Word count: 3,977

Abstract word count: 248

2 Figures, 3 Tables

Supplemental material: 4 Tables

The Role of Parental Psychopathology and Family Environment for Social Anxiety Disorder in the First Three Decades of Life

(parental psychopathology and family environment in social anxiety disorder)

Susanne Knappe, Dipl. Psych.¹

Roselind Lieb, PhD^{2,3}

Katja Beesdo, PhD¹

Lydia Fehm, PhD⁴

Nancy Chooi Ping Low, MD MS⁵

Andrew T. Gloster, PhD¹

Hans-Ulrich Wittchen, PhD^{1,3}

1 Institute of Clinical Psychology and Psychotherapy, Technische Universität Dresden, Germany

2 Epidemiology and Health Psychology, University of Basel, Switzerland

3 Clinical Psychology and Epidemiology, Max Planck Institute of Psychiatry Munich, Germany

4 Department of Psychology, Humboldt University Berlin, Germany

5 Department of Psychiatry, McGill University, Montreal, Canada

corresponding author and reprints:

Dipl. Psych. Susanne Knappe

Institute of Clinical Psychology and Psychotherapy, Technische Universität Dresden

Chemnitzer Str. 46

01187 Dresden, Germany

Phone: ++49-351-463-39727

Fax: ++49-351-463-36984

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

Abstract

Background. To examine the role of parental psychopathology and family environment for the risk of social anxiety disorder (SAD) in offspring from childhood to early adulthood, covering an observational period of 10 years.

Method. A community sample of 1,395 adolescents (aged 14 to 17 years at baseline) was prospectively followed-up over the core high risk period for SAD onset. DSM-IV offspring and parental psychopathology was assessed using the Munich-Composite International Diagnostic Interview; direct diagnostic interviews in parents were supplemented by family history reports from offspring. Parental rearing was assessed by the Questionnaire of Recalled Rearing Behavior in offspring, family functioning by the McMaster Family Assessment Device in parents.

Results. Parental SAD was associated with the offspring's risk to develop SAD (OR = 3.3, 95%CI: 1.4-8.0). Additionally, other parental anxiety disorders (OR = 2.9, 95%CI: 1.4-6.1), depression (OR = 2.6, 95%CI: 1.2-5.4) and alcohol use disorders (OR = 2.8, 95%CI: 1.3-6.1) were associated with offspring SAD. Offspring's reports of parental overprotection, rejection and lack of emotional warmth, but not parental reports of family functioning were associated with offspring SAD. Analyses of interaction of parental psychopathology and parental rearing indicated combined effects on the risk for offspring SAD.

Conclusions. These findings extend previous results in showing that both parental psychopathology and parental rearing are consistently associated with the risk for offspring SAD. As independent and interactive effects of parental psychopathology and parental rearing may have already manifested in early adolescence, these factors appear crucial and promising for targeted prevention programs.

Keywords: social anxiety, parental psychopathology, rearing styles, adolescence, interaction

corresponding author and reprints:

Dipl. Psych. Susanne Knappe

Institute of Clinical Psychology and Psychotherapy, Technische Universität Dresden Chemnitzer Str. 46

01187 Dresden, Germany

Phone: ++49-351-463-39727

Fax: ++49-351-463-36984

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

Introduction

Social anxiety disorder (SAD) is one of the most prevalent anxiety disorders with an estimated mean lifetime prevalence rate of 6.65%, with rates generally higher in females than in males (Fehm et al., 2005). SAD typically has its first onset during adolescence and young adulthood (Lépine et al., 1993; Magee et al., 1996; Merikangas et al., 2002; Wittchen & Fehm, 2003; Kessler et al., 2005; Beesdo et al., 2007). Parental psychopathology (Fyer et al., 1993; Mancini et al., 1996; Stein et al., 1998; Merikangas et al., 1999, 2002; Kessler et al., 2005) and several measures of family environment such as parental rearing styles (overprotection, rejection and emotional warmth), family functioning, and characteristics of parent-child interactions (Lieb et al., 2000b; Woodruff-Borden et al., 2002; Rapee & Spence, 2004; Gar et al., 2005) have been suggested as risk factors for the development of SAD among children and adolescents.

However, this evidence is frequently limited to clinical samples, retrospective reports, cross-sectional designs, and short periods of observation. To date, only Lieb et al. (2000b) prospectively examined these assumed risk factors in a 2-year follow up community sample of adolescents aged 14 to 17 years. They found that parental SAD, other parental psychopathology (other anxiety disorders, depressive disorders, alcohol use disorders), and perceived parental overprotection and parental rejection were associated with the development of SAD in offspring. Analyses of the interaction between parental psychopathology and rearing on the risk for offspring SAD also indicated that when parents were affected by SAD or any other psychopathology the association between parental rejection and adolescents' SAD was stronger. These findings by Lieb et al. were limited by the fact that a considerable proportion of this study sample of adolescents had not yet passed through the high risk period for onset of SAD. Thus, the number of cases in their analyses was limited, thereby probably diminishing effect sizes and statistical power. The authors had hypothesized that the coverage of a longer observation period with more diagnostic outcomes may lead to stronger parent-offspring associations, especially with regard to the generalized SAD subtype as well as for interactions with parental rearing and family functioning.

The current paper re-examines the sample of Lieb et al. and expands on this study in two important ways: First, using the original community sample of adolescents aged 14 to 17 at baseline, we examine the course and outcome over a period of up to 10 years, thus extending the observational period by an additional 8 years so that *all* respondents have now passed the core risk period for the

incidence of SAD. Second, we attempt to examine and replicate the associations between parental SAD and other parental psychopathology, parental rearing styles, and family functioning on the risk of SAD in the offspring, with a special focus on the interaction of these risk factors. Findings can improve our understanding of the associations between parental and offspring psychopathology. Identification of potentially malleable factors that interact with parental psychopathology and alter its effects on offspring SAD are of great importance to understand the development of the disorder and to design effective prevention programs.

Methods and Materials

Sample

Data were collected as part of the prospective longitudinal Early Developmental Stages of Psychopathology (EDSP) – Study. The EDSP Study is designed as a random regional representative population sample of a German community in the metropolitan area of Munich to study the natural course of early stages of substance use and other mental disorders and to identify risk factors for their onset and course. Detailed descriptions of the EDSP design and field procedures are reported elsewhere (Wittchen et al., 1998a, b; Lieb et al., 2000a). The study consists of a baseline survey conducted in 1995 (T0) with N = 3,021 individuals (response rate 71%) of a younger (aged 14-17 years at baseline) and a older study cohort (18-24 years at baseline). The first follow up (T1) was conducted approximately two years after baseline only for the younger cohort (mean interval 1.64 years, SD = 0.19, range 1.2-2.1) (N = 1,228; response rate: 87.8%). The second (T2) (N = 2,548, mean interval 3.47 years, SD = 0.25, range 2.8-4.1, response rate: 84.3%) and third follow up (T3) (N = 2,210, mean interval 8.38 years, SD = 0.65; range 7.4-10.6, response rate: 73.3%) were conducted approximately four and ten years after baseline for both cohorts. As the current study aims to study the original Lieb et al. (2000b) sample, we solely refer to the younger study cohort aged 14 to 17 at baseline (T0: N = 1,395, T1: N = 1,228; T2: N = 1,169; T3: N = 1,022). There was no selective drop out (attrition) from baseline (T0: N = 1,395) to 10-year follow up (T3: N = 1,022) for SAD (OR = 0.5, 95%CI: 0.3-1.1).

A key feature of the EDSP study is a special family supplement (EDSP-FS; Lieb et al., 2000a) to investigate familial contributions to the development of mental disorders in offspring. Primarily the mothers were interviewed. Fathers were interviewed if the mother was not available (lack of time,

deceased or not locatable). Parents of 1,053 respondents were interviewed directly, namely in 1,026 cases the mother, and in 27 cases the father (response rate 86%).

The EDSP project and its family genetic supplement has been approved by the Ethics Committee of the Medical Faculty of the Technische Universität Dresden (No: EK-13811). All participants (in cases of aged 18 or younger the parents) provided written informed consent.

Diagnostic assessment

Assessment of respondents

Mental disorders were assessed with the computer-assisted version of the Munich-Composite International Diagnostic Interview (DIA-X/M-CIDI; Wittchen & Pfister, 1997), an updated version of the World Health Organization's CIDI version 1.2 (WHO, 1990, 1992). Participants were interviewed face to face by trained clinical interviewers.

The M-CIDI allows for the standardized assessment of symptoms, syndromes and diagnoses of 48 mental disorders according to DSM-IV and ICD-10 criteria along with information about onset, duration, and severity. Consistent with prior publications from the EDSP study, the lowest age of onset reported was used. All diagnoses are based on the DSM-IV-DIA-X algorithms. Reliability and validity is moderate to good for all the disorders covered by the DIA-X/M-CIDI (Lachner et al., 1998, Reed et al., 1998). Test-Retest reliability for the SAD module is good ($\kappa = .57$) (Reed et al., 1998).

At baseline, the DIA-X/M-CIDI was used to assess lifetime and 12 month diagnoses. The follow-up surveys administered a modified version of the DIA-X/M-CIDI that covered the time interval since the last interview. The DIA-X/M-CIDI also incorporates an algorithm for the generalized subtype of SAD (3+ anxiety situations). To increase validity, respondent lists regarding possible situations of social fears and a list of social fear symptoms are used (Wittchen et al., 2001). Individuals who denied the DIA-X/M-CIDI stem question for "ever having a persistent, irrational fear of, and compelling desire to avoid a situation in which the respondent attended social affairs, like going to a party or meeting", were classified as non-cases.

Assessments of parents

Diagnostic information on parental psychopathology was derived from both direct and indirect sources. Direct interviews were conducted with one or both parents at the first follow up, indirect information was collected by family history reports from their offspring at baseline.

Parents' direct interviews were assessed using the DIA-X/M-CIDI at the first follow-up. All interviews were conducted by trained clinical interviewers who were blind to the diagnostic findings of their offspring. Most interviews took place in the parents' homes, separate from their offspring. Indirect parental diagnoses were derived from family history data collected with the offspring as informants at baseline using the M-CIDI family history module according to the Family History Research Diagnostic Criteria (Andreasen et al., 1977). Offspring were asked M-CIDI – questions to assess the key symptoms of parental DSM-IV disorders and whether their parent sought professional help because of his or her respective symptoms.

Assignment of parental diagnoses

Parental diagnoses (SAD, any other anxiety disorder, depressive disorders, alcohol use disorders) were estimated on the basis of *a priori* established algorithms, analogous to Lieb et al. (2000b). A parental diagnosis was assigned when positive diagnostic information was available from direct diagnostic interviews or family history information. When no indications for parental diagnoses from any of the two sources was given, parents were classified to have “no diagnosis”. According to their diagnosis parents were classified into four groups: parents with SAD with or without comorbid disorders, parents with other anxiety disorders than SAD (panic disorder, agoraphobia, generalized anxiety disorder, simple phobia, obsessive compulsive disorder, posttraumatic stress disorder), parents with depressive disorders excluding SAD (major depressive disorder, dysthymia), and parents with alcohol use disorders excluding SAD (alcohol abuse or dependence).

Lieb et al. (2000b) reported low sensitivity, but high specificity of agreement between directly assessed parental diagnoses and family history data.

Assessment of family environment

Parenting style was assessed with the Questionnaire of Recalled Parental Rearing Behavior (FEE, Schuhmacher et al., 1999), which assesses the offspring's perceived parental rearing behavior with regard to parental rejection, emotional warmth, and overprotection. Reliability and validity of the FEE have been reported to be high (Arrindel et al., 1994; Schuhmacher et al., 1999). The German Version of the McMaster Family Assessment Device (FAD) was used to assess six dimensions of family functioning in directly interviewed parents: problem solving, communication, role behavior, affective responsiveness, affective involvement, and behavior control. The FAD provides scores for

each of these subscales and also a “general functioning scale”, representing the overall ‘family climate’. Lower scores on the FAD scales reflect lower levels of family functioning. The questionnaire is based on the McMaster Model of Family Functioning and its reliability and validity have been well established (Kabacoff et al., 1990).

Statistical analysis

Respondents’ diagnostic information from the four assessment waves were aggregated for cumulative incidences (CLI; T0, T1, T2, T3) using the last observation carried forward (LOCF) method. Thus, N = 1,395 of the younger study cohort were available for Kaplan-Meier survival analysis and cox regressions (stratified for age and gender) to provide hazard ratios (HR) for the estimated cumulative incidences of respondents SAD, excluding observed SAD cases with missing age of onset (N = 2). Associations between parental psychopathology, parental rearing behavior, and family functioning with respondents SAD were assessed with odds ratios (ORs) from logistic regressions, controlled for gender and age. Associations between parental psychopathology and number of feared social situations were assessed with risk ratios (RR) from negative binomial regressions. Analyses were done using Stata 10.0 (Statacorp, 2007) with the alpha level fixed a priori at 0.05. We did not adjust for multiple testing because the individual tests were related to individual hypotheses and adjustment would treat them as reflecting a global hypotheses – which is questionable in substantive terms (Savitz & Olshan, 1995). Data (percentages, ratios, coefficients) were weighted for age, gender, geographic location, non-contact and non-response to match the distribution of the sampling frame (Lieb et al., 2000a); frequencies (N) are reported unweighted.

Results

Cumulative lifetime incidences for SAD

The observed cumulative lifetime incidence for respondents SAD up to the age of 28 (maximum age of oldest respondents) at T3 was 8.7% (weighted, unweighted N = 92/1,395), with females (weighted 10%, unweighted number N = 52/92) being more often affected than males (weighted 7.4%, unweighted number N = 40/92, gender ratio: OR = 1.7, 95%CI: 1.01-2.5). Twenty-five percent of SAD cases (weighted, unweighted number N = 23/92) fulfilled criteria for generalized SAD

at least once across all data waves. There was no association with gender in the proportion of generalized and nongeneralized SAD cases in the sample (OR = 0.7, 95%CI: 0.3-1.9).

The estimated age-dependent cumulative incidence rate for SAD at 28 years of age was 9.2% for the total sample, and 7.01% and 11.3% for males and females respectively (gender ratio: HR = 1.7, 95%CI: 1.1-2.5, $p = 0.010$) (Figure 1). At the ages of nine and ten, incidence rates were especially increased in females, compared to males, and this gender difference plateaued by age 14.

- Insert Figure 1 about here -

2. Familial risk factors and SAD in offspring

Parental psychopathology

Offspring of parents with SAD were more often affected by SAD (13.2%) than respondents without any parental psychopathology (4.2%, OR = 3.3, 95%CI:1.4-8.0) (Table 1). Compared to respondents without any parental psychopathology, rates of offspring SAD were also elevated when parents were affected by other anxiety disorders (11.7%, OR = 2.9, 95%CI: 1.4-6.1), depressive disorders (10.5%, OR = 2.6, 95%CI: 1.2-5.4), or alcohol use disorders (11%, OR = 2.8, 95%CI: 1.3-6.1). We also considered comorbidity among parental SAD. Results indicated the highest parent-offspring-association when parental SAD was highly comorbid (3 or more comorbid disorders) (OR = 7.8, 95%CI: 2.8-21.9). However, the specific parent-offspring-association for SAD remained significant after controlling for other parental psychopathology (OR = 1.3, 95%CI: 1.1-1.6).

- Insert Table 1 about here -

Compared to offspring without any parental psychopathology, the age-dependent incidence rate increased at most for offspring with parental SAD (HR = 2.9, 95%CI:1.3-6.5, $p = 0.013$) and respondents with any other parental psychopathology (HR = 2.3, 95%CI: 1.2-4.5, $p = 0.016$) (Figure 2). Controlling for any other parental psychopathology (anxiety disorders, depressive disorders, alcohol use disorders) did not change the association for offspring with parental SAD (HR = 2.3, 95%CI: 1.2-4.4, $p = 0.016$). Survival curves of respondents with parental SAD were not significantly

higher than those with any other parental psychopathology (HR = 1.3, 95%CI: 0.7-2.3, p = 0.473). Age-dependent incidence rates for the risk of SAD in offspring by parental diagnoses did not differ between male and female offspring.

- Insert Figure 2 about here -

Up to the 10-year follow up, among the 23 cases of generalized SAD only three had a parent without SAD, any other parental anxiety, depressive or alcohol use disorder, precluding sufficient statistical power for complete parent-offspring association testing. Thus, the number of social fears (range from 0 to 6) was used as a proxy for generalized SAD. We found that parental SAD (RR = 1.5, 95%CI: 1.02-2.2), parental depressive disorders (RR = 1.4, 95%CI: 1.1-2.1), marginally other parental anxiety disorders (RR = 1.4, 95%CI: 1.0-2.0), but not parental alcohol use disorders (RR = 1.3, 95%CI: 0.9-1.9) were associated with a higher number of feared social situations.

Family environment

Respondents with SAD reported more parental overprotection (OR = 1.5, 95%CI: 1.2-1.9), rejection (OR = 1.5, 95%CI: 1.2-1.9), and less emotional warmth (OR = 0.7, 95%CI: 0.5-0.9) compared to their non-socially phobic counterparts (Table 2). No differences between family functioning as measured by respondents' SAD status were found.

- Insert Table 2 about here –

As parental rearing may also be influenced by parents' mental health, associations between parental psychopathology and respondents SAD were controlled for parental rearing (upper part of Table 3). When controlling for parental rearing styles, all associations between parental psychopathology and offspring SAD remained significant. Also when associations between parental rearing and offspring SAD were controlled for parental psychopathology, associations remained significant (lower part of Table 3).

- Insert Table 3 about here -

Interested in whether the diagnostic status of the parent and parental rearing, and disturbed family functioning respectively, have combined effects on the offspring's risk for SAD, we first report on results regarding the interaction between parental psychopathology (SAD as well as other parental disorders) and rearing styles, and second, on results regarding the interaction between parental psychopathology and family functioning on the risk for offspring SAD.

Reports on parental rearing styles did not differ between offspring of parents with vs. without SAD. However, there was an interaction whereby the risk for SAD was increased in offspring of parents with SAD who reported greater rejection (OR = 3.2, 95%CI: 1.3-8.1), more overprotection (OR = 3.4, 95%CI: 1.4-8.5), and less emotional warmth (OR = 3.5, 95%CI: 1.4-8.8), compared to offspring of unaffected parents reporting less negative rearing styles. Because not only parental SAD, but also other parental anxiety disorders, depressive disorders, and alcohol use disorders were associated with offspring SAD, we collapsed all diagnostic categories into one, and re-run the main effect and interaction analyses for 'any parental disorder'. Associations with negative rearing styles and the risk for SAD were higher in offspring with any parental disorder, as compared to offspring without parental psychopathology and corresponding parental rearing styles (rejection: OR = 7.1, 95%CI: 2.02-24.8; overprotection: OR = 2.8, 95%CI: 1.0-8.1; lack of emotional warmth: OR = 1.9, 95%CI: 1.1-3.3). An interaction was found, whereby the risk for SAD was elevated in offspring of parents affected by any parental disorder and who reported greater overprotection (OR = 3.9, 95%CI: 1.4-11.1) or lack of emotional warmth (OR = 4.8, 95%CI: 1.5-14.9), compared to offspring of unaffected parents reporting lower overprotection or higher emotional warmth.

Regarding family functioning, parents with SAD scored lower on problem solving, communication, role behavior, affective responsiveness, behavior control and on the composite score of general functioning, possibly reflecting that parents with SAD report a more disturbed family functioning, irrespective of the child's diagnostic status. However, between parental SAD and family functioning, no interaction was found on the risk for offspring SAD. Notably, we observed interaction between any parental disorder and some scales of family functioning for the risk of offspring SAD. The risk for offspring SAD was higher in offspring of affected parents, when parents reported lower scores on problem solving (OR = 3.1, 95%CI: 1.1-8.5), role behavior (OR = 3.01, 95%CI: 1.2-7.9), behavior control (OR = 2.8, 95%CI: 1.1-7.01), and marginally on the composite score of general functioning (OR = 2.9, 95%CI: 1.0-7.8), compared to offspring of unaffected parents with higher FAD-scores.

- please see supplemental tables for further details -

Discussion

This study confirms and expands the prior work of Lieb et al. (2000b) to an observational period of almost 10 years, after all respondents of a young cohort from a representative community study have now passed through the high risk period for SAD onset. This extended observational period enhanced the validity of our prior results and increased the statistical power to describe patterns of SAD incidence within the first three decades of life. We confirmed associations between parental psychopathology and negative parental rearing styles for the risk of SAD onset in offspring. Beyond this, we were able to investigate the familial transmission of the generalized subtype of SAD, and to examine the interaction of parental psychopathology and family environment.

Consistent with previous family (e.g. Fyer et al., 1993; McClure et al., 2001) and twin studies (Kendler et al., 1999; Hettema et al., 2001), parental SAD was associated with onset of SAD in offspring. The strength of this association was also of similar magnitude to previous studies (e. g. Mancini et al., 1996; Lieb et al., 2000b; McClure et al., 2001). Also in agreement with the Lieb et al. original study, other parental mental disorders were found to substantially increase the risk for offspring SAD, indicating that a range of parental diagnoses may confer similar risk to the development of SAD in offspring. As the majority of parents reported multiple diagnoses (instead of SAD alone), the question arised to what degree rates of SAD in offspring are being driven by parental SAD specifically, versus other parental disorders and comorbidity among parental psychopathology in general. In our study, the parent-offspring-association for SAD remained significant, even after controlling for other parental psychopathology. Generally speaking, SAD is highly comorbid (Wittchen & Fehm, 2003; Ruscio et al., 2008) and associations between different parental disorders and offspring SAD merely reflect the high general psychopathology in families at risk for SAD or other mental disorders. In particular, evidence for the specificity of parent-to-offspring transmission of SAD is modest (Fyer et al. 1993; Cooper et al. 2006), at least in comparison to other anxiety disorders (McClure et al., 2001; Low et al., 2007). As further hypothesized by Lieb et al. in the original sample and supported by previous studies in clinical samples (Fyer et al., 1993; Stein et al., 1998), we tested whether the familial aggregation of the generalized SAD subtype is stronger, using the number of feared social situations as a proxy to the subtype: We found that associations for the number of social

fears among offspring were most pronounced when parental SAD, other anxiety disorders and depression (but no parental alcohol use disorders) were present, suggesting a possible familial liability for internalizing disorders.

Our findings underline the importance of parental psychopathology in the development of SAD. However, our observational data do not allow to draw conclusions about genetic, shared and non-shared environmental or gene-environmental interaction effects for SAD onset. As not all offspring of affected parents develop SAD, this indicates that further environmental factors may contribute to the development of SAD. For example, exposure to fear-inducing stimuli might be correlated in family members, hence predicting some familial-environmental component of liability to SAD (Kendler et al., 1999).

Accordingly, and in line with prior studies (Bögels et al., 2001; Woodruff-Borden et al., 2002; Greco & Morris, 2002; Taylor & Alden, 2006), family environment measures of perceived parental rejection, overprotection and lack of emotional warmth are associated with offspring SAD. Our interaction findings contribute further to this issue in that if parents are affected by psychopathology *in combination with* the offsprings' perception of parental rearing as more rejecting, overprotective or less warm, offspring are at increased risk for SAD. However, it remains open whether or not offspring's perception of pathological parenting corresponds to actual (pathological) parenting (Whalsey et al., 1999) or, if the perception is a result of offspring psychopathology. Our findings indicate that socially anxious offspring may elicit parental responses, and/or parents with SAD or other psychopathology respond to their offsprings behavior in a pathological manner. The rationale for this interpretation is that socially anxious offspring of affected parents did not report more negative parental rearing styles than offspring of unaffected parents and the lack of associations between family functioning and offspring SAD (also see Derisley et al., 2005). As these results provide the first description of an interaction between parental psychopathology and family environment on the risk for offspring SAD in a community sample, they require replication.

Future analyses following these findings would be to examine the mediator or moderator role of parental psychopathology and parental rearing. Results of Turner et al. (2003) indicate a moderator role of parental psychopathology. They found that anxious parents did not overtly restrict their children from engaging in a risky play or direct their children to less risky activities compared to nonanxious parents. However, anxious parents reported higher levels of distress, and more often expressed concern and negative affect about the activities of their children to them. Further research on the

mediator or moderator role of parental psychopathology should also consider, whether associations between parenting and the offspring diagnostic status differ by parental (anxiety) disorders. McClure et al. (2001) did not find evidence for a mediating role of perceived parenting in the association between maternal and child anxiety disorders, and concluded that anxiety disordered children are more likely to perceive their parents negatively if the parents also have an anxiety disorder – a conclusion not supported by our findings.

Limitations of our study include the low prevalence of SAD in fathers, so that parent-offspring-associations may be dominated by maternal SAD and full examination of parental gender effects on offspring SAD was not possible. Though offspring's reports of parental rearing styles were assessed retrospectively, this is alleviated by the fact that most of the respondents still live with their parents making their recollection more immediate.

Conclusions

The vast majority of SAD cases have their onset in early adolescence or the early 20s. Given the high risk for comorbidity and poorer outcomes in later life, this 'time window' appears to provide a critical opportunity for screening, detection and diagnosis, as well as early intervention. Importantly, associations between parental psychopathology and rearing style, in addition to their interaction which may confer increased risk for SAD in offspring, may have already manifested their effects by early adolescence. Further delineation of these relationships seems warranted as malleable components of these risk factors (e.g. possible treatment of SAD in parents) may provide potential targets for prevention programs that may alter the onset, course and sequel of this highly prevalent anxiety disorder.

Acknowledgements

This work is further part of the Early Developmental Stages of Psychopathology (EDSP) Study and is funded by the German Federal Ministry of Education and Research (BMBF) project no. 01EB9405/6, 01EB 9901/6, EB01016200, 01EB0140, and 01EB0440. Part of the field work and analyses were also additionally supported by grants of the Deutsche Forschungsgemeinschaft (DFG) LA1148/1-1, WI2246/1-1, WI 709/7-1, and WI 709/8-1.

Principal investigators are Dr. Hans-Ulrich Wittchen and Dr. Roselind Lieb.

Core staff members of the EDSP group are: Dr. Kirsten von Sydow, Dr. Gabriele Lachner, Dr. Axel Perkonigg, Dr. Peter Schuster, Dr. Michael Höfler, Dipl.-Psych. Holger Sonntag, Dr. Tanja Brückl, Dipl.-Psych. Elzbieta Garczynski, Dr. Barbara Isensee, Dipl.-Psych. Agnes Nocon, Dr. Chris Nelson, Dipl.-Inf. Hildegard Pfister, Dr. Victoria Reed, Dipl.-Soz. Barbara Spiegel, Dr. Andrea Schreier, Dr. Ursula Wunderlich, Dr. Petra Zimmermann, Dr. Katja Beesdo, Dr. Antje Bittner, Dipl.-Psych. Silke Behrendt and Dipl.-Psych. Susanne Knappe. Scientific advisors are Dr. Jules Angst (Zurich), Dr. Jürgen Margraf (Basel), Dr. Günther Esser (Potsdam), Dr. Kathleen Merikangas (NIMH, Bethesda), Dr. Ron Kessler (Harvard, Boston) and Dr. Jim van Os (Maastricht).

Financial disclosures and conflict of interest

S. Knappe, L. Fehm, N. Low, AT Gloster declare to have no conflict of interest.

R. Lieb has received speaking honoraria from Wyeth.

K. Beesdo has received speaking honoraria from Pfizer.

HU Wittchen has received Research Support from Eli Lilly and Company, Novartis, Pfizer, Schering-Plough. He is currently or has been a consult for Eli Lilly, GlaxoSmithKline Pharmaceuticals, Hoffman-La Roche Pharmaceuticals, Novartis, Pfizer, Wyeth. He has received speaking honoraria from Novartis, Schering-Plough, Pfizer, Wyeth.

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Table Legend

Table 1: Associations between parental psychopathology and offspring social anxiety disorder
(T0-T3, N = 1,395)

Table 2: Associations between parenting styles, family functioning and offspring social anxiety disorder

Table 3: Adjusted associations between parental psychopathology, parenting styles and offspring social anxiety disorder

Figure Legend

Figure 1: Onset of social anxiety disorder for total sample (N = 1,395) and by gender (based on n = 714 males and n = 681 females)

Figure 2: Onset of social anxiety disorder in offspring by parental psychopathology status (based on 1,053 respondents for whom diagnostic information of parents was available)

Table 1: Associations between parental psychopathology and offspring social anxiety disorder (T0-T3, N = 1,395)

Parental psychopathology	Offspring social anxiety disorder (SAD)							
	no SAD (N = 1,303)		SAD (N = 92)		OR	95%CI		p
	N	%w	N	%w				
social anxiety disorder	122	86.9	15	13.2	3.3	1.4	8.01	0.007
any other anxiety disorder	339	88.4	46	11.7	2.9	1.4	6.05	0.005
any depressive disorder	343	89.5	41	10.5	2.6	1.2	5.4	0.012
any alcohol use disorder	267	88.9	32	11.03	2.8	1.3	6.1	0.009
no anxiety, depressive or alcohol use disorder*	249	95.8	11	4.2				
<i>Comorbidity among parents with social anxiety disorder</i>								
no comorbidity (SAD alone)	10	84.6	1	15.4	2.9	0.4	23.01	0.296
low comorbidity**	79	88.8	6	51.2	1.7	0.6	5.3	0.346
high comorbidity***	33	11.2	8	48.8	7.8	2.8	21.9	0.000

*reference group for all comparisons

**defined as one or two comorbid disorders

***defined as three or more comorbid disorders

OR odds ratio, adjusted for age and gender

CI confidence intervall

p = 0.05, bold prints indicate statistical significance

Table 2: Associations between parenting styles, family functioning and offspring social anxiety disorder

Family environment	Offspring social anxiety disorder (SAD)							
	no SAD (N = 1,303)		SAD (N = 92)		OR*	95%CI		p
	mean	SD	mean	SD				
parenting style (FEE)								
overprotection	13.3	2.78	14.55	3.67	1.5	1.2	1.9	0.000
emotional warmth	23.71	3.86	22.96	4.63	0.7	0.5	0.9	0.002
rejection	9.51	1.58	10.3	2.45	1.5	1.2	1.9	0.002
family functioning (FAD)								
problem solving	1.76	0.44	1.78	0.41	1.2	0.7	1.9	0.508
communication	1.73	0.42	1.75	0.43	1.1	0.6	1.9	0.691
role behavior	1.85	0.37	1.88	0.33	1.09	0.6	1.9	0.771
affective responsiveness	1.64	0.51	1.59	0.53	0.9	0.5	1.4	0.527
affective involvement	1.61	0.42	1.62	0.41	0.8	0.4	1.4	0.399
behavior control	2.03	0.39	2.04	0.41	0.8	0.4	1.6	0.611
general functioning	1.62	0.42	1.66	0.45	1.06	0.6	1.9	0.847

Note: FEE = questionnaire of Recalled Parental Rearing Behavior (Schuhmacher et al., 1999), FAD = McMaster Family Assessment Device (Kabacoff et al., 1990)

*reference group are respondents without social anxiety disorder

OR odds ratio, adjusted for age and gender

CI confidence intervall

p = 0.05, bold prints indicate statistical significance

Table 3: Adjusted associations between parental psychopathology, parenting styles and offspring social anxiety disorder

	Offspring social anxiety disorder (SAD)			
	OR [*]	95%CI		p
Parental psychopathology status, adjusted for parenting style				
social anxiety disorder	3.3	1.3	8.6	0.013
any other anxiety disorder	2.5	1.1	5.4	0.024
any depressive disorder	2.3	1.02	5.09	0.044
any alcohol use disorder	2.5	1.08	5.8	0.033
Comorbidity among parents with social anxiety disorder				
no comorbidity (SAD alone)	5.1	0.6	46.8	0.148
low comorbidity**	2.9	0.4	23.01	0.296
high comorbidity***	8.1	2.8	23.8	0.000
Parenting style (FEE) adjusted for parental psychopathology status				
overprotection	1.5	1.2	1.9	0.001
emotional warmth	0.7	0.5	0.9	0.003
rejection	1.5	1.2	1.8	0.002

Note: FEE = questionnaire of Recalled Parental Rearing Behavior (Schuhmacher et al., 1999)

*reference group are respondents without parental psychopathology

**defined as one or two comorbid disorders

***defined as three or more comorbid disorders

OR odds ratio, adjusted for age and gender

CI confidence interval

p = 0.05, bold prints indicate statistical significance

Figure 1: Onset of social anxiety disorder (SAD) for total sample (N = 1,395) and by gender
(based on n = 714 males and n = 681 females)

Figure 2: Onset of social anxiety disorder in offspring by parental psychopathology status[#] (based on N = 1,053 respondents for whom diagnostic information of parents was available)

[#]: due to low case numbers, the graph of the cumulative incidence for respondents with parental social anxiety disorder was cut off

Supplemental material

S. Knappe, R. Lieb, K. Beesdo, L. Fehm, N. C. P. Low, A.T. Gloster, H.-U. Wittchen

The Role of Parental Psychopathology and Family Environment for Social Anxiety Disorder in the First Three Decades of Life

(parental psychopathology and family environment in social anxiety disorder)

corresponding author and reprints:

Dipl. Psych. Susanne Knappe

Institute of Clinical Psychology and Psychotherapy, Technische Universität Dresden

Chemnitzer Str. 46

01187 Dresden, Germany

Phone: ++49-351-463-39727

Fax: ++49-351-463-36984

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

Supplemental Table Legend

Suppl. Table 1: Interaction between parental SAD and parental rearing on offspring SAD

Suppl. Table 2: Interaction between any parental disorder and parental rearing on offspring SAD

Suppl. Table 3: Interaction between parental SAD and family functioning on offspring SAD

Suppl. Table 4: Interaction between any parental disorder and family functioning on offspring SAD

Suppl. Table 1: Interaction between parental SAD and parental rearing on offspring SAD

parental SAD* parental rearing		N	%w	risk for SAD in offspring			
				OR	95% CI		p
<i>overprotection</i>							
0	P+, high	54	5.62	3.44	1.39	8.52	0.008
1	P+, low	68	7.11	2.51	0.94	6.72	0.067
2	P-, high	400	41.75	2.15	1.23	3.77	0.007
3	P-, low	426	45.52	1.00			
0 vs. 1				1.29	0.38	4.40	0.688
0 vs. 2				1.60	0.66	3.86	0.294
<i>emotional warmth</i>							
0	P+, low	68	7.29	3.47	1.37	8.77	0.009
1	P+, high	54	5.42	2.63	0.96	7.19	0.059
2	P-, low	456	47.92	2.12	1.18	3.81	0.012
3	P-, high	371	39.38	1.00			
0 vs. 1				1.26	0.33	4.82	0.741
0 vs. 2				1.64	0.70	3.85	0.255
<i>rejection</i>							
0	P+, high	61	6.07	3.22	1.28	8.12	0.013
1	P+, low	61	6.64	1.59	0.63	4.02	0.324
2	P-, high	381	39.63	1.33	0.77	2.32	0.307
3	P-, low	446	47.66	1.00			
0 vs. 1				2.00	0.62	6.44	0.247
0 vs. 2				2.45	0.99	6.04	0.052

Note: P+, - indicated w/o parental social anxiety disorder

high: high rates on parental rearing scales (FEE above median split)

low: low rates on parental rearing scales (FEE, below median split)

OR odds ratio, adjusted for age and gender

CI confidence interval

p < 0.05, bold prints indicate statistical significance

Suppl. Table 2: Interaction between any parental disorder* and parental rearing on offspring SAD

				risk for SAD in offspring			
any parental disorder* parental rearing		N	%w	OR	95% CI		p
<i>overprotection</i>							
0	P+, high	347	35.88	3.95	1.41	11.08	0.009
1	P+, low	377	40.25	1.93	0.67	5.56	0.223
2	P-, high	95	9.82	1.36	0.34	5.54	0.665
3	P-, low	130	14.05	1.00			
0 vs. 1				2.05	1.18	3.54	0.010
0 vs. 2				2.84	0.99	8.14	0.052
<i>emotional warmth</i>							
0	P+, low	413	43.4	4.76	1.52	14.90	0.007
1	P+, high	311	32.74	2.59	0.79	8.45	0.115
2	P-, low	111	11.81	2.15	0.52	8.86	0.289
3	P-, high	114	12.06	1.00			
0 vs. 1				1.86	1.05	3.31	0.034
0 vs. 2				1.64	0.70	3.85	0.255
<i>rejection</i>							
0	P+, high	355	36.97	2.15	0.88	5.27	0.093
1	P+, low	368	39.12	1.25	0.50	3.13	0.638
2	P-, high	99	10.4	0.30	0.07	1.31	0.110
3	P-, low	126	13.51	1.00			
0 vs. 1				1.74	1.01	2.98	0.046
0 vs. 2				7.08	2.02	24.77	0.002

Note: P+, - indicated w/o parental social anxiety disorder

high: high rates on parental overprotection, rejection, emotional warmth (above median split)

low: low rates on parental overprotection, rejection, emotional warmth (below median split)

* includes parental social anxiety disorder, other anxiety disorders, depressive disorders, alcohol use disorders

OR odds ratio, adjusted for age and gender

CI confidence interval

p < 0.05, bold prints indicate statistical significance

Suppl. Table 3: Interaction between parental SAD and family functioning on offspring SAD

parental SAD* family functioning				risk for SAD in offspring			
				N	%w	OR	95% CI
problem solving							
0	P+, high	64	5.97	0.87	0.26	2.87	0.822
1	P+, low	72	6.87	2.30	1.05	5.04	0.037
2	P-, high	320	30.93	1.14	0.67	1.96	0.623
3	P-, low	584	56.23	1.00			
0 vs. 1				0.37	0.11	1.26	0.110
0 vs. 2				0.72	0.21	2.50	0.610
communication							
0	P+, high	69	6.53	0.79	0.24	2.60	0.698
1	P+, low	67	6.31	2.56	1.16	5.67	0.020
2	P-, high	353	34.58	1.13	0.67	1.90	0.652
3	P-, low	551	52.58	1.00			
0 vs. 1				0.29	0.09	1.00	0.050
0 vs. 2				0.69	0.21	2.27	0.538
role behavior							
0	P+, high	81	7.57	1.16	0.42	3.18	0.773
1	P+, low	55	5.27	3.06	1.26	7.40	0.013
2	P-, high	397	38.64	1.57	0.94	2.61	0.087
3	P-, low	507	48.52	1.00			
0 vs. 1				0.38	0.11	1.27	0.115
0 vs. 2				0.75	0.27	2.04	0.573
affective responsiveness							
0	P+, high	72	6.94	0.69	0.21	2.26	0.534
1	P+, low	64	5.90	2.61	1.18	5.73	0.017
2	P-, high	344	34.00	0.94	0.55	1.60	0.809
3	P-, low	560	53.16	1.00			
0 vs. 1				0.25	0.07	0.89	0.032
0 vs. 2				0.73	0.21	2.49	0.613
affective overinvolvement							
0	P+, high	82	7.59	1.00	0.39	2.53	0.996
1	P+, low	54	5.25	2.28	0.92	5.61	0.074
2	P-, high	368	36.07	0.92	0.55	1.55	0.762
3	P-, low	535	51.09	1.00			
0 vs. 1				0.45	0.13	1.55	0.205
0 vs. 2				1.09	0.41	2.86	0.862

Suppl. Table 3: Interaction between parental SAD and family functioning on offspring SAD (continued)

parental SAD* family functioning		N	%w	risk for SAD in offspring			
				OR	95% CI		p
<i>behavior control</i>							
0	P+, high	71	6.94	1.03	0.38	2.80	0.955
1	P+, low	65	5.93	2.69	1.13	6.41	0.026
2	P-, high	413	39.85	1.30	0.78	2.17	0.321
3	P-, low	489	47.29	1.00			
0 vs. 1				0.41	0.11	1.47	0.170
0 vs. 2				0.79	0.28	2.19	0.650
<i>general functioning</i>							
0	P+, high	82	7.81	0.83	0.28	2.45	0.739
1	P+, low	54	5.03	3.27	1.40	0.76	0.006
2	P-, high	414	40.19	1.21	0.73	2.02	0.458
3	P-, low	490	46.97	1.00			
0 vs. 1				0.25	0.07	0.84	0.025
0 vs. 2				0.69	0.23	2.02	0.494

Note: P+, - indicated w/o parental social anxiety disorder

high: high rates on family functioning scales (FAD, above median split)

low: low rates on family functioning scales (FAD, below median split)

OR odds ratio, adjusted for age and gender

CI confidence interval

p < 0.05, bold prints indicate statistical significance

Suppl. Table 4: Interaction between any parental disorder* and family functioning on offspring SAD

				risk for SAD in offspring			
any parental disorder* family functioning		N	%w	OR	95% CI		p
<i>problem solving</i>							
0	P+, high	304	29.12	3.07	1.10	8.55	0.031
1	P+, low	477	45.90	3.77	1.43	9.95	0.007
2	P-, high	80	7.78	2.27	0.60	8.51	0.225
3	P-, low	179	17.20	1.00			
0 vs. 1				0.82	0.47	1.41	0.466
0 vs. 2				1.45	0.48	4.32	0.509
<i>communication</i>							
0	P+, high	339	32.86	2.16	0.87	5.33	0.096
1	P+, low	442	42.16	2.41	1.01	5.74	0.047
2	P-, high	83	8.26	0.76	0.19	2.96	0.688
3	P-, low	176	16.72				
0 vs. 1				0.90	0.54	1.51	0.689
0 vs. 2				2.94	0.92	9.40	0.069
<i>role behavior</i>							
0	P+, low	384	37.03	3.02	1.16	7.86	0.024
1	P+, high	397	37.99	2.58	0.98	6.77	0.055
2	P-, low	94	9.18	1.31	0.35	4.90	0.693
3	P-, high	165	15.80	1.00			
0 vs. 1				1.17	0.70	1.94	0.550
0 vs. 2				2.34	0.83	6.54	0.107
<i>affective responsiveness</i>							
0	P+, low	324	32.06	1.98	0.76	5.17	0.162
1	P+, high	457	42.96	2.71	1.10	6.70	0.031
2	P-, low	92	8.89	0.87	0.24	3.23	0.840
3	P-, high	167	16.09	1.00			
0 vs. 1				0.74	0.43	1.25	0.255
0 vs. 2				2.32	0.79	6.81	0.127
<i>affective overinvolvement</i>							
0	P+, low	362	34.81	1.92	0.78	4.68	0.154
1	P+, high	419	40.21	2.28	0.95	5.48	0.066
2	P-, low	89	8.85	0.60	0.15	2.37	0.467
3	P-, high	170	16.13	1.00			
0 vs. 1				0.84	0.51	1.40	0.509
0 vs. 2				3.37	1.04	10.92	0.043

Suppl. Table 4: Interaction between any parental disorder* and family functioning on offspring SAD (continued)

any parental disorder* family functioning		N	%w	risk for SAD in offspring			
				OR	95% CI		p
<i>behavior control</i>							
0	P+, low	381	36.98	2.77	1.10	7.01	0.031
1	P+, high	399	38.07	2.79	1.10	7.08	0.030
2	P-, low	103	9.80	1.29	0.34	4.87	0.711
3	P-, high	155	15.14	1.00			
0 vs. 1				0.99	0.59	1.65	0.963
0 vs. 2				2.20	0.74	6.51	0.154
<i>general functioning</i>							
0	P+, low	401	38.68	2.50	0.92	6.80	0.073
1	P+, high	380	36.34	2.90	1.07	7.83	0.036
2	P-, low	95	9.32	1.19	0.33	4.36	0.788
3	P-, high	164	15.66	1.00			
0 vs. 1				0.87	0.52	1.44	0.576
0 vs. 2				2.09	0.79	5.52	0.135

Note: P+, - indicated w/o parental social anxiety disorder

high: high rates on family functioning scales (FAD, above median split)

low: low rates on family functioning scales (FAD, below by median split)

* includes parental social anxiety disorder, other anxiety disorders, depressive disorders, alcohol use disorders

OR odds ratio, adjusted for age and gender

CI confidence interval

p < 0.05, bold prints indicate statistical significance