Age of onset of social anxiety disorder in depressed outpatients

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Abstract

Onset of social anxiety disorder (SAD) often precedes that of major depressive disorder (MDD) in patients with this comorbidity pattern. The current study examined the association between three SAD onset groups (childhood, adolescent, adulthood) and clinical characteristics of 412 psychiatric outpatients diagnosed with MDD and SAD based on a semi-structured diagnostic interview. Childhood and adolescent SAD onset groups were more likely to report an onset of MDD prior to age 18 and have made at least one prior suicide attempt compared to the adulthood onset group. The childhood SAD onset group also was more likely to have chronic MDD, poorer past social functioning, and an increased hazard of MDD onset compared to the adulthood onset group. Findings suggest that patients with an onset of SAD in childhood or adolescence may be particularly at risk for a more severe and chronic course of depressive illness.

Keywords:
Social phobia
Major depression
Comorbidity
Severity
Impairment

Social anxiety disorder (SAD) is the fourth most common psychiatric disorder in the United States (Kessler et al., 2005), and the most common comorbid anxiety disorder in patients with major depressive disorder (MDD; Belzer & Schneier, 2004). Despite high occurrence of SAD with MDD, SAD often goes under-recognized and under-treated in depressed outpatients (Zimmerman & Chelminski, 2003). Patients with SAD rarely seek treatment primarily for it and instead seek treatment for another, more acute disorder such as MDD (Lecrubier, 1998). However, when directly asked, approximately 75% of people diagnosed with SAD desire treatment for it in addition to treatment for MDD (Dalrymple & Zimmerman, 2007).

Several studies have found that age of onset of SAD often precedes age of onset of MDD (Beesdo et al., 2007; Brown, Campbell, Lehman, Grisham, & Mancill, 2001; Dalrymple & Zimmerman, 2007; Kessler, Stang, Wittchen, Stein, & Walters, 1999; Parker et al., 1999), with a typical onset of SAD around mid-adolescence (Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992). However, re-analysis of epidemiological studies has found two peaks of onset of SAD, with some patients reporting an onset before the age of 5 and others reporting an onset in mid-adolescence (Juster, Brown, & Heimberg, 1996; Juster & Heimberg, 1995; Stein, Chavira, & Jang, 2001). In contrast, the average age of onset of MDD ranges from 25 to 35 years of age (Parker et al., 1999; Weissman et al., 1999; Zisook et al., 2007).

Research has shown an association between an early onset of SAD and greater severity, such as the generalized subtype (Wittchen, Stein, & Kessler, 1999). In addition, a childhood onset of SAD has been associated with greater severity of SAD symptoms throughout childhood and adolescence compared to an adolescent or adulthood onset (Dalrymple, Herbert, & Gaudiano, 2007), and those with a childhood onset reported greater severity of SAD after 12 weeks of cognitive behavior therapy compared to those with an adolescent or adulthood onset despite similar pre-treatment scores (Dalrymple et al., 2007). For MDD, the following factors have been associated with an early onset: increased familial loading for depression (Klein, Lewinsohn, Seeley, & Rohde, 2001); female gender (Kornstein et al., 2000); higher rates of alcohol use and other substance use disorders (Klein et al., 1999); elevated rates of subsequent depressive episodes in early adulthood (Weissman et al., 1999); more suicidality (Kovacs, Goldston, & Gatsonis, 1993); greater chronicity and disability (Parker, Roy, Hadzi-Pavlovic, Mitchell, & Wilhelm, 2003); higher numbers of medical and psychiatric hospitalizations (Klein et al., 1999); and greater work, family, and social impairment (Rao et al., 1995). An early onset of MDD also is associated with higher rates of anxiety disorders (Biederman, Farone, Mick, & Lelon, 1995; Parker et al., 2003), particularly SAD and specific phobia (Alpert et al., 1999).

Although prior research has examined an early onset of SAD and MDD separately, few studies have examined the effect of an early versus late onset of comorbid SAD on the severity, course of illness, and functional impairment of MDD. In a prospective, epidemiological study on the subsequent risk of depression in patients with comorbid depression and SAD (Beesdo et al., 2007), the risk of subsequent depression increased by twofold in individuals with comorbid depression and SAD compared to depression alone. This
was most pronounced for individuals who experienced an onset of SAD prior to ages 11 and 16 rather than at later ages, which suggests that the earlier that SAD begins the more likely the individual will experience future depression.

Further research needs to be conducted on onset of SAD and subsequent onset of MDD, given that prior research has found high comorbidity rates between SAD and MDD (Belzer & Schneier, 2004), an age of onset of SAD often preceding that of MDD (Beesdo et al., 2007; Belzer & Schneier, 2004; Brown et al., 2001; Kessler et al., 1999; Parker et al., 1999), and an association between the presence of comorbid SAD and greater severity (Dalrymple & Zimmerman, 2007). Additional comorbidity was high in this sample, with patients on average having at least two other current Axis I diagnoses in addition to MDD and SAD (Table 3).

1.2. Procedure

Individuals presenting for treatment were asked to participate in a diagnostic evaluation prior to meeting with their treating clinician, using the Structured Clinical Interview for DSM-IV for Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1996). Procedures for the study were approved by the institutional review committee at Rhode Island Hospital, and informed consent was obtained before administering the SCID. Diagnosticians were research assistants with bachelor's degrees in social or biological sciences and doctoral-level clinical psychologists. Information regarding the training of diagnosticians has been presented elsewhere (Zimmerman & Mattia, 1999). Forty-eight joint-interview reliability evaluations conducted over the entire course of the project have demonstrated excellent reliability for mood and anxiety disorders (Dalrymple & Zimmerman, 2007).

1.3. Measures

Depression severity was rated by diagnosticians using the Clinical Global Impressions Scale (CGI; National Institute of Mental Health, 1985), and overall impairment was rated using the Global Assessment of Functioning Scale (GAF). Interrater reliability for CGI and GAF scores was high (intraclass correlation coefficient [ICC] = 0.79 and 0.80, respectively). Adolescent and current social functioning and time out of work due to psychopathology were measured using items from the Schedule for Affective Disorders and Schizophrenia (SADS; Spitzer & Endicott, 1977). Adolescent and current social functioning were rated by diagnosticians on Likert scales ranging from 1 (superior) to 7

### Table 1

Demographic characteristics of depressed outpatients with a childhood, adolescent, or adulthood onset of comorbid social anxiety disorder.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total sample (n = 412)</th>
<th>Child onset (n = 272)</th>
<th>Adol. onset (n = 74)</th>
<th>Adult onset (n = 66)</th>
<th>Statistic</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, M (SD)</td>
<td>38.0 (11.0)</td>
<td>38.3 (11.1)</td>
<td>34.7 (10.8)</td>
<td>40.2 (11.5)</td>
<td>$F = 4.64$</td>
<td>0.01$^{b}$</td>
</tr>
<tr>
<td>Gender, n (%)</td>
<td>280 (68.0)</td>
<td>187 (68.8)</td>
<td>48 (64.9)</td>
<td>45 (68.2)</td>
<td>$\chi^2 = 0.41$</td>
<td>0.82</td>
</tr>
<tr>
<td>Male</td>
<td>132 (32.0)</td>
<td>85 (31.2)</td>
<td>26 (35.1)</td>
<td>21 (31.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race, n (%)</td>
<td>346 (84.0)</td>
<td>231 (84.9)</td>
<td>61 (82.4)</td>
<td>54 (81.8)</td>
<td>$\chi^2 = 6.46$</td>
<td>0.78</td>
</tr>
<tr>
<td>Caucasian</td>
<td>27 (6.6)</td>
<td>15 (5.5)</td>
<td>7 (9.5)</td>
<td>5 (7.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>African Amer.</td>
<td>11 (2.7)</td>
<td>7 (2.6)</td>
<td>2 (2.7)</td>
<td>2 (3.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asian</td>
<td>4 (1.0)</td>
<td>3 (1.1)</td>
<td>0 (0)</td>
<td>1 (1.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Portuguese</td>
<td>9 (2.2)</td>
<td>4 (1.5)</td>
<td>2 (2.7)</td>
<td>3 (4.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>137 (33.3)</td>
<td>111 (40.8)</td>
<td>24 (32.4)</td>
<td>22 (33.3)</td>
<td>$\chi^2 = 5.31$</td>
<td>0.87</td>
</tr>
<tr>
<td>Marital status, n (%)</td>
<td>157 (38.1)</td>
<td>111 (40.8)</td>
<td>24 (32.4)</td>
<td>22 (33.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married</td>
<td>137 (33.3)</td>
<td>111 (40.8)</td>
<td>24 (32.4)</td>
<td>22 (33.3)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Living together</td>
<td>3 (0.7)</td>
<td>2 (0.7)</td>
<td>0 (0)</td>
<td>1 (1.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Widowed</td>
<td>53 (12.9)</td>
<td>39 (14.3)</td>
<td>7 (9.5)</td>
<td>7 (10.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Divorced</td>
<td>42 (10.2)</td>
<td>31 (11.7)</td>
<td>7 (9.5)</td>
<td>7 (10.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never married</td>
<td>5 (1.2)</td>
<td>4 (1.5)</td>
<td>0 (0)</td>
<td>1 (1.5)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Education, n (%)</td>
<td>242 (58.7)</td>
<td>155 (57.0)</td>
<td>48 (64.9)</td>
<td>39 (59.1)</td>
<td>$\chi^2 = 15.25$</td>
<td>0.51</td>
</tr>
<tr>
<td>Less than HS</td>
<td>53 (12.9)</td>
<td>39 (14.3)</td>
<td>7 (9.5)</td>
<td>7 (10.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HS/GED</td>
<td>48 (11.7)</td>
<td>34 (12.5)</td>
<td>6 (8.1)</td>
<td>6 (9.1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>College</td>
<td>98 (23.8)</td>
<td>66 (24.3)</td>
<td>17 (22.9)</td>
<td>15 (22.7)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Graduate</td>
<td>19 (4.6)</td>
<td>12 (4.4)</td>
<td>2 (2.7)</td>
<td>5 (7.6)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: College = 2- or 4-year college degree.

Variables with significant findings are italicized.

$a$ Significant difference on post hoc comparison between childhood and adolescent onset groups.

$b$ Significant difference on post hoc comparison between adolescent and adulthood onset groups.

### Notes

1. **Demographic characteristics of depressed outpatients with a childhood, adolescent, or adulthood onset of comorbid social anxiety disorder.**

2. **Table 1**

3. **Variables with significant findings are italicized.**

4. **a** Significant difference on post hoc comparison between childhood and adolescent onset groups.

5. **b** Significant difference on post hoc comparison between adolescent and adulthood onset groups.
powerful than traditional familywise error correction procedures was used to calculate (meaning that we were willing to incur a maximum of 5% false positives) considered to still be significant if the comparison, which measures the minimum FDR that is incurred after the FDR correction ($q$-value = 0.03). This comparison remained significant when examining the FDR ($q$-value = 0.03). There was no significant difference between the childhood and adulthood onset groups on age. There were no other differences between onset groups on demographic variables.

A greater percentage of patients with an onset of SAD in adulthood also had chronic MDD compared to patients with an adolescent and adulthood onset of SAD, which remained significant after the FDR correction ($q$-value = 0.004; childhood vs. adolescent onset $\chi^2 = 11.45$, $p = 0.001$; childhood vs. adulthood onset $\chi^2 = 5.30$, $p = 0.02$). In addition, a greater percentage of patients with a childhood and adolescent onset of SAD had at least one prior suicide attempt compared to the adulthood onset group, which remained significant after the FDR correction ($q$-value = 0.04; childhood vs. adulthood onset $\chi^2 = 8.33$, $p = 0.004$; adolescent vs. adulthood onset $\chi^2 = 4.84$, $p = 0.03$). The initial comparison on CGI scores was significant, but it was no longer significant after the FDR correction ($q$-value = 0.07). There were no significant differences between groups on occurrence of prior inpatient or partial hospitalizations (Table 2).

A greater percentage of patients with a childhood onset of SAD were rated as having fair or worse social functioning as an adolescent compared to patients with an onset of SAD in either adolescence or adulthood (childhood vs. adolescent onset $\chi^2 = 4.73$, $p = 0.03$; childhood vs. adulthood onset $\chi^2 = 13.35$, $p < 0.001$), and this also remained significant after the FDR adjustment ($q$-value = 0.004). The three groups did not differ on current social functioning or time out of work in the past 5 years due to psychopathology. Regarding comorbidity, a greater percentage of patients with a SAD onset in adolescence met current criteria for an impulse control disorder compared to patients with a SAD onset in childhood ($\chi^2 = 6.25$, $p = 0.01$), but this did not remain significant after the FDR correction ($q$-value = 0.07). There were no differences for other specific Axis I disorders (Table 3).

For the Cox regression analysis (Table 4 and Fig. 1), age was entered in the first block and the SAD onset group variable in the...
Variables with significant findings are bold italicized. This comparison was not significant after the false discovery rate correction. Significant difference on post hoc comparisons between childhood and adolescent onset of SAD. Significant difference on post hoc comparisons between childhood and adulthood onset of SAD. Missing data from one childhood onset participant (n = 271). Welch's variance weighted F statistic is reported due to unequal variances. Total sample size reduced to 229 (childhood onset n = 93; adolescent onset n = 70; adulthood onset n = 66).

3. Discussion

The majority of patients with comorbid MDD and SAD in this sample had an age of onset of SAD prior to that of MDD, and these results are consistent with other studies (Brown et al., 2001; Kessler et al., 1999). In general, results from the current study suggested that an onset of SAD in childhood and adolescence was associated with a shorter time to MDD onset.
with greater severity of MDD compared to patients with a SAD onset in adulthood, in terms of an onset of MDD prior to age 18, chronic MDD, and presence of at least one prior suicide attempt. This is consistent with results from prospective studies (Beesdo et al., 2007) and suggests that the presence of SAD early in life may put an individual at increased risk of experiencing more severe or treatment-resistant forms of depressive illness.

Results from the Cox regression also indicated a shorter time to MDD onset for those with a SAD onset in childhood compared to adulthood. One could argue that these results merely reflect that once one disorder has developed it is likely that a comorbid disorder will develop later on, and that the earlier the first disorder onsets, the earlier the second one will onset. However, prior research has shown that the onset of SAD tends to precede that of all other disorders (Brown et al., 2001). In terms of social development, children are presented with social interactions from a very young age. Anxiety in social situations and social withdrawal/avoidance tend to start a cycle, such that less exposure to social interactions at a young age interferes with the normal development of social skills, which then reinforces the anxiety and may foster other negative consequences such as lower self-esteem (Messer & Beidel, 1994; Rubin & Burgess, 2001). Social anxiety and withdrawal in childhood also may be accompanied by other symptoms related to depression, such as loneliness, poor concentration, and feelings of hopelessness (Rubin & Burgess, 2001). In fact, prior research has shown that social withdrawal in childhood predicts later depression in adolescence (Rubin, Chen, McDougall, Bowker, & McKinnon, 1995). Therefore, research is converging to show that perhaps there is a specific link between early development of SAD and a later development of MDD.

Timing of SAD onset was not associated with the overall number of additional Axis I current diagnoses, or with the presence of specific disorders. Although initial results suggested a difference on the presence of a current impulse control disorder, it did not remain significant after the FDR correction (with a false positive rate of 7%). However, this variable should continue to be studied in future research, as a recent study from the National Comorbidity Survey-Replication identified a subset of individuals with SAD and an atypical pattern of anger, aggression, moderate to high sexual impulsivity, and substance use problems (Kashdan, McKnight, Richey, & Hofmann, 2009). Other studies have also found high rates of SAD in patients diagnosed with pathological gambling (Zimmerman, Chelminski, & Young, 2006) and male sex offenders with paraphilia or impulse control disorder (Hoyer, Kunst, & Schmidt, 2001).

A childhood onset of SAD in patients with MDD was associated with poorer social functioning as a teenager, but there were no differences between onset groups in terms of current social functioning and number of social fears endorsed. This suggests that despite the timing of the SAD onset the severity of SAD may be comparable at the time of presentation, at least in this treatment-seeking sample.

Results from the current study add further evidence to the existing literature advocating for the early identification and treatment of SAD (Morris, Hirshfeld-Becker, Henin, & Storch, 2004). This is particularly important because SAD has a chronic and unremitting course (Davidson, Hughes, George, & Blazer, 1994), its age of onset often precedes that of other psychiatric problems (Brown et al., 2001), and it appears to result in subsequent onset of mood disorders (Beesdo et al., 2007; Kessler et al., 1999; Stein, Fuetsch, et al., 2001). However, prior research has shown that SAD often goes under-recognized in depressed outpatients (Zimmerman & Chelminski, 2003), and this under-recognition therefore may affect patients’ ability to receive adequate treatment. In fact, emerging evidence suggests that untreated comorbid SAD may affect the treatment outcome of MDD (DeRubeis et al., 2005; Holma, Holma, Melarint, Rysala, & Isometsa, 2008), but future research needs to be conducted to further delineate this potential relationship.

Although it may be more difficult to conduct semi-structured diagnostic interviews in routine practice settings, research suggests that clinicians are 15 times more likely to identify the presence of SAD in depressed outpatients when using semi-structured diagnostic interviews compared to unstructured clinical interviews (Zimmerman & Chelminski, 2003). Given that semi-structured diagnostic interviews are lengthy and would prove to be burdensome in many outpatient mental health settings, an alternative is to administer brief self-report screening measures that can help to identify potential problem areas for further inquiry. Several evidenced-based self-report measures exist that could be used for this purpose, such as the SAD section of the Psychiatric Diagnostic Screening Questionnaire (PDSQ: Zimmerman & Mattia, 2001), the Brief Social Phobia Scale (BSPS; Davidson et al., 1991), and the Social Phobia Inventory (SPIN; Connor et al., 2000).

Some limitations should be considered when interpreting results from the current study. Data were collected from a single site and the sample was primarily Caucasian; therefore, results may not be generalizable to other settings or populations. In addition, all patients in this study were seeking treatment. Many individuals seeking treatment tend to over-report psychopathology in general (DuFort, Newman, & Bland, 1993), and therefore the results from the current study may not be generalizable to non-treatment-seeking individuals with MDD and SAD. Also because this sample was treatment-seeking, SAD may have been under-represented given that many individuals with SAD do not seek treatment due to the nature of the disorder (Olsson et al., 2000). Therefore, this research should be replicated in epidemiological samples. Furthermore, the number of statistical comparisons may have resulted in an increase in Type I error. However, the FDR correction method was applied (Benjamini & Hochberg, 2000), and results on adolescent social functioning, an onset of MDD before age 18, chronic MDD, and presence of at least one prior suicide attempt incurred a less than 5% FDR. In fact, three of the comparisons (MDD before age 18, chronic MDD, and adolescent social functioning) incurred less than a 1% FDR. Therefore, it is unlikely that these results were false positive results, although these variables should be examined further in future research.
Finally, results obtained from the present study may be a reflection of a reporting bias, given that the ages of onset were retrospectively reported (e.g., they may also have been experiencing depression at the time of SAD onset but do not remember). However, results on reported ages of onset were consistent with other studies, including prospective studies (Beesdo et al., 2007). Nonetheless, further research, particularly prospective studies similar to Beesdo et al., are needed to determine the degree to which SAD onset actually precedes the onset of MDD and therefore serves as a significant risk factor to later developing MDD.

In conclusion, results from the present study indicate that patients with MDD and SAD tend to experience a more severe form of depressive illness when their SAD onset in childhood or adolescence, compared to adulthood. In addition, they experienced a more rapid onset to MDD when their SAD onset in childhood compared to adulthood. Although causal implications cannot be derived from these results given their retrospective nature, they do suggest the need for further research to examine the relationship between an early onset of SAD and subsequent onset of MDD. Results from the present study highlight the importance of the early detection and treatment of SAD, as many patients with SAD tend to experience further psychiatric problems into adulthood.

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References


