

# Associations of familial risk factors with social fears and social phobia: evidence for the continuum hypothesis in social anxiety disorder?

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**Abstract** We examined parental psychopathology and family environment in subthreshold and DSM-IV threshold conditions of social anxiety disorder (SAD) in a representative cohort sample of 1,395 adolescents. Offspring and parental psychopathology was assessed using the DIA-X/M-CIDI; recalled parental rearing and family functioning via questionnaire. Diagnostic interviews in parents were supplemented by family history reports from offspring. The cumulative lifetime incidence was 23.07% for symptomatic SAD, and 18.38 and 7.41% for subthreshold and threshold SAD, respectively. The specific parent-to-offspring association for SAD occurred for threshold SAD only. For subthreshold and threshold SAD similar associations were found with other parental anxiety disorders, depression and substance use disorders. Parental rearing behaviour, but not family functioning, was associated with offspring threshold SAD, and although less strong and less consistent, also with subthreshold SAD. Results suggest a continued graded relationship between familial risk factors

and offspring SAD. Parental psychopathology and negative parental styles may be used defining high-risk groups to assign individuals with already subthreshold conditions of SAD to early intervention programs.

**Keywords** Social phobia · Social anxiety disorder · Parental psychopathology · Family environment · Continuum

## Introduction

Social Phobia, also known as social anxiety disorder (SAD), is characterized by a marked and persistent fear of at least one social or performance situation, in which the person is exposed to the scrutiny of others and where embarrassment or humiliation might occur (American Psychiatric Association 1994). DSM-IV SAD is one of the most prevalent and burdensome mental disorders with a mean lifetime prevalence of 6.7% in European (Fehm et al. 2005) and up to 12.1% in US-samples (NCS-R; Kessler et al. 2005). SAD has its onset typically in early adolescence (Beesdo et al. 2007; Lépine et al. 1993; Magee et al. 1996; Merikangas et al. 2002; Kessler et al. 2005; Wittchen et al. 2001) and often persists into adulthood (Chartier et al. 1998; Keller 2006). Findings from prospective studies indicate low diagnostic stability on the DSM-IV threshold level, but also a low likelihood of full remission from DSM-IV SAD, and frequent shifts of SAD symptoms around the diagnostic threshold (Emmelkamp and Wittchen 2008; Merikangas et al. 2002). Accordingly, prevalence rates of those who suffer from social fears below the DSM-IV diagnostic threshold range nearly up to 25% in general population samples (Davidson et al. 1994; Egger and Angold 2006; Fehm et al. 2008; Milne et al. 1995; Ruscio et al. 2008;

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Wittchen et al. 1999). Social fears below the diagnostic threshold are comparable to DSM-IV threshold SAD in terms of associated impairment, disability in various domains of daily living (Davidson et al. 1994; Fehm et al. 2008; Keenan et al. 1997; Stein et al. 2000), and development of comorbid/subsequent anxiety, depression and substance use disorders (Crum and Pratt 2001; Merikangas et al. 2002; Zhang et al. 2004). Social fears below the diagnostic threshold are further associated with an increased risk for progression into DSM-IV threshold SAD (Costello et al. 1999; Ialongo et al. 1995), and thereby are a sensitive and useful indicator for later psychopathology and substantial impairment in psychosocial functioning. Thus, early diagnosis of first SAD symptoms may assist in the prevention of more severe psychiatric symptoms.

Parental SAD and other parental mental disorders, negative parental rearing styles (Lieb et al. 2000b; Turner et al. 2003; Woodruff-Borden et al. 2002), and problematic family functioning or attachment (Bögels et al. 2001; Hudson and Rapee 2001) have been suggested as important risk factors for DSM-IV threshold SAD. However, it remains unclear to what degree these familial risk factors may also contribute to SAD below the diagnostic threshold. The aim of the present study is therefore to examine the associations of parental psychopathology, parental rearing and family functioning with subthreshold and threshold SAD. Findings are based on a cohort of 1,395 adolescents from a representative community sample aged 14 to 17 years that was followed-up prospectively across the high risk period for the SAD onset from early adolescence into the third decade of life. Results of the present study may also contribute to the question of whether social fears below the diagnostic threshold are part of a continuum of severity or rather represent a discrete phenomenon with more or less arbitrarily derived thresholds distinct from DSM-IV threshold SAD. Implications from these results for clinical practice will be discussed.

## Materials and methods

### 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 (Lieb et al. 2000a; Wittchen et al. 1998). 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 an older study cohort (18–24 years at baseline). The first follow-up (T1) was conducted approximately 2 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%) were conducted approximately 4 and 10 years after baseline for both cohorts. In this study, we refer to the younger study cohort aged 14–17 at baseline (T0:  $N = 1,395$ , T1:  $N = 1,228$ ; T2:  $N = 1,169$ ; T3:  $N = 1,022$ ), because family environment variables were only assessed in the younger subsample. No selective drop out (attrition) from baseline (T0:  $N = 1,395$ ) to 10-year follow-up (T3:  $N = 1,022$ ) occurred 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 offspring, namely, in 1,026 cases the mother, and in 27 cases the father (response rate 86%), were interviewed directly.

The EDSP project and its family genetic supplement have been approved by the Ethics Committee of the Medical Faculty of the Technische Universität Dresden (No: EK-13811). All participants provided written informed consent.

### Diagnostic assessment

#### *Assessment of offspring psychopathology*

Mental disorders were assessed with the computer-assisted version of the Munich-Composite International Diagnostic Interview (DIA-X/M-CIDI) (Wittchen and 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 DIA-X/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 the onset, duration, and severity. All diagnoses are based on the DSM-IV/DIA-X algorithms. Reliability and validity are 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 acceptable ( $kappa = 0.57$ ; 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. To increase validity, respondent lists regarding possible situations of social fears and a list of social fear symptoms were used (Wittchen et al. 2001).

The diagnostic status of offspring was categorized into four mutually exclusive groups using the highest diagnostic status across all waves. Offspring who positively affirmed exclusively 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 labelled as symptomatic SAD. Offspring were asked to give a concrete example for each item endorsed to allow for clarification. For subthreshold SAD, additionally a marked and persistent fear of social or performance situations in which the person is exposed to unfamiliar people or to possible scrutiny by others (criterion A) and three of the following criteria had to be fulfilled: Exposure to social or performance situations almost invariably provokes an immediate anxiety response (criterion B), individuals recognize that their fear is excessive or unreasonable (criterion C), the feared situations are avoided or else are endured with intense distress (criterion D), and social fears interfere significantly with psychosocial functioning (criterion E). If all DSM-IV criteria (A, B, C, D, and E) were met, a threshold SAD diagnosis (DSM-IV) was assigned. As estimation of impairment due to SAD may be of limited reliability in respondents of younger age, for this study, the impairment criterion (E) was only required for an SAD diagnosis, when respondents were 18 years or older. Individuals, who denied the stem question, were classified as non-socially anxious.

#### *Assessments of parental psychopathology*

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 were collected by family history reports from their offspring at baseline.

Parents' direct interviews were assessed using the DIA-X/M-CIDI. 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 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, analogue 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 of parental diagnoses from any of the two sources were given, parents were classified to have “no diagnosis”. According to their diagnoses 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, and obsessive compulsive disorder), parents with depressive disorders excluding SAD (major depressive disorder and 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 in offspring at first follow-up with the Questionnaire of Recalled Parental Rearing Behaviour (German: Fragebogen zum Erinnerten Elterlichen Erziehungsverhalten, FEE, Schumacher et al. 1999, original by Perris et al. 1980), administered to offspring. The FEE assesses three dimensions of offsprings perceived parental rearing behaviour, namely, parental rejection, emotional warmth, and overprotection. Reliability and validity of the FEE have been reported to be high. The German Version of the McMaster family assessment device (FAD, Arrindel et al. 1994) was used to assess six dimensions of family functioning, namely, problem solving, communication, role behaviour, affective responsiveness, affective involvement, and behaviour control, in directly interviewed parents at first follow-up. The FAD provides scores for each of these subscales and also a ‘general functioning scale’, representing the overall ‘family climate’. 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*

Offspring diagnostic information from the four assessment waves was aggregated for observed cumulative lifetime

incidences (CLI; T0, T1, T2, and T3), using the last observation carried forward (LOCF) method. Analyses are based on respondents for whom information about parental psychopathology was available ( $N = 1,053$ ). Associations between parental psychopathology, parental rearing behaviour, and family functioning with offspring SAD were assessed with odds ratios (ORs) from multinomial logistic regressions, controlled for age and gender. Analyses were done using Stata 10.0 (StataCorp 2007). Results (percentages, ratios, coefficients) are 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 up to age 28 for SAD

Observed cumulative lifetime incidence up to age 28 (maximum age of oldest respondents at last wave) was 7.41% ( $N = 82/1,053$ ) for DSM-IV threshold SAD, and 18.38% ( $N = 190/1,053$ ) and 23.07% ( $N = 244/1,053$ ) for subthreshold and symptomatic SAD, respectively. Females were significantly more often affected by threshold SAD (8.96 vs. 5.90%, OR = 1.92, 95% CI 1.16–3.20,  $P = 0.012$ ) and subthreshold SAD (21.89 vs. 14.96%, OR = 1.83, 95% CI 1.28–2.60,  $P = 0.001$ ) than males. For symptomatic SAD, no gender difference was found (22.50 vs. 23.66%, OR = 1.32, 95% CI 0.95–1.82,  $P = 0.099$ ).

### Parental psychopathology and the risk for offspring SAD

Among the 1,053 mothers and fathers,  $N = 137$  parents (134 mothers and 3 fathers) met criteria for SAD,  $N = 385$  parents (375 mothers and 10 fathers) reported any other anxiety disorder,  $N = 384$  parents (368 mothers and 16 fathers) reported any depressive disorder, and  $N = 299$  parents (288 mothers and 11 fathers) reported any alcohol use disorder. In sum,  $N = 793$  parents (769 mothers, 24 fathers) were affected by any of these disorders, and only  $N = 260$  parents (257 mothers and 3 fathers) reported no anxiety, depressive or alcohol use disorder. The latter group served as reference group for the following comparisons.

As shown in Table 1, the proportion of offspring affected by SAD and whose parents are affected by psychopathology themselves increased with the diagnostic level of offspring SAD (column percentages). Among offspring without SAD, 12.10% had parents with SAD.

For offspring with symptomatic, subthreshold and DSM-IV threshold SAD, rates of parental SAD were 13.77, 11.67 and 17.41%, respectively. Rates of affected parents similarly increased with the diagnostic status of offspring SAD when parents were affected by other anxiety disorders (range 53.72–80.42%), depressive disorders (range 53.80–78.42%), or alcohol use disorder (range 47.87–73.89%).

Table 1 additionally shows the associations between parental psychopathology and offspring SAD status (row percentages, ORs), indicating the risk for offspring SAD when parents are affected by SAD, any other anxiety disorder, depressive disorders or alcohol use disorder in comparison to offspring of unaffected parents. For symptomatic SAD in offspring, no associations with parental psychopathology were found. For subthreshold SAD in offspring, associations with other parental anxiety disorders (20.82%, OR = 1.92, 95% CI: 1.17–3.16,  $P = 0.010$ ), depressive disorders (22.25%, OR = 1.99, 95% CI 1.21–3.26,  $P = 0.006$ ) and alcohol use disorder (21.71%, OR = 1.96, 95% CI 1.16–3.30,  $P = 0.011$ ) were found. Offspring of parents with SAD did not report higher rates of subthreshold SAD (16.75%, OR = 1.44, 95% CI: 0.75–2.78,  $P = 0.271$ ), compared to offspring of unaffected parents (13.54%). Relative to symptomatic SAD, associations between parental psychopathology and offspring subthreshold SAD were not substantially higher (results not shown in table; parental SAD: OR = 1.11, 95% CI 0.54–2.28,  $P = 0.771$ ; other parental anxiety disorders: OR = 1.51, 95% CI 0.85–2.69,  $P = 0.156$ ; parental depressive disorders: OR = 1.70, 95% CI 0.96–3.00,  $P = 0.070$ ; parental alcohol use disorder: OR = 1.67, 95% CI 0.91–3.05,  $P = 0.098$ ).

Strongest associations with parental SAD occurred for offspring DSM-IV threshold SAD (10.08%, OR = 3.21, 95% CI 1.21–8.49,  $P = 0.019$ ) in comparison to offspring of unaffected parents (3.65%). Because also other parental anxiety disorders (10.13%, OR = 3.44, 95% CI 1.58–7.53,  $P = 0.002$ ), depressive disorders (9.11%, OR = 3.02, 95% CI 1.35–6.59,  $P = 0.007$ ), and alcohol use disorder (8.89%, OR = 3.07, 95% CI 1.34–7.03,  $P = 0.008$ ) were substantially associated with offspring DSM-IV threshold SAD, the specific parent-to-offspring association for SAD was controlled for other parental psychopathology. However, the association remained marginally significant (OR = 1.30, 95% CI 1.00–1.71,  $P = 0.057$ ). Noteworthy, associations between parental psychopathology and offspring DSM-IV threshold versus subthreshold SAD did not differ for parental SAD (results not shown in table; parental SAD: OR = 2.34, 95% CI 0.78–7.02,  $P = 0.131$ ), or for other parental anxiety disorders (OR = 1.83, 95% CI 0.76–4.39,  $P = 0.178$ ), depressive disorders (OR = 1.76, 95% CI 0.76–4.39,

**Table 1** Associations between parental psychopathology and offspring SAD status

	Parental psychopathology						Offspring SAD status														
	No SAD (N = 537)		Symptomatic SAD (N = 244)		Subthreshold SAD (N = 190)		DSM-IV threshold SAD (N = 82)		Symptomatic vs. no SAD		Subthreshold vs. no SAD		DSM-IV threshold vs. no SAD								
	N	%w**	%w***	N	%w**	%w***	N	%w**	%w***	OR	95% CI	P	OR	95% CI	P						
Social anxiety disorder	67	12.10	48.35	36	13.77	24.82	22	11.67	16.75	1.30	0.76	2.23	0.342	1.44	0.75	2.78	0.271	3.21	1.21	8.49	0.019
Any other anxiety disorder <sup>a</sup>	179	53.72	46.85	81	58.87	22.20	83	69.50	20.82	1.23	0.80	1.90	0.347	1.92	1.17	3.16	0.010	3.44	1.58	7.53	0.002
Any depressive disorder <sup>b</sup>	178	53.80	47.81	83	56.89	20.83	86	70.53	22.25	1.13	0.73	1.73	0.584	1.99	1.21	3.26	0.006	2.99	1.35	6.59	0.007
Any alcohol use disorder <sup>c</sup>	144	47.87	47.67	66	52.04	21.65	62	64.87	21.71	1.17	0.74	1.83	0.509	1.96	1.16	3.30	0.011	3.07	1.34	7.03	0.008
Any disorder <sup>d</sup>	381	70.90	48.27	185	75.21	23.10	155	81.66	19.98	1.23	0.85	1.79	0.275	1.77	1.12	2.80	0.014	2.88	1.37	6.07	0.005
No anxiety, depressive or alcohol use disorder <sup>*</sup>	156	29.10	59.81	59	24.79	22.99	35	18.34	13.54	1.00				1.00							1.00

SAD social anxiety disorder, SAD groups are mutually exclusive, OR odds ratio, adjusted for age and gender, CI confidence interval, N unweighted

\* Reference group for all comparisons, %w weighted percentages, %w\*\*\* column percentages, %w\*\*\* row percentages

P < 0.05, italic values indicate statistical significance

<sup>a</sup> Panic disorder, agoraphobia, generalized anxiety disorder, simple phobia, obsessive compulsive disorder; parents with social anxiety disorder were excluded

<sup>b</sup> Major depressive disorder, dysthymia; parents with social anxiety disorder were excluded

<sup>c</sup> Alcohol abuse and dependence; parents with social anxiety disorder were excluded

<sup>d</sup> Social anxiety disorder, any other anxiety disorder, any depressive disorder, any alcohol use disorder

$P = 0.178$ ) or alcohol use disorder ( $OR = 1.77$ , 95% CI 0.68–4.62,  $P = 0.244$ ).

Overall, associations between parental psychopathology and offspring SAD status were independent of offspring age or gender.

### Family environment and the risk for offspring SAD

For offspring with *symptomatic SAD*, no associations with recalled parental rearing behaviour occurred (Table 2). Offspring with *subthreshold SAD* reported more parental overprotection ( $OR = 1.08$ , 95% CI: 1.01–1.16,  $P = 0.021$ ) and more parental rejection ( $OR = 1.12$ , 95% CI 1.01–1.25,  $P = 0.043$ ) than offspring without SAD. In comparison to threshold SAD, offspring with subthreshold SAD reported less rejection ( $RR = 0.84$ , 95% CI 0.72–0.98,  $P = 0.024$ ), and marginally less overprotection ( $RR = 0.91$ , 95% CI 0.83–1.01,  $P = 0.058$ ).

Offspring with *DSM-IV threshold SAD* reported significantly more parental overprotection ( $OR = 1.19$ , 95% CI 1.09–1.30,  $P = 0.000$ ), more parental rejection ( $OR = 1.34$ , 95% CI 1.15–1.55,  $P = 0.000$ ), and less emotional warmth ( $OR = 0.90$ , 95% CI 0.83–0.97,  $P = 0.007$ ) than their non-socially phobic counterparts (Table 2). Overall, perceptions of negative parental rearing styles increased with the diagnostic status of offspring SAD.

Analyses of the family functioning scales revealed no associations between any specific scale and offspring SAD. Therefore, only the overall general functioning scale score is reported in Table 2.

As parental psychopathology and parental rearing may influence each other, we controlled the associations between parental psychopathology and offspring SAD for parental rearing (Table 3, upper part), as well as the associations between parental rearing and offspring SAD for parental psychopathology (Table 3, lower part). This did not alter the associations.

## Discussion

The present study examined the associations of parental psychopathology and familial environment with subthreshold and threshold conditions of SAD in a cohort study of a representative community sample of adolescents and young adults. The major finding of our study is that associations of parental psychopathology and negative parental rearing styles with social fears increased in magnitude towards the DSM-IV threshold, suggesting a continued graded relationship between familial risk factors and SAD in offspring.

**Table 2** Associations of parenting styles and family functioning and offspring SAD status

Family environment	Offspring SAD status																			
	No SAD		Symptomatic SAD		Subthreshold SAD		DSM-IV threshold SAD		Symptomatic versus no SAD		Subthreshold versus no SAD		DSM-IV threshold versus no SAD							
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	OR <sup>a</sup>	95% CI	P	OR <sup>b</sup>	95% CI	P	OR <sup>c</sup>	95% CI	P			
<i>Parenting style (FEE)</i>																				
Overprotection	13.25	2.74	13.41	2.86	13.75	3.16	14.64	3.28	1.02	0.96	1.08	0.583	1.08	1.01	1.16	0.021	1.19	1.09	1.30	0.000
Emotional warmth	23.79	3.92	23.54	3.71	23.28	4.08	22.21	4.94	1.00	0.95	1.04	0.832	0.96	0.92	1.01	0.106	0.90	0.83	0.97	0.007
Rejection	9.43	1.50	9.69	1.74	9.72	1.72	10.38	2.50	1.08	0.97	1.19	0.156	1.12	1.01	1.25	0.043	1.34	1.15	1.55	0.000
<i>Family functioning (FAD)</i>																				
General functioning	1.60	0.41	1.65	0.44	1.67	0.43	1.68	0.47	1.24	0.84	1.83	0.289	1.30	0.86	1.96	0.206	1.51	0.81	2.80	0.190

SAD social anxiety disorder, SAD groups are mutually exclusive, FEE questionnaire of recalled parental rearing behaviour, FAD McMaster Family Assessment Device, SD standard deviation, OR odds ratio, adjusted for age and gender, CI confidence interval

$P < 0.05$ , italic values indicate statistical significance

**Table 3** Associations between parental psychopathology, parenting style and offspring SAD status

	Offspring SAD status											
	Symptomatic SAD			Subthreshold SAD			DSM-IV threshold SAD					
	OR	95% CI	<i>P</i>	OR	95% CI	<i>P</i>	OR	95% CI	<i>P</i>			
<i>Parental psychopathology status, controlled for parenting style (FEE)</i>												
Social anxiety disorder	1.32	0.75	2.36	0.338	1.43	0.71	2.92	0.319	3.27	1.14	9.34	0.027
Any other anxiety disorder <sup>a</sup>	1.26	0.79	2.02	0.326	2.01	1.18	3.44	0.011	3.02	1.31	6.98	0.010
Any depressive disorder <sup>b</sup>	1.15	0.73	1.82	0.539	1.94	1.14	3.31	0.015	2.71	1.16	6.34	0.022
Any alcohol use disorder <sup>c</sup>	1.19	0.73	1.93	0.490	1.91	1.08	3.38	0.026	2.86	1.18	6.97	0.021
Any disorder <sup>d</sup>	1.25	0.84	1.88	0.273	1.80	1.09	2.95	0.021	2.74	1.23	6.12	0.014
<i>Parenting style (FEE), controlled for parental psychopathology</i>												
Overprotection	1.02	0.96	1.08	0.584	1.08	1.01	1.16	0.021	1.18	1.08	1.29	0.000
Emotional warmth	1.00	0.96	1.04	0.875	0.96	0.92	1.01	0.130	0.91	0.84	0.98	0.012
Rejection	1.08	0.97	1.19	0.159	1.12	1.00	1.25	0.048	1.33	1.15	1.54	0.000

SAD social anxiety disorder, SAD groups are mutually exclusive, FEE questionnaire of recalled parental rearing behaviour, OR odds ratio, adjusted for age and gender, comparison group are offspring without parental psychopathology (upper part) and offspring without SAD (lower part), respectively, CI confidence interval

$P < 0.05$ , bold values indicate statistical significance

<sup>a</sup> Panic disorder, agoraphobia, generalized anxiety disorder, simple phobia, obsessive compulsive disorder; parents with social anxiety disorder were excluded

<sup>b</sup> Major depressive disorder, dysthymia; parents with social anxiety disorder were excluded

<sup>c</sup> Alcohol abuse and dependence; parents with social anxiety disorder were excluded

<sup>d</sup> Social anxiety disorder, any other anxiety disorder, any depressive disorder, any alcohol use disorder

Consistent with previous family studies (Fyer et al. 1993; Hettema et al. 2001; Kendler et al. 1999; Lieb et al. 2000b; McClure et al. 2001; Stein et al. 2001), parental SAD was associated with offspring DSM-IV threshold SAD, but not with subthreshold or symptomatic SAD. However, associations with other parental anxiety, depressive or alcohol use disorders were found for DSM-IV threshold as well as for subthreshold SAD in similar magnitude. Noteworthy, controlling the specific SAD parent-offspring association for other parental disorders did not substantially change the findings. Current knowledge regarding the specificity of the intergenerational transmission of SAD is still scarce and heterogeneous (e.g. Fyer et al. 1993; but see Cooper et al. 2006), likely due to the fact that SAD frequently co-occurs with other disorders, and pure (non-comorbid) cases are rare. Our results indicate low to moderate specificity for the intergenerational transmission of SAD: the specific parent-to-offspring association for SAD remained significant also after controlling for other parental disorders. Nevertheless, other parental psychopathology (other anxiety, depressive, substance use disorders) were similarly associated with DSM-IV threshold SAD, but also with subthreshold SAD. From that, the familial aggregation of SAD may likely also be determined by cross-disorder risk factors (Johnson et al. 2006), as well as family based developmental conditions.

Concordant with prior studies (Bandelow et al. 2004; Bögels et al. 2001; Lieb et al. 2000b; Taylor and Alden

2006; Woodruff-Borden et al. 2002), offspring reports of higher parental overprotection, rejection and lower emotional warmth were associated with DSM-IV threshold SAD. Although attenuated in magnitude, and less consistently for emotional warmth, offspring with subthreshold SAD similarly reported a more negative parental rearing, probably reflecting their SAD risk status. However, it remains unclear whether offspring reports of parental rearing correspond to actual 'pathological parenting' or, instead, rather reflect an information processing (Christensen et al. 2003; Heerey and Kring 2007; Voncken et al. 2006) or attributional bias (Alfano et al. 2006; Taylor and Wald 2003; Wilson and Rapee 2005) in offspring with SAD. Given that our findings did not change after controlling for parental psychopathology and that parental reports of family functioning were not associated with the risk for SAD in offspring, offspring perceptions of negative parental rearing may merely reflect their psychopathology.

Overall, our findings point to similarities and differences between subthreshold and threshold SAD. Subthreshold and threshold SAD were similarly associated with parental psychopathology, except for parental SAD. Also, offspring negative perceptions of family environment were similarly associated with subthreshold and threshold SAD, except for the lack of emotional warmth. Thus, parental psychopathology and familial environmental conditions may both be useful in identifying individuals at risk for subthreshold

and threshold SAD. In addition, there may be dose-response relationship between familial risk factors and offspring SAD status as indicated by the increasing magnitude of associations towards the DSM-IV diagnostic threshold. It will be of interest for future research to investigate, in a more detailed manner, whether a certain number or pattern of risk factors is associated with subthreshold or threshold SAD. Importantly, prospective analyses to identify the temporal relationship of parental psychopathology, parental rearing and the transition from subthreshold to threshold SAD status are needed.

The present findings may also contribute to the discussion about whether social fears below and above the diagnostic threshold represent a continuum of severity (Kessler et al. 2003) or are discrete phenomena with more or less arbitrarily derived thresholds. Mild to moderate social fears are an almost universal experience, and it is crucial to find the balance between under-recognition and under-treatment of SAD on the one hand, and overpathologizing SAD symptoms on the other hand (Wakefield et al. 2005). Previous research indicated that even social fears below the diagnostic threshold are associated with high comorbidity (Fehm et al. 2008; Merikangas et al. 2002; Zhang et al. 2004), impairment and disability (Davidson et al. 1994; Keenan et al. 1997; Stein et al. 2000), comparable to DSM-IV threshold SAD. Individuals at risk for SAD may shift across a continuum for SAD liability (Kendler et al. 1999), depending on the number or kind of risk factors that are present. A continuum-hypothesis would also more precisely reflect the natural, oscillating course of the disorder (Merikangas et al. 2002; Spitzer 1998; Wittchen and Fehm 2003). A broadened conception of SAD would have the advantage of increasing the statistical power of studies examining potential genetic and environmental risk factors for the development of excessive social fears.

Our results should also be discussed in the light of several limitations. At first we could not examine if the offspring risk for SAD in general and for DSM-IV threshold SAD in particular is additionally increased when both parents are affected (as compared to one affected parent) due to the low prevalences of affected fathers. A second limitation concerns the retrospective assessment of parental rearing, although the majority of adolescents still lived with their parents at the time of assessment.

In conclusion, the current study confirms the importance of familial risk factors for SAD in a representative community sample of adolescents and young adults. Important novel findings indicate that (1) the graded relationship between familial risk factors and offspring SAD status provides further evidence for a continuum of social fears and social phobia, (2) there may be a dose-response relationship between familial risk factors and

diagnostic status of SAD in offspring, and (3) the specific intergenerational transmission of SAD between parents and their offspring is modest and may be limited to DSM-IV threshold SAD. Given the associated burden of social fears below the diagnostic threshold, parental psychopathology and offspring perceptions of the family environment are useful in identifying individuals at risk for subthreshold and threshold SAD. With regard to implications for clinical practice, assessment of parental psychopathology and perceived parental rearing may not only help to identify individuals at risk for subthreshold and threshold conditions of SAD, but also help to offer treatment at an early state to prevent them from developing DSM-IV threshold SAD and other, comorbid or subsequent psychopathology.

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