revised (ACQ-R; Rapee et al. 1996). The ACQ-R is an 18-item self-report measure used to assess an individual's perceptions of control over emotions, stress, and threat. Items consist of self-descriptive statements that are rated using a six-point Likert-type scale ranging from 0 (*strongly disagree*) to 5 (*strongly agree*). The reliability, validity, and latent structure of the ACQ-R have been supported in clinical samples (Brown et al. 2004).

Functional Impairment A latent variable for functional impairment was created using the Subjective Symptoms Scale (SSS)—Self-Report and the SSS—Clinician-Report. These measures are composed of five-items rated using Likert scales ranging from 0 (*not at all*) to 8 (*severe*), and assess interference due to symptoms of psychopathology in a variety of spheres (e.g., work/school, home management, social life, private leisure activities, relationships). The SSS scales are modified versions of the Work and Social Adjustment Scale, introduced by Hafner and Marks (1976), and have demonstrated good internal consistency within clinical samples (e.g., Brown et al. 1995).

Lifetime Comorbidity A latent variable representing total number of lifetime diagnoses was formed by summing the total number of current and past diagnoses assigned during ADIS-IV-L administration (excluding social phobia). This approach has been used in prior structural models examining the relevance of age of onset in predicting severity of comorbidity (Campbell et al. 2003). As previously mentioned, the ADIS-IV-L has been shown to reliably diagnosis the majority of the anxiety and mood disorders (see Brown et al. 2001b).

Age of Onset and Stress Age of social phobia onset and stress at the time of onset were assessed during administration of the social phobia section of the ADIS-IV-L. Prior research has demonstrated that retrospective self-report may be influenced by memory deficits and/or recall biases (cf., Henry et al. 1994); thus, this study attempted to maximize assessment reliability by coding the data according to categories of onset (i.e., based on educational milestones) and stress (i.e., acute versus chronic stress) rather than assessing these constructs continuously. In other words, individuals were expected to more reliably report onset and stress categories than specific ages of onset or details pertaining to stress severity.

Using the ADIS-IV-L, patients are asked "When did anxiety about social situations begin to be a problem?" Because many patients are unable to identify an exact age of onset, further questioning is utilized in order to link the onset to objective events in their life (e.g., a particular grade of school, moving to a new city, seasons of the year, specific holidays, etc.). If provided, specific ages were also recorded. Based on this collective information, age of onset was coded in an ordinal fashion: 1 = early childhood (e.g., "as long as I can remember," prior to age 10), 2 = middle childhood (e.g., "middle school," ages 10–13), 3 = adolescence (e.g., "high school," ages 14–17), 4 = young adulthood (e.g., "college," ages 18–22), or 5 = adulthood (e.g., "after college," age 23 and older).

Subsequent to inquiry about age of onset, patients were also asked if they "could recall anything that may have led" to their social anxiety. These open-ended responses were recorded and subsequently coded dichotomously to capture the presence or absence of chronic stressors (e.g., critical parenting, bullying, teasing) and the presence or absence of acutely stressful events (e.g., having a panic attack in a social situation, starting middle school, high school, or college, moving). If no events were identified, the two stress variables were coded as "0." In order to determine the reliability of these coded qualitative data, 67 (31.9 %) of these responses were separately rated by three individuals. Kappa coefficients demonstrated good inter-rater reliability across all three raters when coding for both the presence of chronic stress (rs=.70 to .81) and acute stress (rs=.65 to .79).

Data Analysis Measurement, structural regression, and logistic regression models were analyzed using direct maximum likelihood estimation (Mplus 5.2, Muthén and Muthén 1998–

2009). Whereas confirmatory factor analysis was used to evaluate a measurement model of the aforementioned latent and single indicator variables, structural equation modeling was used to evaluate the unique relationships between age of social phobia onset and clinical outcomes of interest (e.g., social phobia severity, temperament, comorbidity) while controlling for duration of social phobia. Logistic regression was used to evaluate relationships between age of onset and the presence of chronic and acute stress at the time of onset while controlling for duration of social phobia. Fit of the measurement model was examined using the Tucker-Lewis Index (TLI), comparative fit index (CFI), root mean square error of approximation (RMSEA) and its test of close fit (C-Fit), and standardized root mean square residual (SRMR). Multiple goodness-of-fit indices were evaluated to examine various aspects of model fit (i.e., absolute fit, parsimonious fit, fit relative to the null model; cf. Brown 2006). Unstandardized and completely standardized solutions were examined to evaluate the significance and strength of parameter estimates. Standardized residuals and modification indices were used to determine the presence of any localized areas of strain in the solutions.

Results

Distribution of Social Phobia Onset

Twenty-one percent of the overall sample reported social phobia onset during early childhood (i.e., onset at age 10 or younger), 10.0 % during mid-childhood (onset between ages 10 and 13), 28.1 % during adolescence (onset between ages 14 and 17), 19.5 % during late adolescence/young adulthood (onset between ages 18 and 22), and 11.4 % during adulthood (onset at age 23 or older).

Measurement Model of Continuous Outcomes

Confirmatory factor analysis was used to evaluate a measurement model composed of eight latent variables (i.e., outcomes of interest) and two observed variables (i.e., age of onset, current age). The variables were specified to freely correlate with one another. Two single indicators were treated as latent variables by specifying the error variance of the observed variable. Specifically, error variances were derived from reliability estimates and the sample variances of the single indicators. Whereas the reliability estimate for the Perceived Control factor was based on overall ACQ scale reliability from a psychometric study previously conducted at our clinic (ρ=.848, Brown et al. 2004), the coefficient for the Lifetime Comorbidity factor was derived from the inter-rater reliability of total number of lifetime diagnosis based on administrations of the ADIS-IV-L at our clinic since 1997 (r=.75). Error variances for current age and age of onset were fixed at zero. The eight-factor, two-observed variable measurement model provided excellent fit, χ^2 (129)=220.81 p<.001, SRMR=0.05, RMSEA=.06 (CFit p=.15), TLI=.95, CFI=.96. Although this model indicated some localized areas of strain (e.g., modification indices, MIs, suggesting salient factor cross-loadings or error covariances), none appeared to be substantively justified (e.g., largest MI=15.39 suggesting a correlated error between the lifetime comorbidity and ADIS-IV-L major depression indicators). Table 1 presents the factor loadings for the measurement model, all of which were statistically significant (all ps<.001). Correlations among the variables are presented in Table 2. At the zero-order level, social phobia age of onset was significantly associated with a number of the outcomes of interest (many rs≥.15 or ≤−.15).

Table 1 Factor loadings for the measurement model

Latent variable and indicator	Factor loading
Social phobia severity	
ADIS-IV-L clinician fear ratings	.95
ADIS-IV-L clinician avoidance ratings	.96
DSM-IV-L social phobia criteria clinician ratings	.68
Depression symptoms	
BDI-II	.93
DASS—Depression	.83
ADIS-IV-L major depression ratings	.83
Autonomic arousal	
BAI	.96
DASS—Anxiety	.82
Neurotic temperament	
NEO-FFI—Neuroticism	.91
EPQ—Neuroticism	.79
BIS/BAS—Behavioral inhibition	.62
Extraverted temperament	
NEO-FFI—Extraversion	.95
EPQ—Extraversion	.78
BIS/BAS—Behavioral activation	.61
Functional impairment	
SSS—Self-report	.80
SSS—Clinician-report	.64
Lifetime comorbidity	.87
Perceived control	.93
Age of social phobia onset	1.00
Current age	1.00

Completely standardized estimates are provided. DSM-IV = Diagnostic and Statistical Manual of Mental Disorders (4th ed.); ADIS-IV-L = Anxiety Disorders Interview Schedule: Lifetime version; BDI-II = Beck Depression Inventory; DASS = Depression Anxiety and Stress Scales; BAI = Beck Anxiety Inventory; NEO-FFI = NEO-Five Factor Inventory; EPQ = Eysenck Personality Questionnaire; BIS/BAS = Behavioral Inhibition/Behavioral Activation Scales; SSS = Subjective Symptom Scale

Table 2 Zero-order correlations among latent and observed variables in the measurement model

	Onset age	Current age	Social phobia severity	Depression symptoms	Autonomic arousal	Neurotic temp.	Extraverted temp.	Functional impairment	Lifetime comorbid	Perceived control
Onset age										
Current age	−.01									
Social phobia severity	−.17*	−.22***								
Depression symptoms	−.22***	.07	.29***							
Autonomic arousal	−.15*	−.19***	.17*	.38***						
Neurotic temp.	−.16**	−.06	.46***	.70***	.28***					
Extraverted temp.	.23***	−.05	−.50***	−.37***	.01	−.46***				
Functional impairment	−.17*	.06	.37***	.77***	.51***	.59***	−.35***			
Lifetime Comorbid	−.02	.04	.05	.58***	.35***	.50***	−.16	.64***		
Perceived control	.19**	.19**	−.38***	−.57***	−.50***	−.69***	.39***	−.49***	−.33***	

Completely standardized estimates are provided

Temp. = Temperament; Comorbid = Comorbidity

*p<.05, **p<.01, ***p<.001

Notably, strong correlations were also found among the outcomes pertaining to anxiety and depression severity, lifetime comorbidity, and emotional disorder vulnerabilities (rs=−.69 to .77)

Structural Regression Model of Continuous Outcomes

A structural regression model was used to evaluate the relationships between age of social phobia onset and outcomes pertaining to anxiety and depression severity, comorbidity, impairment, and emotional disorder vulnerabilities. Duration of social phobia (i.e., length of illness) was controlled for by simultaneously evaluating age of onset and current age as exogenous variables. Overall fit of the structural regression model was identical to that of the measurement model because the regression portion of the model was structurally just-identified.

The completely standardized regression coefficients for the structural model are presented in Fig. 1. The model was consistent with study hypotheses; age of social phobia onset significantly predicted Social Phobia Severity (completely standardized path, γ=−.17), Autonomic Arousal (γ=−.15), and Depression Symptoms (γ=−.22) such that earlier age of onset was associated with increased severity of social phobia, autonomic arousal, and depression. In addition, age of onset was a predictor of the biological and psychological vulnerability constructs; whereas earlier onset was associated with greater levels of Neurotic Temperament (γ=−.16), later onset was associated with greater levels of Extraverted Temperament (γ=.23) and Perceived Control (γ=.19). Earlier age of onset also predicted greater levels of Functional Impairment (γ=−.16). Despite statistical significance, it is notable that the size of these effects were small (i.e., f^2=.03 to .08). Contrary to hypotheses, age of social phobia onset did not predict Lifetime Comorbidity.

Logistic Regression of Dichotomous Outcomes

Logistic regression analyses were conducted to examine if age of onset was uniquely associated with the categorical outcomes of acute and chronic stress. Sixteen cases were excluded from this analysis due to missing age of onset and/or current age data (n=194; these missing data were handled using direct maximum likelihood estimation in the structural model). Whereas 33.2 % of the sample was able to identify the presence of an acutely stressful event that may have led to the development of social phobia, 45.6 % identified the presence of chronic stress around the time of onset. Two regression models were evaluated using the presence of acute and chronic stress as dependent variables, respectively. To control for the potential confounding effects of duration of social phobia, age of onset and current age were again simultaneously entered into the regression equation as independent variables. As shown in Table 3 and consistent with hypotheses, age of onset significantly predicted the presence of acute stress at the time of onset (B=.44, p<.001). In other words, later onset social phobia was more likely to emerge in the context of an acutely stressful event (odds ratio=1.55, 95 % confidence interval=1.23 to 1.95). In contrast, age of social phobia onset did not predict the presence of chronic stress at the time of disorder onset.

Discussion

This study aimed to clarify the relevance of social phobia age of onset in predicting symptoms of anxiety (e.g., social phobia, autonomic arousal) and depression, functional impairment, and vulnerability-stress risk factors (e.g., neuroticism/behavioral inhibition, extraversion, perceived control). Nearly half of

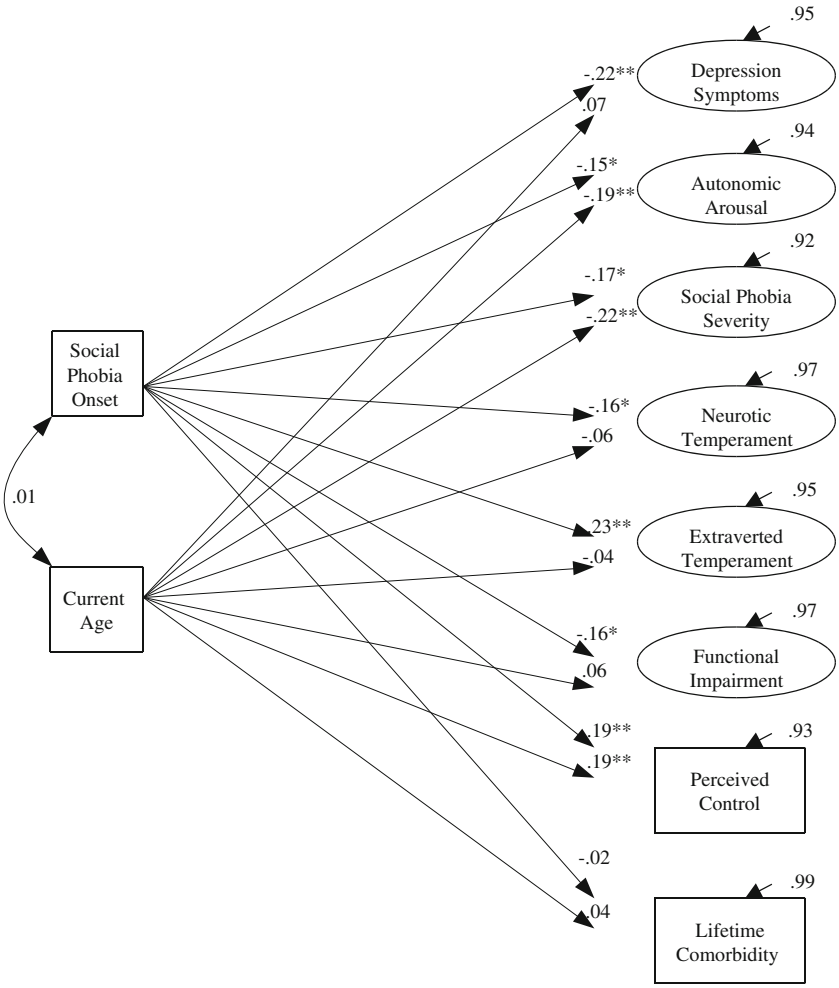

Fig. 1 Completely standardized solution of the structural model. All residual variances among endogenous variables were permitted to be intercorrelated (not shown for presentational clarity). * $p<.05$, ** $p<.01$

the sample reported that the disorder began either prior to age 10, or between ages 14 and 17. This finding is generally consistent with studies that have found a bimodal pattern of onset in early childhood and mid-adolescence (e.g., Juster and Heimberg 1995; Stein et al. 2001). Unlike prior studies evaluating relationships between age of social phobia onset and psychopathological outcomes, duration of social phobia was controlled for by simultaneously evaluating age of onset and current age as independent variables in the structural and logistic regression models. To our knowledge, this is the first study to examine the effects of social phobia age of onset while controlling for the potentially confounding effects of duration of the illness (i.e., longstanding social phobia may better account for current severity of depression, anxiety, and impairment). Although this analytical approach was unique, duration of social phobia was not positively associated with any of the outcomes in the structural model (i.e., longer duration did not predict increased severity or impairment). Thus, despite controlling for duration, our findings were largely in accord with the extant literature (e.g., Dalrymple et al. 2007; Dalrymple and Zimmerman 2011; Ruscio et al. 2008; Wittchen et al. 1999); earlier onset social phobia was significantly associated with more severe symptoms of social phobia, autonomic arousal, and depression. Additionally, earlier age of onset was found to predict increased levels of impairment (e.g., in work, school, relationships); a finding

Table 3 Logistic regression models of age of social phobia onset predicting the presence of acute and chronic stress at time of onset

Predictor variables	Model		
	B	*t*	OR (95 % CI)
	Presence of acute stress		
Age of onset	.44	13.37***	1.55 (1.23–1.95)
Current age	−.02	1.81	.98 (.96–1.01)
Constant:	−1.30	2.41*	
	Presence of chronic stress		
Age of onset	.13	1.42	.88 (.81–1.09)
Current age	.02	3.93*	1.02 (1.00–1.05)
Constant:	.55	1.10	

OR = Odds Ratio; 95 % CI = 95 % Confidence Interval

*$p<.05$, ***$p<.001$

that is in line with prior studies demonstrating significant associations between untreated social phobia and poor functioning (e.g., school dropout, unemployment; Wittchen and Beloch 1996; Wittchen et al. 1999).

Age of social phobia onset was also examined in relation to factors that are believed to function as vulnerabilities in the development of emotional disorders. In general, individuals with earlier onset social phobia exhibited more pronounced levels of vulnerability factors believed to be influenced by both genetics and early environmental experiences. Despite the fact that several prominent conceptual models (e.g., Barlow 2000; Ollendick and Hirshfeld-Becker 2002) have underscored the importance of risk factors such as neuroticism, behavioral inhibition, and perceived control in the development of social phobia, this is the first study to evaluate the relevance of age of social phobia onset in predicting these vulnerabilities. Although the cross-sectional nature of our study design precludes steadfast conclusions about predispositional effects of risk factors in the development of social phobia, our findings are generally consistent with Barlow's (2000) etiological model for the emotional disorders. Specifically, Barlow's model emphasizes the importance of generalized biological (e.g., neuroticism, behavioral inhibition, extraversion) and generalized psychological (e.g., perceived control) vulnerabilities in the development of anxiety disorders such as social phobia. A combination of high Neurotic Temperament (i.e., a tendency to experience negative affect such as anxiety, shame, and guilt), low Extraverted Temperament (i.e., a tendency to be socially withdrawn or shy), and low Perceived Control (i.e., beliefs that the occurrence of unpleasant events and negative affect are uncontrollable) may predispose individuals to develop social phobia at a very young age. These factors, along with a more specific vulnerability to view social evaluation as dangerous, may lead individuals to: (1) interpret interpersonal interactions and their associated physical sensations and emotions as negative, and (2) view these events as stressful or threatening and their associated emotional reactions as out of their control. Children and adolescents who are exposed to social interactions that are subjectively experienced as an uncontrollable threat and elicit negative affect may subsequently develop earlier onset social phobia. In contrast, lower levels of Neurotic Temperament and higher Extraverted Temperament and Perceived Control may serve as a buffer against developing social phobia at early age.

Collectively, findings from the structural regression model have significant clinical implications with regard to the importance of effective detection and intervention of social phobia early in its development. Early detection and intervention may help reduce some of the long-term negative consequences of early onset social phobia, such as increased severity of the disorder, symptoms of anxiety and depression, and functional impairment. Likewise, interventions aimed at curtailing the effects of vulnerability factors such as neuroticism/ behavioral inhibition, extraversion, and perceived control may also assist in abating some of the negative outcomes associated with early onset social phobia. Administration of behavioral tests (e.g., laboratory observation; see Biederman et al. 2001) and self-report questionnaires (e.g., the Social Phobia and Anxiety Inventory for Children; see Beidel et al. 1995) shown to predict social phobia in childhood may allow clinicians to detect the disorder early in its course and subsequently recommend appropriate interventions. As reviewed by Segool and Carlson (2008), effective treatments for childhood social phobia range from cognitive-behavioral therapy to a combination of psychoeducation (e.g., information for parents and children about the nature and maintenance of social anxiety) and pharmacotherapy (e.g., selective serotonin reuptake inhibitors).

In addition to evaluating outcomes of psychopathology and vulnerabilities factors, our study also examined the role of stress in the differential onset of social phobia. Whereas earlier research (e.g., Stemberger et al. 1995) revealed that approximately one-half of adults suffering from social phobia could identify a single embarrassing event that may have contributed to the onset of their disorder, approximately one-third (i.e., 33.2 %) of our sample identified the presence of an acutely stressful event that may have led to the development of social phobia. Logistic regression was used to demonstrate that later onset social phobia predicted the presence of an acute stressor at the time of onset; a one unit increase in age of onset was associated with a 1.55 times greater risk of having experienced a specific event related to the development of social phobia. In contrast, no significant association was found between age of onset and the presence of chronic stress. Considering results from the structural regression model (i.e., associations between early onset social phobia and temperament vulnerabilities) in conjunction with conditioning-focused etiological models of social phobia (e.g., Mineka and Zinbarg 1995), these findings may indicate two pathways to the development of social phobia. Whereas individuals with earlier onset social phobia may develop the illness primarily because of vulnerabilities related to temperament and perceptions of control (but not chronic or acute stress, i.e., vulnerability-only etiology), the disorder may be more likely to emerge in adolescence or adulthood among individuals with certain emotional disorder vulnerabilities who also experience an acutely stressful event (i.e., vulnerability-stress etiology) such as having a panic attack during a speech, moving, or beginning a new educational milestone (e.g., starting middle, high school, or college).

Our evaluation of the relevance of age of onset to the psychopathology of social phobia had a number of strengths including controlling for duration of illness, multiple indicators for the psychopathological outcomes and vulnerability factors (i.e., using a structural equation modeling to account for measurement error of the constructs), and a diagnostically diverse clinical sample. Despite the strengths of our study, there are also several limitations to consider. First, age of social phobia onset and the occurrence of acute and chronic

stress were assessed using retrospective self-report. This method of assessment may be influenced by memory deficits (especially among individuals with anxiety and depression) as well as recall biases (cf., Henry et al. 1994). However, this study attempted to increase the reliability of these assessments by creating categories of age of onset that broadly encompassed important developmental periods (e.g., elementary, middle, and high school, after college, etc.) and types of stress (e.g., acute versus chronic). Participants were further aided in recall by the interviewer asking about general life stressors or adverse experiences that might have led to developing social phobia symptoms. Moreover, a recent study that compared methods of assessing adverse childhood experiences revealed similar rates of reported adverse life events for both prospective and retrospective assessment methods (Hardt et al. 2010). This may indicate that retrospective reporting of such events may be more reliable than previously suggested. Regardless, future studies would benefit from following participants starting in childhood and assessing factors related to onset of the disorder closer to its first emergence. Likewise, the measurement of the vulnerability factors as well as anxiety and depressive symptoms solely at the time of the diagnostic assessment may have led these constructs to be influenced by patients' mood states or heightening symptoms (e.g., mood state distortion, see Brown 2007). Measurement of traits and symptoms at multiple time points (i.e., assessing vulnerability factor prior to the development of psychopathology) would help minimize the influence of natural mood fluctuations while allowing an opportunity to examine the reliability of participants' reports.

Finally, the conclusions of this study are limited given the sizes of the effects of age of onset on the outcomes of interest; despite statistical significance, age of social phobia onset did not demonstrate medium or large effects on any of the outcomes in the structural model (i.e., potentially suggesting that this study was overpowered). Moreover, epidemiological studies have demonstrated that the majority of people with social phobia do not seek treatment (i.e., Grant et al. 2005), thus the present findings may not generalize to non-clinical samples. While our findings about the relationships between age of onset and psychopathological outcomes and vulnerability-stress factors provide important information (e.g., suggesting the potential utility of early detection and treatment of social phobia), future studies could expand the literature by using a longitudinal approach in clinical and non-clinical community samples to evaluate the relevance of age of social phobia onset. For instance, research may aim to determine how age of onset predicts trajectories of various clinical outcomes (e.g., social phobia and depression severity, treatment response).

Acknowledgments This study was supported by Grant MH039096 from the National Institute of Mental Health to the fifth author. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute of Mental Health or the National Institutes of Health.

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