Research Article

THE DEVELOPMENTAL PSYCHOPATHOLOGY OF SOCIAL ANXIETY IN ADOLESCENTS

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To evaluate a developmental psychopathology approach for understanding adolescent social anxiety, parent-reported predictors of social anxiety were examined in a nonclinical sample of adolescents. Structured diagnostic interviews were obtained from biological parents of 770 participants. Potential risk factors assessed included child characteristics: negative affect, shyness, separation anxiety disorder, and childhood chronic illness, as well as parent characteristics: major depression, panic disorder, and agoraphobia. Adolescent social anxiety was measured multiple times during high school. Findings indicate stability in social anxiety symptoms across time. Parent-reported, childhood negative affect, shyness, and chronic illness as well as parental panic disorder or agoraphobia were associated with adolescent social anxiety. Interactions were observed between parent-reported childhood shyness and gender and between parent-reported childhood shyness and parent-reported childhood chronic illness in the prediction of social anxiety. Parent-reported childhood shyness was a stronger predictor of adolescent social anxiety in females compared to males. The combined effect of subjects being positive for both parent-reported childhood shyness and parent-reported childhood chronic illness was greater than would be expected based on additive effects. This study provides support for a multifactorial and developmentally informed understanding of adolescent social anxiety. Depression and Anxiety 25:200–206, 2008.

Key words: social anxiety; adolescents; risk factors; parents

INTRODUCTION

Who is at risk for developing social anxiety disorder (SAD) in youth? To best address this question, Ollendick and Hirshfeld-Becker [2002] suggest using a developmental psychopathology framework, which incorporates the following tenets: (1) because manifestations of disorder change, studies should focus on symptom continuities and discontinuities over time; (2) factors from different domains and developmental periods combine and interact to impact risk; (3) studying both normal and abnormal development informs our understanding of psychopathology [Cicchetti and Cohen, 1995]. Although heuristically appealing, applications of this model are rare in the literature, and only a few studies have tested multiple, interacting predictors of social anxiety disorder (SAD) over time. Evidence abounds for both individual and parental predictors of child social anxiety. For example, within

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the individual domain, behavioral inhibition and shy temperament both elevate risk [Hayward et al., 1998; Prior et al., 2000; Schwartz et al., 1999]. Of course, the majority of those with symptoms or disorder do not have histories of shyness or an inhibited temperament [Hayward et al., 1998; Prior et al., 2000], highlighting the need to identify other factors that might contribute to the development of SAD. A range of parental factors have been shown to impact the development of offspring SAD. For example, parental psychopathology, including anxiety disorders and depression, has been associated with SAD in samples of adolescents. In addition, the quality of the parent–child relationship [Lieb et al., 2000] and an overprotective parenting style have both been important predictors [Masia and Morris, 1998].

In spite of the many well-designed studies suggesting important contributions for individual factors contributing to the development of SAD, there have been few attempts to describe study results in a developmental psychopathology framework. In this study, the evaluation of social anxiety incorporates features of the developmental psychopathology model: continuity of symptoms; multiple and interacting factors from different domains; and predictors across a spectrum of illness. Further, to add to previous findings, this study incorporates data drawn from parents. The use of parent-reported information provides cross validation of previous studies using only subject report [Chartier et al., 2001; Hayward et al., 1998] and allows examination of factors best obtained from parents (e.g., parental psychopathology).

Constructs were evaluated in this study if they had been associated with SAD in previous studies. These variables included: negative affect [Hayward et al., 1998]; shyness [Prior et al., 2000]; separation anxiety [Warren et al., 1997]; childhood loss [Chartier et al., 2001]; parental major depression [Lieb et al., 2000]; and parental panic disorder or agoraphobia [Goldstein et al., 1994]. We also assessed childhood chronic illness because of the reported association between childhood somatic symptoms and later anxiety disorders [Ehlers, 1993]. The primary outcome measure is a dimensional measure of social anxiety symptoms.

METHOD

PARTICIPANTS

The eligible sample consisted of 95% (N = 2,365) of all students enrolled in four high schools near San Jose, California. These schools were racially diverse and located in middle-class, suburban areas. Details of this sample characteristics are described elsewhere [Hayward et al., 1998].

Parent sample. Because only two of the four schools allowed access to parents, eligibility was reduced considerably from the full sample. A total of 700 interviews were obtained from biological mothers and 529 from biological fathers. Of these, 459 represented interviews on both biological parents. To avoid differences in risk factor status based solely on differences associated with having two versus one parent contribute information, only one parent interview was used for data analyses. We chose to use the biological mother’s interview if available. Otherwise, the biological father’s interview was used. This resulted in a sample of 770 subjects on whom there was either a maternal interview (n = 700) or paternal interview (n = 70). Tests comparing means and frequencies of all predictors as well as all outcomes revealed no differences between data obtained from mothers versus fathers. Finally, to assess selection bias associated with having a parent interview, means and frequencies of demographic and outcome variables were compared between adolescents with and without a parent interview (n = 770 and 1621, respectively). Those adolescents for whom parent data were available differ in some ways. They were slightly younger and endorsed less anxiety sensitivity at entry, and their parents were less likely to be white or have graduate degrees.

PROCEDURE

Yearly evaluations took place each spring for 4 years. At every assessment, participants completed a structured clinical interview as well as a self-report measure. Passive parental consent was obtained, and identification numbers were used to protect subjects’ confidentiality. Different lengths of follow-up are reported because subjects entered the study at any point during the 4 years and because of attrition. Many students who left the participating schools continued to be followed for the duration of the study. Attrition averaged 15% annually and was caused by students dropping out of or changing schools (75%), absenteeism (20%), and refusal (5%). A more detailed description of attrition is described in Hayward et al. [1998].

Parents were administered structured interviews over the phone by four graduate students. These interviewers were trained and supervised by the first author. In two 4-hr training sessions, they watched and discussed videotaped interviews. During data collection, the first author reviewed all interviews that yielded a positive or uncertain diagnosis. When more information was needed to assess a diagnosis, the interviewer phoned the parent again to gather this information. Only parents who spoke English well enough to understand the interview questions were included. Biological fathers were only interviewed if biological mothers were unavailable.

MEASURES

Outcome

Social anxiety. A dimensional assessment of social anxiety was obtained with the social phobia subscale of the Social Phobia and Anxiety Inventory (SPAI; Turner et al. [1989]), which each adolescent completed. The SPAI is an empirically derived self-report instrument.
that measures cognitive, somatic, and behavioral responses to potentially fear-producing social situations and assesses the degree of distress or impairment resulting from these experiences. Clark et al. [1994] reported high internal consistency (Cronbach $\alpha = .97$), as well as reasonable construct and concurrent validity, for the social phobia subscale in an adolescent sample [Clark et al., 1994]. In this sample the Cronbach’s $\alpha$ for the social phobia subscale was .98.

**Predictors**

**Parent-reported child characteristics.** Negative affect and shyness in the child were measured with the parent version of the Emotionality Activity Sociability Scale, a well-validated measure of temperament developed by Buss and Plomin [1984]. The overall instrument consists of 20 questions and is designed to measure aspects of temperament. The instrument has been modified by Buss and Plomin [1984] to refer to the respondent's child, rather than the respondent himself or herself. For example, “I get emotionally upset easily,” has been modified to “Child gets emotionally upset easily.” Research has found this scale to possess acceptable reliability and validity [Buss and Plomin, 1984; Mathiesen and Tambs, 1997].

To diagnose history of Separation Anxiety Disorder in the adolescent, pertinent parts of the Kiddie Schedule for Affective Disorders and Schizophrenia were administered to parents (modified by Last [1986] using DSM-III-R criteria, unpublished manuscript). Interviewers assessed specific symptoms of separation anxiety disorder by querying experiences of the child from the beginning of school through the present. Items included questions regarding school reluctance or refusal and fear of separation through calamitous events. This instrument has been shown to have good concurrent validity and test–retest reliability by Kaufman et al. [1997].

Childhood loss was assessed by asking the parent if he or she was divorced or separated during the child’s lifetime. In addition, the parent was asked if the child had ever experienced the death of a sibling or parent. Childhood chronic illness was assessed by asking the parent if his or her child ever suffered from a chronic illness.

**Parent characteristics.** The pertinent parts of the structured clinical interview for the DSM-III-R nonpatient version [Spitzer et al., 1997] were used to diagnose panic disorder, agoraphobia, and major depression in the parents in the nonclinical sample.

**DATA ANALYSIS**

In the entire sample (i.e., those with and without parent data), correlations between social anxiety scores were examined across all time points to assess stability of this measure over time. Among those with parent-reported data, mean scores on the SPAI across all available time points were used to create an averaged social anxiety score. This average then served as the primary outcome variable. To examine bivariate associations between each predictor and mean SPAI scores, Pearson correlations were used for continuous variables and Student's $t$-tests were calculated for dichotomous predictors. For each dichotomous predictor, effect sizes were calculated as the difference between mean SPAI scores for those with and without the predictor variable, divided by the pooled standard deviation on the SPAI.

**Signal detection.** To explore multivariate associations, we used signal detection methods [Link, 1994]. Signal detection uses recursive partitioning, which is an empirically derived iterative process that identifies optimal cutpoints in predicting an identified dichotomous outcome. The cutpoints on the predictor measures create subgroups of participants who are more versus less likely to score positively on the outcome. For example, signal detection might produce a result as follows: a score of 20 on a shyness measure yields the most sensitive and specific prediction of positive social anxiety compared to all other measures in the model; 75% of those who exceed 20 on shyness are categorized as anxious whereas only 15% of those who score below 20 are anxious. In this study, the grouping variable was created to represent two levels of social anxiety, defined as highest 20th percentile versus lowest 80th percentile on the SPAI.

We decided to use signal detection because it is immune to the impact of multicolinearity among predictor variables [Kiernan et al., 2001], an immunity that circumvents potential bias in estimated weights for predictors. We also decided to use signal detection methods over regression because the former is better suited to identify interactions.

Signal detection methods generate decision trees that balance optimal sensitivity and specificity in discriminating those who do from those who do not meet some criteria. In this study, we focus on developing optimally efficient algorithms to best distinguish those with high scores on a measure of social anxiety from the remainder of the sample.

We used software from MIRECC@stanford.edu to run the signal detection analyses. All predictors described in the Method section were entered into the program. The criterion for terminating the iterative process was set at a $\chi^2$ of $P < .01$. Although informative, these analyses are exploratory, in part because the method tests every permutation (e.g., shyness = 1, shyness = 2, shyness = 3, etc.) for every chosen variable in the model. As a result, findings require corroboration in future validation samples.

**RESULTS**

**STABILITY OF SOCIAL ANXIETY OVER TIME**

Means, standard deviations, and intercorrelations among SPAI scores at each time point (years 1–4) are
presented in Table 1. SPAI scores at all time points were significantly correlated \( (P < .001) \). These means are consistent with those obtained in other community samples of adolescents (e.g., Clark et al. [1994]; \( M = 59.7, SD = 31.7 \)).

### BIVARIATE ASSOCIATIONS

Pearson correlation coefficients between both parent-reported childhood emotionality and parent-reported childhood shyness with adolescent-reported SPAI scores were significant but small (.08 and .17 respectively). Table 2 shows the results of the \( t \)-tests that include effect sizes for the associations between the dichotomous predictors and adolescent social anxiety. Childhood chronic illness as well as parental panic disorder or agoraphobia were significant predictors of adolescent SPAI scores.

To test for gender effects, centered transformations of gender and each predictor variable, as well as their interactions, were entered into multiple linear regression models to predict SPAI scores. Although there was no main effect for gender, an interaction \( (\beta = .07, t(636) = 1.8, P = .08) \) emerged between childhood shyness and gender \( (\beta = .09, t(636), P = .03) \). Childhood shyness was more strongly associated with social anxiety in females compared to males. To illustrate this interaction, a median split was created on the childhood shyness variable creating four groups (male-low shyness, male-high shyness, female-low shyness, female-high shyness). The interaction between childhood shyness and gender using these four groups is illustrated in Figure 1.

### EXPLORATORY ANALYSES

Signal detection using recursive partitioning. The results of the recursive partitioning are shown in Figure 2. High social anxiety was defined as or exceeding the 20th percentile on the SPAI. Four important subgroups were identified. Depending on this group membership, risk for high social anxiety ranged from 13 to 52%. Childhood history of chronic illness was shown to be the most important predictor of high social anxiety.

#### Table 1. Means, standard deviations, ranges, and intercorrelations among SPAI scores at each assessment year

<table>
<thead>
<tr>
<th>Year</th>
<th>SPAI Year 1 ((N = 964))</th>
<th>Year 2 ((N = 1,606))</th>
<th>Year 3 ((N = 1,299))</th>
<th>Year 4 ((N = 1,112))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Year 1</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Year 2</td>
<td>.55</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Year 3</td>
<td>.54</td>
<td>.61</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Year 4</td>
<td>.48</td>
<td>.60</td>
<td>.66</td>
<td>—</td>
</tr>
<tr>
<td>(M (SD))</td>
<td>53.0 (33.3)</td>
<td>58.4 (34.0)</td>
<td>53.0 (34.3)</td>
<td>48.5 (33.7)</td>
</tr>
<tr>
<td>Range</td>
<td>0–192</td>
<td>0–190</td>
<td>0–170</td>
<td>0–192</td>
</tr>
</tbody>
</table>

*Note:* Sample sizes vary at each time point because of some missing cases and the time point at which adolescents entered the study. All correlations are significant at \( P < .001 \), two-tailed. SPAI, Social Phobia Anxiety Inventory (social phobia subscale).

#### Table 2. Means, standard deviations, \( t \)-tests, and effect sizes for dichotomous predictors of adolescent social anxiety among those with and without predictors

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Mean SPAI ((SD)) predictor absent</th>
<th>Mean SPAI ((SD)) predictor present</th>
<th>( t )</th>
<th>( df )</th>
<th>( P )</th>
<th>ES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Separation anxiety disorder</td>
<td>53 (28)</td>
<td>61 (37)</td>
<td>1.6</td>
<td>644</td>
<td>.12</td>
<td>.28</td>
</tr>
<tr>
<td>Parental depression</td>
<td>53 (29)</td>
<td>59 (34)</td>
<td>1.8</td>
<td>708</td>
<td>.08</td>
<td>.20</td>
</tr>
<tr>
<td>Parental panic disorder or agoraphobia</td>
<td>53 (30)</td>
<td>66 (31)</td>
<td>2.7</td>
<td>705</td>
<td>.01</td>
<td>.47</td>
</tr>
<tr>
<td>Childhood chronic illness</td>
<td>52 (29)</td>
<td>63 (31)</td>
<td>2.7</td>
<td>656</td>
<td>.01</td>
<td>.34</td>
</tr>
<tr>
<td>Childhood loss</td>
<td>53 (30)</td>
<td>54 (30)</td>
<td>0.6</td>
<td>710</td>
<td>.60</td>
<td>.04</td>
</tr>
</tbody>
</table>

ES, effect size.

Figure 1. Interaction between childhood shyness and gender in relation to Social Phobia and Anxiety Inventory (SPAI) scores.

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illness was identified as the most optimal predictor. Participants with such a history had a 31% chance of experiencing high social anxiety (the right-hand side of Fig. 2). Among those with a history of illness, childhood shyness further differentiated risk. Whereas a lower score (< 3) on the measure of shyness was associated with a 21% risk for high social anxiety, those with higher shyness scores had a 52% chance of being in the high social anxiety group. Overall, those without a chronic illness history had a 15% chance of having high social anxiety (the left-hand side of Fig. 2). Within this group, shyness further differentiated those at higher versus lower risk for social anxiety. Those who scored above 3.2 on the measure of shyness had a 23% chance of being in the high social anxiety group. In contrast, scoring below that cutoff yielded a 13% risk. These results indicate an interaction between childhood shyness and childhood chronic illness in the prediction of social anxiety. Subjects with both factors are four times more likely to have high social anxiety compared to those with neither. The risk of having high social anxiety with only one risk factor (i.e., either childhood chronic illness or childhood shyness) is 1.6 and 1.8, respectively (risk ratio compared to those with neither).

DISCUSSION

In this study, a developmental framework was used to examine factors that predict social anxiety in youth. The tenets of developmental psychopathology are emphasized. First, there is evidence for continuity in social anxiety symptoms across time during adolescence. As shown in Table 1, there was a high degree of stability in SPAI scores throughout the 4 years in high school. Other studies have demonstrated both heterotypic and homotypic continuity of social anxiety over time. Most of those with SAD in youth do not have SAD as adults but do have either another anxiety disorder or major depression (heterotypic continuity; Pine et al. [1998]). In the Munich study there was substantial oscillation across levels of social anxiety severity over time but substantial stability for having some degree of social anxiety (homotypic continuity; Merikangas et al. [2002]).

Second, multiple factors from different domains (i.e., parental psychopathology, child temperament, and childhood illness experiences) are all associated with higher social anxiety during adolescence. Other studies support these findings. Lieb et al. [2000] found evidence for associations between SAD and both parent psychopathology and parenting style in youth. Longitudinal studies demonstrate that childhood behavioral inhibition, shyness, and negative affect increase risk for SAD or social anxiety symptoms in adolescents [Hayward et al., 1998; Prior et al., 2000; Schwartz et al., 1999]. Third, potential risk factors were identified from a nonclinical sample providing some evidence that these factors contribute across a spectrum of symptomatology. Fourth, interactions between important risk factors were identified. Regression analyses yielded a significant interaction between childhood shyness and gender in that the association between shyness and social anxiety is greater in females than in males. In addition, the exploratory signal detection analyses indicated that childhood illness and shyness alone increase the risk for social anxiety symptoms only slightly ($RR = 1.6$ and $1.8$, respectively), whereas they increase the risk for high social anxiety fourfold when present together.

This study also provides information regarding predictors for adolescent social anxiety symptoms using parent-reported data. The use of parent-reported data provides cross validation for risk factors identified from a previous study that used only adolescent-reported risk factors [Hayward et al., 1998] and offers tests of additional risk factors that are best obtained from
parents. This corroboration with parental informants in combination with our previous report that used adolescent reports supports the emerging data indicating that childhood negative affect and shyness are related to the later development of social anxiety.

Although there are studies that suggest illness during childhood increases the risk for panic symptoms [Goldstein et al., 1994], this study marks the first time childhood chronic illness has been associated with social anxiety symptoms. Indeed no one has investigated this link to date. It may be that childhood illness also represents a nonspecific risk for anxiety generally, but alternate explanations abound. For example, parents with a sick child may behave more cautiously in an effort to protect this child, which may inadvertently exacerbate anxious or avoidant tendencies. Further speculation awaits additional studies that will need to address this construct with validated measures and more details regarding the specific illnesses.

There are other important factors not measured in this study that are likely to be important in a developmental psychopathology model of social anxiety. For example, parenting style (particularly parent enhancement of avoidant responses) and peer rejection during adolescence may create or exacerbate social anxiety for those with an inhibited temperament [La Greca and Lopez, 1998]. Further, neurohormonal and genetic factors related to social behavior also contribute, and ought to be included in a more complete, multifactorial understanding of social anxiety [Young, 2002].

There are other limitations to consider in interpreting our results. In some cases, the parent was likely interviewed after the onset of their adolescent’s social anxiety symptoms. This may have impacted the parents’ report of risk factors, for example their recollection of their child’s shyness. In addition, a better assessment of childhood illness would have included multiple items rather than the one question we used here. We used data from only one parent to measure parental disorders. This approach may have underestimated family psychopathology and may help explain why we failed to find an association between parental depression and social anxiety in contrast to other investigations. Because this study was designed to focus on panic symptoms in adolescents, the family history assessment did not include assessing parental SAD. Future studies on risk factors for SAD would benefit from including family SAD history in addition to the other factors we assessed here. Finally, that only half of the four schools allowed us to collect data from parents may introduce biases not assessed with our measures; therefore this limitation should also be considered when interpreting our findings.

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