The Natural Course of Social Anxiety Disorder among Adolescents and Young Adults

Katja Beesdo-Baum, PhD[1*]

Susanne Knappe, PhD[1]

Lydia Fehm, PhD[2]

Michael Höfler, PhD[1]

Roselind Lieb, PhD[3,4]

Stefan G. Hofmann, PhD[5]

Hans-Ulrich Wittchen, PhD[1,4]

[1] Institute of Clinical Psychology and Psychotherapy, Technische Universitaet Dresden, Germany

[2] Department of Psychology, Humboldt University Berlin, Germany

[3] Clinical Psychology and Epidemiology, University of Basel, Switzerland

[4] Max Planck Institute of Psychiatry, Munich, Germany

[5] Department of Psychology, Boston University, Boston, MA, USA

Running Title: Natural Course of Social Anxiety Disorder

Word count: 5,320 (main body text)

Abstract word count: 200

Address of corresponding author: *

Katja Beesdo-Baum, PhD

Institute of Clinical Psychology and Psychotherapy, Technische Universitaet Dresden

Chemnitzer Str. 46

01187 Dresden, Germany

Phone: ++49-351-463-36989

Fax: ++49-351-463-36984

E-mail: beesdo@psychologie.tu-dresden.de

ABSTRACT

Objective. To examine the natural course of social anxiety disorder (SAD) in the community and to explore predictors for adverse long-term outcomes.

Method. A community sample of N=3,021 subjects aged 14-24 was followed-up over 10 years using the DSM-IV/M-CIDI. Persistence of SAD is based on a composite score reflecting the proportion of years affected since onset. Diagnostic stability is the proportion of SAD subjects still affected at follow-up.

Results. SAD reveals considerable persistence with more than half of the years observed since onset spent with symptoms. 56.7% of SAD cases revealed stability with at least symptomatic expressions at follow-up; 15.5% met SAD threshold criteria again. 15.1% were completely remitted (no SAD symptoms and no other mental disorders during follow-up). Several clinical features (early onset, generalized subtype, more anxiety cognitions, severe avoidance and impairment, co-occurring panic) and vulnerability characteristics (parental SAD and depression, behavioural inhibition, harm avoidance) predicted higher SAD persistence and -less impressively- diagnostic stability.

Conclusion. A persistent course with a considerable degree of fluctuations in symptom severity is characteristic for SAD. Both, consistently meeting full threshold diagnostic criteria and complete remissions are rare. Vulnerability and clinical severity indicators predict poor prognosis and might be helpful markers for intervention needs.

Keywords: social phobia, stability, remission, persistence, epidemiology

Significant outcomes:

(1) Diagnostic stability of SAD above the DSM-IV threshold level over long periods of time but also complete remissions (neither SAD symptoms nor other psychopathology) are rare. SAD has considerable persistence considering subthreshold and symptomatic expressions.

(2) Isolated fears of exams/tests in adolescence have the lowest persistence, whereas generalized and early onset social fears show the highest persistence and stability.

(3) Symptom complexity and severity as measured with SAD diagnostic criteria as well as co-occurring conditions are important clinical characteristics that predict a persistent and stable course of SAD, suggesting that this diagnostic information is useful and practical to inform about prognosis and need for intervention.

Limitations:

(1) Stability and persistence estimates were based on up to four symptom and diagnostic assessments conducted with standardized diagnostic interview (DIA-X/M-CIDI) across a time period of up to ten years. Assessments did not include specific questions on course patterns of SAD, which impedes the differentiation of recurrence vs. chronicity.

(2) Stability and persistence estimates are conservative given that maximum age of respondents was 34 years at last follow-up and given that some SAD cases had short follow-up periods.

(3) Despite the prospective-longitudinal design of the study, data are based on retrospective recall and thus are subject to bias which may particularly have influenced the persistence measure.

INTRODUCTION

Social anxiety disorder (SAD) is a prevalent mental disorder with an estimated mean lifetime prevalence of 6.7% (range 3.9-13.7%) in European countries (1), and rates up to 12.1% in US community studies (2, 3). SAD typically has its onset in adolescence (2-9) and is associated with high comorbidity (1, 3-5, 9-12), substantial impairment and disability in psychosocial functioning (4, 11-15).

Relatively little is known about the *natural course* of the disorder which can be described in terms of (1) *persistence* and (2) *diagnostic stability* versus *spontaneous remission*. Retrospective cross-sectional and clinical data predominantly from adults indicate that SAD is highly persistent, with duration estimates of 10 years and longer (4, 5, 8, 12, 16-22). These findings suggest a stable and unremitting course of the condition. In contrast, longitudinal community studies that allow prospective examinations of diagnostic stability and spontaneous remission suggest a waxing and waning course of SAD with frequent oscillations around the DSM-IV diagnostic threshold (7, 8, 23). These studies were largely based on non-adult samples. Spontaneous remissions from SAD have also been reported (24-29). Empirical evidence for complete remission, i.e. the absence of any psychopathology, however, is rare (24, 25). No prior study has used a longitudinal approach to study the natural course of SAD both in terms of persistence and in terms of diagnostic stability and spontaneous remission. Conducting such a study during the high risk period for onset and potential subsequent chronicity of SAD would be particularly important to advancing our understanding of SAD prognosis and treatment interventions. Given prior findings on disability, psychosocial functioning, comorbidity, economic costs, partial recoveries or symptom fluctuation, consideration of subthreshold SAD appears particularly critical (5, 30, 31) as is the additional differentiation of even milder, symptomatic social fear expressions (5, 7, 29, 32).

Even less is known about the *predictors for persistence and diagnostic stability* of SAD and to what degree they differ from established vulnerability and risk factors for initial SAD onset. Several studies have documented associations between parental

psychopathology (33-35), temperamental and personality characteristics (36-39), as well as psychopathological risk factors such as panic attacks (40) and SAD, suggesting relevance for *disorder onset*. Few studies have examined whether such variables also predict *high persistence* and *stable course* of SAD. One recent study found that lack of emotional warmth and dysfunctional family functioning characteristics were associated with higher SAD persistence, particularly in interaction with parental psychopathology (41). With regard to clinical characteristics as predictors of course, many studies merely refer to anxiety disorders in general, with early age of onset (42), degree of impairment (43), or comorbidities (17-19) being associated with an unfavourable outcome. The few available SAD studies yield heterogeneous findings: whereas one 1.5 year follow-up study in young women did not find any disorder characteristics such as severity or duration of symptoms as predictors (29), other studies with partially longer follow-up periods and including both genders reported that baseline severity (44), symptom duration and comorbid panic disorder (22) were related to poor outcomes of SAD.

Overall, conclusions on the natural course of SAD and its predictors are limited to heterogeneous findings. Studies often differ in terms of their design (cross-sectional vs. longitudinal), time period, and selected SAD-related characteristics and risk factors. Using data from a representative community sample of adolescents and young adults followed prospectively over 10 years, which covers the high risk period for initial SAD onset and potential sequelae, we previously described the incidence patterns of SAD and subsequent onset of depression (9, 45), risk factors for the onset of SAD (34, 46), predictors for the onset of subsequent depression (9, 47), as well as select familial risk factors for persistence of SAD (41). The current study aims to use these data to describe in greater detail the natural course of SAD.

Aims of the study

The aims of the current study are:

(1) to provide a comprehensive description of the natural course of DSM-IV SAD both in terms of persistence and in terms of diagnostic stability and remission, following a longitudinal approach that takes into account different diagnostic threshold levels and comorbid conditions, and

(2) to examine a range of distal and proximal predictors for an unfavourable course (high persistence and stability versus remission) of SAD symptoms after initial threshold SAD onset.

MATERIAL AND METHODS

The prospective longitudinal Early Developmental Stages of Psychopathology (EDSP) study assessed mental disorders and associated risk factors in a representative sample of N=3,021 adolescents and young adults aged 14-24 years at baseline (T0). The study also includes follow-up surveys (T1/T2/T3), a family history component (T0/T2/T3) and direct assessments of parents (T1/T3). Methods, design and information on representativeness and response rates have been previously reported (48, 49).

Briefly, the baseline sample was drawn in 1994 from government registries (greater Munich area, Germany); N=3,021 interviews were conducted (response rate (RR)=71%). The first follow-up (T1; range 1.2-2.1 years since baseline) was conducted only for the younger study cohort (age 14-17 at T0; N=1,228; RR=88%), whereas the second (T2, range 2.8-4.1 years since baseline; N=2,548, RR=84%) and third follow-up (T3, range 7.3-10.6 years since baseline; N=2,210; RR=73%) were conducted among all subjects.

All participants provided written informed consent, except for those younger than 18 years, in which case the parents provided written informed consent. The EDSP project and its family genetic supplement have been approved by the Ethics Committee of the Medical Faculty of the Technische Universitaet Dresden (No: EK-13811).

Assessments

Symptoms, syndromes and diagnoses of DSM-IV mental disorders were assessed face-to-face by clinically trained interviewers with the computer-assisted version of the Munich-Composite International Diagnostic Interview (DIA-X/M-CIDI) (50). The DIA-X/M-CIDI is supplemented by a separate respondent's booklet that includes disorder-specific questionnaires as well as symptom lists and cognitive aids to assist the respondent in answering complicated symptom questions and in dating symptom onset and recency (51). Reliability and validity was moderate to good for all the disorders covered by the DIA-X/M-CIDI (52-54). Kappa for diagnostic test-retest reliability was 0.72 for DSM-IV SAD and 0.75 for SAD stem items (52). Validity of the *DSM-IV* SAD diagnoses compared with independent clinical consensus diagnoses by treating physicians was estimated with a kappa of 0.80 (53). The intraclass coefficient for SAD age of onset was 0.70 (52). At baseline, the DIA-X/M-CIDI was used to assess lifetime diagnoses; follow-up assessments covered the time interval since the last interview. The SAD section began with a series of stem questions ("Have you ever had an unusually strong fear or avoidance of doing things in front of others or of being the centre of attention? For example, have you ever had an unusually strong fear of ...") to assess the presence of strong fears regarding the following 6 social and performance situations (seven situations from T1 on): eating or drinking while others are watching, writing while others are watching, going to a meeting or party, taking an exam or interview at work or school although well prepared, speaking in front of others, speaking with others, and from T1 on 'other' social fears (DSM-IV criterion A-1). To improve recall and memory, questions were visually accompanied with a list of these situations (51). Respondents were also asked to give a concrete example for each item endorsed to allow for clarification. After at least one social fear situation was elicited, a subsequent series of nine questions asked about anxiety cognitions that occur when confronted with such social fear situations (e.g. something embarrassing or shameful could happen, being regarded as dumb or weak, being regarded as crazy, to experience an anxiety (panic) attack, etc.), of which at least 1 must be endorsed (criterion A-2). Criterion B (exposure to social or performance situations almost invariably provokes an immediate anxiety response) was assessed by a list of anxiety symptoms (e.g.,

sweating, heart racing, etc.), of which at least 2 must have occurred when thinking about or when being exposed to social fear situations. Respondents further indicated whether they considered either the anxiety or the avoidance to be excessive or unreasonable (criterion C), and whether they frequently avoided the situations or, if not, endured the situations with distress (criterion D). The clinical significance (criterion E) was assessed by determining whether the respondent reported that the social fears or avoidance interfered a lot with normal routines, whether they sought professional help for the fears, or whether they repeatedly used medication due to these fears. Respondents were classified as *threshold DSM-IV SAD* cases when they met criteria A to E. In contrast to an earlier study (9) but in line with more recent contributions (41, 55, 56), criterion E was required when respondents were 18 years or older (49). Taking all available information from all assessment waves into account, N=209/3,021 (6.6%) respondents met criteria for DSM-IV SAD. For N=156/209 (75.3%) SAD respondents at least one subsequent follow-up assessment was available. Among those, mean follow-up duration was 6.9 years (range 1-10 years). The remaining N=53/209 cases either reported threshold SAD at the last assessment wave for the first time (N=29, 13.0%) or did not participate at any follow-up assessments (N=24, 11.7%). There was no selective drop out (attrition) from baseline to 10-year follow-up for SAD (OR=1.1, 95%CI: 0.8-1.6).

Besides the DSM-IV SAD diagnosis, we also considered following groups with social anxiety below the full diagnostic threshold for course/persistence analyses: Respondents were classified as *subthreshold SAD* cases when they met criterion A and three of the criteria B, C, D or E. Respondents who were not classified with (sub-)threshold SAD but affirmed at least one of the DIA-X/M-CIDI stem questions referring to 'unusually strong' fears in or avoidance of social and performance situations were labelled as *symptomatic SAD*. The inclusion of these broader categories accounts for previous indications of the waxing and waning nature of psychopathology among youth and young adults (7, 26) and the possibility of partial remissions (29). The differentiation of social fear expressions below the diagnostic threshold (i.e. symptomatic and subthreshold SAD) is further justified by prior findings on

increasing levels of disability and comorbidity and decreasing levels of psychosocial functioning within the social anxiety spectrum (5, 7).

The combination of the symptomatic, subthreshold, and threshold cases will be referred to as *'at least symptomatic SAD'*.

Persistence of SAD. Persistence of SAD was defined as the proportion of years an individual was affected by SAD symptoms given the total number of years observed after initial threshold SAD onset. Using retrospective age of onset and age of recency information on SAD symptoms, a composite score was created: (1) Consistent with prior work (41), age of onset and age of recency information were aggregated across assessments, using the lowest reported age of onset and highest reported age of recency by convention. (2) In order to reduce recall bias leading to overestimation of SAD persistence, a more conservative age of onset convention was used: When age of onset was 10 years or lower, age of onset information was replaced by the age of 10. (3) Starting from the first report of initial threshold SAD, persistence scores were calculated irrespective of prior symptomatic or subthreshold SAD conditions. Persistence scores reflect the proportion of years an individual was affected by either threshold SAD, at least subthreshold SAD, or at least symptomatic SAD after initial onset of threshold SAD. The scores in the total sample range from 0 (no SAD) to 1 (SAD symptoms in all years observed since initial onset of threshold SAD). For example, a respondent aged 15 years at baseline (T0) participated at all subsequent assessment waves (25 years of age at T3). First onset of threshold SAD was reported at age 15, resulting in overall 10 years of being observed. Threshold SAD was present until age 17. From age 17 to 21 no symptoms occurred, but from age 22 to 25 criteria for subthreshold SAD were met. Regarding only threshold SAD, the persistence score reflects 3 years (ages 15–17) spent with threshold SAD, and persistence would be 3/10, indicating that threshold SAD symptoms were present during 33% of the time observed. This persistence rate increases to 70%, when the four years (ages 22–25) of subthreshold SAD were additionally considered [(3 + 4)/10]. We also calculated a total persistence index that considers weights for different diagnostic status (symptomatic 1/3, subthreshold 2/3, threshold 1). To examine validity of the

persistence scores, we used more direct information on persistence as provided by the respondents in the M-CIDI SAD-section. Respondents were asked whether the anxiety and/or the avoidance of social situations persisted for months or even years and if not, whether this was the case because social situations were completely avoided. The sum score of positive responses from all assessment waves (observed range 0-4) was significantly correlated with the various persistence-scores (all p-values <.01), ranging for SAD-subjects with follow-up assessment from $r = .30$ (threshold level) to $r = .51$ (at least symptomatic level).

Diagnostic stability vs. remission of SAD. Diagnostic stability and remission of SAD were strictly prospectively examined by using diagnostic information from follow-up assessments after the person met threshold SAD criteria for the first time (N=156 with "initial threshold SAD"); retrospective age of onset and age of recency information was not taken into account here. After initial threshold SAD (at T0, T1 or T2), the maximum follow-up diagnostic status (at T1-T3, T2-T3 or T3) was described on four levels: no SAD symptoms, symptomatic, subthreshold or threshold SAD. According to the diagnostic status at follow-up, subjects were classified as *stable* if criteria for at least symptomatic SAD were met, or as *remitted* if no SAD criteria were met.

It should be noted that both approaches to describe the course of SAD do not allow a differentiation between recurrence and chronicity.

Predictors for SAD persistence and stability. Based on the previous literature, several clinical characteristics of initial SAD and established vulnerability and risk factors were examined as putative predictors for (a) persistence and (b) stability vs. remission of SAD:

Parental psychopathology (lifetime diagnoses in either mother or father: SAD; any other anxiety disorder including specific phobia, generalized anxiety disorder, panic disorder, agoraphobia; any depressive disorder including major depressive disorder or dysthymia, any substance use disorder including abuse or dependence of alcohol or illicit drugs) was derived

by aggregation of diagnostic information from direct interviews in parents (at T1/T3) and indirect family history information using the respondents as informants (at T0/T2/T3). Following examination of agreement patterns between family history report and available direct interviews, a priority hierarchy was determined (57): If direct information from T3 and/or T1 was available, it was used. If no direct information was available, T3 family history reports were used with the highest priority, followed by T2 and T0 family history reports.

Behavioural inhibition was measured by the Retrospective Self-Report of Inhibition scale (58, 59), *personality measures* were derived from the Tripartite Personality Questionnaire (60).

SAD characteristics were derived from the DIA-X/M-CIDI SAD module. *Age-of-onset* was available for N=208 respondents and is based on the lowest age of onset reported at any of the assessment waves. *Types of feared social situations* at initial threshold SAD that were avoided or endured with anxiety because of doing things in front of others or because of being the centre of attention were: 1) eat/drink in public, 2) public writing, 3) go to a meeting/party, 4) tests/exams, 5) public speaking, and 6) talk to others; at T1/T2/T3 "other" social fears were also assessed. Because explorations of the factorial structure of social fears in our data did not suggest separate factors for interactional or performance fears but indicated a special role of test fears (56), we refrained from grouping social fears based on content type and separately examined the predictive role of test fears and other social fears both overall and in isolation (i.e. without co-occurring other social fears). However, we examined the role of the number of endorsed social fear situations and the *generalized subtype* as stipulated in DSM-IV defined here by the presence of 3+ feared social situations. *Catastrophic anxiety cognitions* refer to nine feared events (e.g. something embarrassing or shameful could happen, being regarded as dumb or weak) while being in situations or assuming situations that involved being the centre of attention. *Degree of avoidance* (1-never to 4-always) refers to the frequency at which social situations were avoided due to anxiety. As outlined above, it should be noted that not all SAD cases must reveal avoidance as they may also fulfill criterion C because they endured such situations with distress. *Degree of*

impairment (1-not at all to 4-very much) reflects how much anxiety or avoidance of social situations interfered with daily life. Again, as outlined above, not all SAD cases must reveal significant impairment because clinical significance (criterion E) may also be established by professional help seeking or medication use. *Comorbid conditions* were assessed using the respective DIA-X/M-CIDI section and included other anxiety (specific phobia, panic disorder, agoraphobia, generalized anxiety disorder), depressive (major depression, dysthymia), substance use (abuse or dependence of alcohol or illicit drugs), somatoform (hypochondrias, pain disorder, undifferentiated somatization disorder) and eating disorders (anorexia nervosa, bulimia nervosa, anorexia nervosa not otherwise specified, bulimia nervosa not otherwise specified) as well as panic attacks. The clinical characteristics including comorbidity were derived at the assessment wave when threshold SAD was reported for the first time and used as predictor variables for SAD course. Table 1 provides an overview of the distribution of the vulnerability and the initial clinical characteristics (when SAD was first reported) in the SAD sample. There were no significant differences between SAD respondents with and without follow-up assessments in these variables except that SAD cases with follow-up assessments reported lower ages of SAD onset and higher levels of behavioural inhibition (p-values<.05).

-Table 1-

Statistical analysis

Results (%, ratios, coefficients) are weighted by age, gender, and geographic location at baseline to match the distribution of the original sampling frame (48); frequencies (N) are unweighted. The Stata Software package (61) was used to compute robust variances, confidence intervals, and p-values (by applying the Huber-White sandwich matrix) required when analyses were based on weighted data (62).

Diagnostic information from the assessment waves were aggregated for cumulative lifetime incidences (T0/T1/T2/T3) or maximum follow-up status after initial threshold SAD

(T1/T2/T3, T2/T3, T3). As we were interested in an accurate picture of the natural course and persistence of SAD, and in order to prevent overestimation, we restricted most analyses on the course and persistence of SAD to subjects with at least one follow-up assessment after initial threshold SAD (N=156/209).

Predictors for course of SAD were examined using univariate regression analyses. For associations with bivariate outcome variables (stability vs. remission), logistic regression analysis were used (odds ratios; OR). For dimensional outcome variables (persistence), linear regression analyses were conducted. Multiple regression analyses included significant predictors from univariate regressions and were used to identify the most powerful predictors.

RESULTS

Persistence of SAD

The mean persistence for threshold SAD in the total SAD sample (N=208) was M=0.62 indicating that on average 62% of the observed time after initial SAD onset was spent with symptoms. This rate further increased when subthreshold (M=0.67) and symptomatic SAD (M=0.70) after initial onset of threshold SAD were additionally taken into account. Overall across the three threshold levels, the mean weighted persistence index was 0.66 in the total sample and was not different in males and females (p>.8). Results also indicated that the persistence score decreased gradually with longer follow-up duration (p<.001).

-Table 2-

Univariate regression analyses using each of the characteristics from Table 1 as putative predictors for higher SAD persistence were performed separately for all SAD cases (N=208) and for those with follow-up assessments (N=156). Because only few differences were found between the total and the follow-up completer group (Table 3), and because the

latter may be assumed to more accurately reflect SAD persistence, we discuss the follow-up completer group in further detail. A higher persistence of SAD was significantly predicted by early age of onset of SAD, the generalized subtype, a greater number of catastrophic anxiety cognitions, more severe avoidance due to social fears, and more severe levels of impairment. Co-occurring panic attacks also predicted a greater persistence of SAD. Significant vulnerability factors were: parental SAD or parental depressive disorder, high levels of self-reported behavioural inhibition in childhood, harm avoidance and low novelty seeking. Multiple regression analyses, taking into account all significant variables, revealed the generalized subtype and high levels of harm avoidance as the most important predictors in the total sample; in the follow-up completer sample, a lower age of onset and more severe impairment were additionally found to contribute significantly to the model (p<.05).

It is noteworthy that in the univariate analyses most individual social fear situations at initial threshold SAD (talking to others, going to meeting/party, public speaking, 'other' social fear) predicted persistence but only when they did not occur in isolation. One notable exception is social fears of exams or tests that were predictive of low SAD persistence, particularly if they occurred in isolation. Overall, SAD cases with fear of exams/tests revealed the lowest average number of social fears (2.6) among all types of social fears (mean feared situations: 3.0 for public speaking to 4.2 for writing). Individuals with exam/test fears also had the lowest probability of belonging to the generalized subtype (44.7%); risk was highest among individuals with fears of going to meetings/parties (82.7%). Overall, as shown in Table 1, fears of exams/tests occurred most frequently "in isolation" among all social fears (23.0%).

-Table 3-

Diagnostic stability and remission of SAD

Strictly prospectively (relying on diagnostic information without consideration of age-of-onset or age-of-recency information), the diagnostic stability rates of threshold SAD,

defined as meeting the full DSM-IV criteria again at a subsequent assessment, ranged

between 7.1% and 15.1%, depending on the considered assessment times and follow-up

periods (Table 4). Overall, among those who had threshold DSM-IV SAD for the first time up

to T2, 15.5% revealed full criteria again during at least one subsequent follow-up wave after

initial report of threshold SAD (Figure 1). Although the majority of SAD cases did not meet

full DSM-IV SAD criteria again at subsequent waves, a substantial proportion still had

subthreshold SAD (12.7-21.2%; overall: 19.7%) or at least some significant SAD symptoms

(9.1%-25.3%; overall: 21.5%).

Of note, although the stability rates for SAD appear numerically rather moderate, SAD

at each time point was, compared to those without SAD, associated with a considerably

increased risk to also have the disorder (OR: 7.1-22.1) or signs and symptoms of the

disorder (OR=2.9-11.0) at later points in time (Table 4). If no SAD was reported during follow-

up, the presence of other disorders was probable (24.6%-35.8%; overall: 28.2%). Only 14.2-

31.5% (overall: 15.1%) of DSM-IV SAD cases were completely remitted at follow-up, i.e. they

revealed neither SAD symptoms nor other disorders.

We also investigated whether initial symptomatic or subthreshold SAD conditions are

associated with follow-up SAD caseness including the development of subsequently more

intense SAD expressions (i.e. subthreshold or full threshold SAD). Multinomial logistic

regression analyses revealed significant findings for all time point and threshold level

combinations (Table available upon request), indicating an overall increased probability to

remain or progress within the SAD spectrum over time.

--Table 4 and Figure 1-

To examine predictors for diagnostic stability of SAD, threshold, subthreshold and

symptomatic SAD outcomes after initial SAD diagnosis (N=156) were combined in one group

'at least symptomatic SAD' (N=93) and compared to those without follow-up SAD symptoms

('SAD remitters', N=63). The SAD-specific stability/remission rate did not differ by follow-up

duration or by gender (Table 5). Age of onset of SAD, however, was predictive in that lower ages of onset were associated with stability. Among the other clinical, comorbidity and vulnerability variables listed in Table 1, the generalized subtype, co-occurring panic attacks, childhood behavioural inhibition, and high harm avoidance were predictors of stability; a trend finding emerged for a higher number of catastrophic anxiety cognitions (OR=1.3, 95%CI: 1.0-1.7, p=.052) mainly due to "something embarrassing or shameful could happen" and "being regarded as dumb or weak". The generalized subtype and high harm avoidance were revealed as the most powerful predictors for stability in a multiple regression analysis (p<.05).

-Table 5-

DISCUSSION

Using data from a large prospective-longitudinal community study of adolescents and young adults followed-up across the high risk period for SAD onset and potential subsequent chronicity, we complemented prior research on the incidence of SAD (9, 45) and its risk factors (33, 34, 40) by examining the natural course of SAD and potential clinically meaningful predictors. In contrast to previous investigations (e.g. 7, 18, 22-25, 29), we examined the course of SAD both in terms of persistence using retrospective age of onset and age of recency information and in terms of diagnostic stability (versus remission). This longitudinal approach takes into account the full range of SAD symptoms, including conditions above and below the diagnostic threshold. Before discussing the findings in detail, some limitations of our study should be noted. First, the EDSP study was not specifically designed and powered to study the course of SAD. The symptom and diagnostic assessment was exclusively based on a standardized diagnostic interview (DIA-X/M-CIDI) that did not include specific questions on course patterns, which impedes differentiation of recurrence vs. chronicity. The between-assessment intervals extended to several years and the number of cases with SAD and at least one follow-up assessment was limited. Second,

not all study participants had reached the maximum age of 34 years at the time of last follow-up and the follow-up time period varied among SAD cases. The overall stability and persistence rates should therefore be considered a conservative estimate of the true rates. Furthermore, no conclusions can be drawn regarding the stability and persistence of SAD in higher ages beyond young adulthood. Third, despite the prospective-longitudinal design of the EDSP study, data are based on retrospective recall and thus is subject to bias which may have particularly influenced the persistence measure.

SAD is one of the most prevalent mental disorders in the community. Our observed cumulative incidence rate of 6.6% for threshold SAD is in line with lifetime findings from other research (1-3). Also consistent with other studies (2-9), the majority of SAD cases reported a symptom onset in childhood or early adolescence, which indicated this developmental phase as the core period for targeting potential prevention and early intervention programs. There is particular need for such interventions, as our study impressively shows a high persistence and stability of symptoms particularly in early onset SAD cases. Unfortunately, treatment rates at this young age are particularly low (63).

Little systematic, methodologically sound research has been conducted to characterize the natural course of SAD in greater detail, particularly in non-patient, representative samples from the community. Cross-sectional epidemiological and clinical studies suggest a chronic, stable and non-remitting course of the disorder (4, 5, 8, 12, 16-22). Yet, their results rely merely on retrospective reports that may be subject to significant recall-bias, particularly when considering long time periods. More specifically, the frequently reported number of years between symptom onset and recency, in terms of a persistence measure, likely overestimates chronicity because symptom-free intervals are not taken into account.

The findings from our multi-wave study do not fully confirm previous retrospective findings from cross-sectional studies that highlight a chronic, stable and non-remitting course of the disorder. In line with the chronicity assumption are the findings of our persistence

measure revealing that individuals with DSM-IV SAD suffer from SAD symptoms at least fifty percent of the years observed after first onset; rates were even higher when subsequent subthreshold and symptomatic SAD expressions were also considered. Persistence rates tended to be lower when the follow-up observation period after initial SAD diagnosis was longer, suggesting either stable remissions occurring during follow-up or the fact that respondents with follow-up assessments had a good chance for symptom-free periods. Generally, these findings indicate a potential methodological artefact arising from cross-sectional research that suggests a very high stability and chronicity of SAD merely based on retrospectively recalled age of onset and age of recency information or by examining the ratio of lifetime to 12-month prevalence.

This interpretation is supported by our prospective diagnostic stability findings that somewhat contradict the assumption of high disorder-specific chronicity. In line with other prospective studies in children, adolescents and young adults (7, 8, 23-27, 29), we find that remissions from or improvements in SAD indeed occur. Our study revealed that only 15.5% of DSM-IV SAD cases met the full criteria again later in the study. However, this rate increased to 56.7% when also considering symptomatic and subthreshold SAD, which have been shown to be associated with considerable disability and comorbidity and impairment in psychosocial functioning (5, 7, 30, 32).

This indicates that a substantial proportion of children, adolescents and young adults continue to have significant SAD symptoms years after SAD initially emerged. Thus, supported by our association analyses showing significant associations between prior and subsequent expressions within the SAD spectrum, our strictly prospective findings are consistent with the assumption of a considerable degree of homotypic continuity, yet with an indication of waxing and waning SAD symptoms and oscillations around the diagnostic threshold as previously described for SAD (7) and other anxiety disorders as well (26). Moreover, even when remitting from SAD, other disorders frequently persist or develop in the years after, making it extremely unlikely that SAD cases turn out to be completely free of psychopathology over the long run (15.1%). Overall, these findings are in line with other

prospective research indicating both disorder-specific (7, 64-66) and more heterotypic longitudinal associations (7, 47, 66).

In light of the ongoing revision process for DSM-5, this necessarily prompts the question of which types of SAD are associated with particular stable-persisting as opposed to instable and remitting courses. Our findings suggest that the generalized subtype (defined here as fearing three or more social situations) is a powerful predictor for a stable-persisting course of SAD. Thus, this subtype differentiation has predictive power in the sense of a more general severity indicator. Given that SAD symptoms appear to fall along a continuum of severity based on the number of social fears (67), a definite criterion of the number of feared situations required to indicate symptom severity however is unlikely (68). We also do not have convincing evidence for an 'interaction' versus 'performance' fear differentiation, as the factor structure of social fears appears unidimensional in our data. The noteworthy exception is social fear of exams/tests. This particular 'performance' fear is, particularly if occurring in isolation which is frequently the case, characterised by low persistence with symptom alleviations likely to occur when finishing school/university.

Besides the breadth of the feared social situations, other measures of SAD symptom complexity and severity, such as the number of catastrophic anxiety cognitions, degree of avoidance and impairment, and co-occurring psychopathology, most consistently panic attacks, were revealed in our study as important clinical characteristics that predict a stable-persisting course of SAD. Our findings are in line with other research (22, 42-44) suggesting the importance of clinical features as course-predictors for SAD and suggest that such diagnostic information is useful and practical to inform prognosis and need for intervention.

In addition to clinical diagnostic measures, parental psychopathology (SAD and depressive disorders) and, more consistently, temperamental measures (behavioural inhibition, harm avoidance) were found to provide significant predictive power. Thus, such factors are not only associated with the risk for onset of SAD (33-37, 57, 59), but also predict adverse outcomes. Importantly, there are indications for complex interactions with other familial factors such as rearing styles (e.g. lack of emotional warmth) and dysfunctional

family functioning contributing to higher persistence of SAD (41). More research is necessary not only to improve understanding of the vulnerability and risk factors for onset of SAD, but also to delineate their role in course and outcome. Overall, our study suggests that familial and temperamental measures along with clinical diagnostic measures inform prognosis and thus appear useful for targeting intervention to prevent adverse long-term outcomes. Future research may more strongly focus on comparative analyses using different methodological approaches on course as there may be differential findings.

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TABLES AND FIGURES

Table 1: Characteristics of SAD cases

		SAD					
Characteristics		total (N = 209)		with follow-up (N = 156)		without follow-up (N = 53)	
Gender							
Males	N, %w col	70	31.6	54	30.9	16	33.8
Females	N, %w col	139	68.4	102	69.1	37	66.2
Clinical characteristics of initial SAD [b]							
Age at SAD onset (in years) [a]	M, SD	13.1	5.4	12.0	4.8	16.2	6.0
<=11	N, %w col	85	40.1	73	45.5	12	23.2
12-14	N, %w col	65	27.4	51	29.3	14	21.5
>=15	N, %w col	58	32.5	32	24.2	26	55.3
Feared social situation							
- talking to others	N, %w col	69	36.6	56	38.2	13	31.6
- going to a meeting or party	N, %w col	57	32.8	40	29.0	17	40.2
- eating or drinking in public	N, %w col	45	23.4	36	23.4	9	23.3
- exams or tests	N, %w col	131	62.7	100	64.3	31	61.7
- public speaking	N, %w col	118	57.9	89	57.6	29	58.8
- writing in public	N, %w col	16	9.2	11	8.2	5	12.1
- other [c]	N, %w col	24	12.3	11	7.3	13	27.7
Number of feared social situations	M, SD	2.3	1.5	2.3	1.4	2.6	1.7
1	N, %w col	93	41.2	67	41.1	26	41.5
2	N, %w col	46	22.3	37	24.7	9	15.1
3+ (~ generalized subtype)	N, %w col	70	36.5	52	34.3	18	43.4
Isolated social fears							
- talking to others	N, %w col	9	4.3	6	3.1	3	4.1
- going to a meeting or party	N, %w col	10	4.4	5	3.4	5	7.4
- eating or drinking in public	N, %w col	7	3.6	5	3.8	2	3.1
- exams or tests	N, %w col	52	23.0	39	23.7	13	20.9
- public speaking	N, %w col	29	12.2	20	11.9	9	13.1
- writing in public	N, %w col	5	2.7	4	3.2	1	1.0
- other [c]	N, %w col	3	1.3	2	1.4	1	1.0
Anxiety cognitions							
- something embarrassing or shameful could happen	N, %w col	103	49.8	90	57.2	13	27.1
- being regarded as dumb or weak	N, %w col	102	52.1	89	60.1	12	27.7
- being regarded as crazy	N, %w col	17	9.3	13	8.9	3	10.4
- experience an anxiety (panic) attack	N, %w col	43	22.6	35	24.5	8	17.0
- to be confused	N, %w col	129	63.9	112	36.4	17	72.8
- to be ashamed	N, %w col	79	40.6	66	44.8	13	27.8
- to throw up	N, %w col	12	8.7	11	10.8	1	2.2
- to loose control over intestinal organs	N, %w col	5	2.8	5	3.7	0	0.0
- to turn red	N, %w col	97	44.7	84	51.2	13	25.0
Number of anxiety cognitions (1-9)	M, SD	3.4	1.6	3.3	1.5	3.6	1.8
Severity measures							
Level of avoidance (1-4)	M, SD	2.2	1.1	2.2	1.0	2.2	1.2
Level of impairment (1-4)	M, SD	2.7	0.9	2.6	0.9	2.8	1.0
Comorbidity at time of initital SAD [g]							
panic attacks	N, %w col	25	15.0	17	13.5	8	19.3
anxiety disorder [h]	N, %w col	76	38.3	52	35.9	24	45.6
depressive disorder [i]	N, %w col	60	33.9	41	31.8	19	40.4
substance use disorder [k]	N, %w col	52	26.6	33	23.7	19	35.6
somatoform disorder [l]	N, %w col	30	15.1	19	13.6	11	19.7
eating disorder [m]	N, %w col	11	6.8	9	7.5	2	4.4
Parental psychopathology							
SAD	N, %w col	22	9.3	19	10.3	3	6.4
other anxiety disorder [h]	N, %w col	97	44.9	73	44.9	24	45.1
depressive disorder [i]	N, %w col	80	39.5	56	37.8	24	44.6
substance use disorder [k]	N, %w col	37	19.3	30	19.4	7	19.1
Temperament/personality							
behavioral inhibition (total sum) [n]	M, SD	2.4	0.5	2.5	0.5	2.2	0.4
behavioral inhibition (social fear) [n]	M, SD	2.7	0.7	2.8	0.7	2.5	0.6
behavioral inhibition (illness fear) [n]	M, SD	2.2	0.6	2.3	0.7	1.9	0.5
novelty seeking [o]	M, SD	16.4	5.2	16.0	5.2	17.9	4.8
harm avoidance [o]	M, SD	17.3	7.0	17.1	7.1	18.6	6.5
reward dependence [o]	M, SD	18.1	4.7	18.0	4.7	18.6	4.8

Note: SAD Social Anxiety Disorder, N unweighted number; %w percent weighted; M mean weighted; SD standard deviation

[a] no age of onset available for N=1 (male; no follow-up assessment)

[b] derived from DIA-X/M-CIDI SAD section when SAD was first reported

[c] only assessed at T1, T2, T3

[g] derived from respective DIA-X/M-CIDI section at time when SAD was first reported

[h] includes specific disorder, generalized anxiety disorder, panic disorder, agoraphobia

[i] includes MDE, dysthymia

[k] includes alcohol and ill. drug abuse or dependence

[l] includes hypochondrias, pain disorder, undifferentiated somatization disorder

[m] includes anorexia nervosa, bulimia nervosa, anorexia nervosa NOS, bulimia nervosa NOS

[n] from Retrospective Self-Report of Inhibition (RSRI)

[o] from Tridimensional Personality Questionaire (TPQ)

Table 2: Persistence of initial full SAD by age of onset and Follow-up duration

| | Persistence of initial threshold SAD | | | | | | | | |
| | total | | | initial threshold SAD with at least one follow-up | | | initial threshold SAD, no follow-up | | |
Persistence Scores [a]	N	M	SD	N	M	SD	N	M	SD
threshold level	208	**0.62**	0.28	156	**0.54**	0.25	52	**0.89**	0.22
at least subthreshold level	208	**0.67**	0.29	156	**0.59**	0.24	52	**0.89**	0.22
at least symptomatic level	208	**0.70**	0.28	156	**0.64**	0.27	52	**0.89**	0.22
Index [b]									
total	208	**0.66**	0.27	156	**0.59**	0.25	52	**0.89**	0.22
by follow-up duration									
none (1 wave only)	-			-			52	**0.89**	0.22
1 to 4 years	-			29	**0.71**	0.22	-		
5 to 8 years	-			83	**0.60**	0.22	-		
9 or 10 years	-			44	**0.50**	0.27	-		
by gender									
males	69	**0.68**	0.28	54	**0.59**	0.24	15	**0.93**	0.22
females	139	**0.66**	0.27	102	**0.59**	0.25	37	**0.88**	0.23
by clinical characteristics (initial SAD) [c]									
Age of onset of SAD									
<=11	85	**0.69**	0.22	73	**0.65**	0.19	12	**0.92**	0.26
12-14	65	**0.68**	0.26	51	**0.61**	0.23	14	**0.98**	0.07
>=15	58	**0.62**	0.34	32	**0.45**	0.30	26	**0.85**	0.24
Subtype									
non-generalized	138	**0.61**	0.28	104	**0.53**	0.25	34	**0.88**	0.22
generalized	70	**0.76**	0.23	52	**0.70**	0.20	18	**0.92**	0.23

Note: SAD Social Anxiety Disorder, N observed number (unweighted), M mean, *SD* standard deviation

[a] Persistence: proportion of years an individual was affected by SAD symptoms given the total number of years observed after initial threshold SAD onset. Persistence was calculated for N = 208 because for N = 1 no age of onset was available.

[b] Persistence Index: weighted for different diagnostic status (symptomatic 1/3, subthreshold 2/3, threshold 1).

[c] derived from DIA-X/M-CIDI SAD section when SAD was first reported

Table 3: Predictors of persistence of initial threshold SAD

	SAD					
	Persistence (Index) [a]					
	total (N = 208)			with follow-up assessment (N = 156)		
Putative predictors	Beta	T	P	Beta	T	P
Clinical characteristics (initial SAD) [b]						
Age at SAD onset (in years, dimensional)	-0.1	-1.4	.162	**-0.4**	**-5.1**	**<.001** §**
Feared social situations						
- talking to others	0.1	1.8	.077	**0.2**	**2.5**	**.013**
- going to a meeting or party	**0.3**	**4.5**	**<.001**	**0.3**	**3.6**	**<.001**
- exams or tests	**-0.2**	**-2.4**	**<.001**	-0.1	-1.5	.133
- public speaking	**0.3**	**4.1**	**<.001**	**0.4**	**4.4**	**<.001**
- other [c]	**0.3**	**4.3**	**<.001**	**0.2**	**2.7**	**.007**
- isolated fear of exams or tests	**-0.3**	**-5.2**	**<.001**	**-0.4**	**-5.1**	**<.001**
Generalized subtype (3+ vs. 1-2 situations)	**0.3**	**4.2**	**<.001** §**	**0.3**	**4.1**	**<.001** §**
Catastrophic anxiety cognitions						
- something embarrassing or shameful could happen	0.0	-0.1	.887	**0.2**	**2.1**	**.038**
- being regarded as dumb or weak	0.0	-0.2	.861	**0.2**	**2.3**	**.026**
- to turn red	0.0	-0.3	.765	**0.2**	**2.1**	**.037**
A higher number of anxiety cognitions (1-9, dimensional)	**0.2**	**2.9**	**.004** §	**0.2**	**2.6**	**.010** §
Severity measures						
More severe avoidance (1-4, dimensional)	**0.3**	**3.5**	**.001** §	**0.3**	**3.0**	**.003** §*
More severe impairment (1-4, dimensional)	**0.2**	**2.5**	**.013** §	**0.2**	**2.7**	**.008** §**
Comorbidity at time of initital SAD [g]						
panic attacks	**0.2**	**3.8**	**<.001** §	**0.2**	**2.8**	**.006** §
anxiety disorder [h]	**0.2**	**2.6**	**.010** §	0.1	1.7	.098
depressive disorder [i]	**0.2**	**2.0**	**.049** §	0.1	0.9	.369
Parental psychopathology						
SAD	0.0	0.7	.516	**0.2**	**2.5**	**.016** §
depressive disorder [i]	0.1	1.3	.186	**0.2**	**2.1**	**.037** §*
Temperament/personality						
behavioral inhibition (total sum) [n]	**0.2**	**2.4**	**.016**	**0.4**	**4.6**	**<.001**
behavioral inhibition (social fear) [n]	**0.2**	**2.5**	**.014** §	**0.3**	**4.1**	**<.001** §
behavioral inhibition (illness fear) [n]	0.1	1.2	.228	**0.2**	**2.7**	**.008** §
novelty seeking [o]	-0.1	-1.4	.172	**-0.2**	**-2.3**	**.025** §
harm avoidance [o]	**0.4**	**4.3**	**<.001** §**	**0.4**	**4.8**	**<.001** §**

Note: SAD Social Anxiety Disorder, Beta = standardized regression coefficient from univariate regression analyses, adjusted for age

[a] persistence index weighted for symptomatic (1/3), subthreshold (2/3) and threshold SAD (1) at follow-up assessment; calculated for N=208 because no age of onset information available for n = 1 (case without follow-up assessment after SAD diagnosis)

[b] derived from DIA-X/M-CIDI SAD section when SAD was first reported

[c] only assessed at T1, T2, T3

[g] derived from respective DIA-X/M-CIDI section at time when SAD was first reported

[h] includes specific disorder, generalized anxiety disorder, panic disorder, agoraphobia

[i] includes MDE, dysthymia

[n] from Retrospective Self-Report of Inhibition (RSRI)

[o] from Tridimensional Personality Questionaire (TPQ)

§ Variable entered in multiple regression analysis, ** p<.05, * p<.1

Table 4: Diagnostic stability of threshold SAD - longitudinal associations

| Assessment times: Threshold SAD ==> Outcome | Outcome at follow-up [a] | | | | | | | | | | | | | | | | | | |
| | **Threshold SAD** | | | | **Subthreshold SAD** | | | | **Symptomatic SAD** | | | | **No SAD symptoms but other disorder** | | | | **No SAD symptoms and no other disorder** |
	% (Ref [b])	OR [c]	95% CI	P	% (Ref [b])	OR [c]	95% CI	P	% (Ref [b])	OR [c]	95% CI	P	% (Ref [b])	OR [c]	95% CI	P	% (Ref [b])
T0 (N=114) ==> T1/2	7.1 (1.9)	7.1	2.9 - 17.3	**<.001**	19.2 (8.6)	4.3	2.3 - 8.1	**<.001**	25.3 (14.5)	3.6	2.0 - 6.6	**<.001**	24.6 (27.8)	1.8	1.0 - 3.4	.069	23.8 (47.1)
T0 (N=97) ==> T3	12.0 (1.4)	15.2	6.5 - 35.5	**<.001**	12.7 (5.2)	4.1	1.9 - 8.7	**<.001**	9.1 (5.8)	2.9	1.3 - 6.5	**.009**	34.8 (28.6)	2.5	1.4 - 4.5	**.003**	31.5 (58.9)
T0 (N=118) ==> T1/2/3	15.1 (2.8)	13.7	6.2 - 30.0	**<.001**	21.2 (10.6)	5.1	2.5 - 10.3	**<.001**	21.5 (15.9)	3.7	1.8 - 7.5	**<.001**	28.0 (32.4)	2.4	1.2 - 4.7	**.015**	14.2 (38.3)
T1/2 (N=40) ==> T3	12.3 (1.6)	22.1	6.5 - 75.6	**<.001**	12.8 (4.9)	7.5	2.3 - 25.0	**.001**	20.6 (5.8)	11.0	3.3 - 36.6	**<.001**	35.8 (28.3)	4.2	1.5 - 11.4	**.005**	18.5 (59.4)

Note: SAD Social Anxiety Disorder, T0 Baseline, T1 first follow-up, T2 second follow-up, T3 third follow-up

[a] Hirarchical and mutually exclusive groups: Threshold SAD, if not, subthreshold SAD, if not, symptomatic SAD, if not, other disorder (includes other anxiety, depressive, substance use, somatoform and eating

[b] Ref: Percentage in Reference group consisting of individuals without prior threshold SAD

[c] OR: Odds Ratio from multinomial logistic regression, Reference group: no prior threshold SAD

Table 5: Diagnostic stability of SAD by various characteristics

Initial SAD [b]		Follow-up [a]			Associations		
		no SAD		at least symptomatic SAD	OR	95%CI	P
Total	N, %w row	63	43.3	93	56.7	-		
Follow-up duration (in years)	M, SD	7.0	2.3	6.9	2.3	1.1	0.9 - 1.3	.478
1 to 4	N, %w row	15	49.0	14	51.0	Ref.		
5 to 8	N, %w row	29	34.7	54	65.3	0.7	0.2 - 2.4	.561
9 or 10	N, %w row	19	53.7	25	46.3	1.9	0.8 - 4.3	.135
Gender								
males	N, %w row	21	43.5	33	56.5	Ref.		
females	N, %w row	42	43.1	60	56.9	1.1	0.5 - 2.4	.768
Clinical characteristics (initial SAD) [c]								
Age of onset of SAD (in years)	M, SD	13.2	5.3	11.1	4.2	**0.9**	**0.8 - 1.0**	**.032** §
<=11	N, %w row	25	37.6	48	62.4	Ref.		
12-14	N, %w row	20	38.0	31	62.0	0.9	0.4 2.1	.793
>=15	N, %w row	18	59.6	14	40.4	0.4	0.2 1.1	.087
Subtype								
non-generalized	N, %w row	50	51.0	54	49.0	Ref.		
generalized	N, %w row	13	28.3	39	71.7	**2.9**	**1.2 - 6.8**	**.014** §**
Catastrophic anxiety cognitions something embarrassing or shameful could happen								
no	N, %w row	37	56.1	29	43.3	Ref.		
yes	N, %w row	26	33.2	64	66.8	**2.8**	**1.3 - 6.1**	**.008** §
being regarded as dumb or weak								
no	N, %w row	34	53.8	33	43.2	Ref.		
yes	N, %w row	29	26.2	60	63.8	**2.3**	**1.1 - 4.7**	**.029** §
Number of anxiety cognitions (1-9 dimensional)	M, SD	3.1	1.5	3.6	1.5	1.3	1.0 - 1.7	.052
Comorbidity at time of initital SAD [g]								
no co-occurring panic attacks	N, %w row	60	47.6	79	52.43	Ref.		
co-occurring panic attacks	N, %w row	3	15.7	14	84.29	**5.4**	**1.3 - 22.1**	**.020** §
Temperament/personality								
behavioral inhibition (total sum) [n]	M, SD	2.4	0.4	2.6	0.5	**1.4**	**1.1 - 1.9**	**.013**
behavioral inhibition (social fear) [n]	M, SD	2.6	0.7	2.9	0.7	**1.4**	**1.0 - 1.8**	**.021** §
harm avoidance [o]	M, SD	15.1	6.3	18.4	7.3	**1.1**	**1.0 - 1.1**	**.025** §**

[a] cumulated across available assessment waves after initial threshold SAD diagnosis (n = 156/209 SAD cases, for n = 53 no follow-up status was available due to drop out or first diagnosis of threshold SAD at T3)

[b] first SAD diagnosis at T0, T1 or T2 and at least 1subsequent follow-up available (N=156)

[g] derived from respective DIA-X/M-CIDI section at time when SAD was first reported

[n] from Retrospective Self-Report of Inhibition (RSRI)

[o] from Tridimensional Personality Questionaire (TPQ)

§ Variable entered in multiple regression analysis, ** p<.05, * p<.1

Figure 1: Diagnostic stability and remission of DSM-IV SAD: Conditional probability for diagnostic status at follow-up (T1, T2, and/or T3) among those with initial thresold DSM-IV SAD diagnosis (at T0, T1 or T2) (N=156).