A Family History Study of Selective Mutism

DENISE A. CHAVIRA, ELISA SHIPON BLUM, CARLA HITCHCOCK, SHARON COHAN, MURRAY B. STEIN

From the Department of Psychiatry, University of California, San Diego and the Selective Mutism Group-Child Anxiety Network

This study was supported in part by an unrestricted research grant from GlaxoSmithKline and a research grant (MH64122) from NIMH to Dr. Stein.

Reprint request to Dr. Chavira, 8950 Villa La Jolla Drive, Ste. B218, La Jolla, CA 92037

ABSTRACT

Objective: To examine the history of psychiatric disorders in the parents of children with selective mutism (SM) compared to parents of children in a control group. **Method:** Seventy parent dyads (n = 140) of children with SM and 31 parent dyads (n = 62) of children without SM were interviewed with the Structured Clinical Interview for DSM-IV (SCID-IV & SCID-II) anxiety disorders, mood disorders, avoidant personality disorder (AVPD), and schizoid personality disorder modules via telephone. Interviewers were blind to proband status. The NEO Personality Inventory (NEO-PI-R) was also administered as an assessment of personality traits. **Results:** Generalized social anxiety disorder (GSAD) was present in 37% of SM parents, compared with 14.1% of control parents ($X^2 = 10.98$, $p < .001$; Odd Ratio = 3.58). AVPD was present in 17.5% of the SM parents and in 4.7% of control parents ($X^2 = 6.18$, $p < .05$; OR = 4.32). The proportion of parents with other psychiatric disorders was not different between groups. SM parents had higher Neuroticism (N) and Openness (O) scores on the NEO-PI-R than control parents. **Conclusions:** These results support earlier uncontrolled findings of a strong relationship between GSAD and SM. Such data also support the familial (though not necessarily solely genetic) nature of SM. **Key Words:** selective mutism, child anxiety, social anxiety, genetics

Selective mutism (SM) is characterized by the inability to speak in select social situations (e.g., school) despite speaking in other situations (American Psychiatric Press, 1994). According to DSM-IV criteria, SM is associated with significant impairment, has a duration of at least one month, and is not due to a lack of knowledge or comfort with speaking a language or accounted for by the presence of a communication, psychotic, or pervasive developmental disorder. SM is a relatively rare disorder, with population prevalence estimates consistently in the range of 1% (Bergman et al., 2002; Brown and Lloyd, 1975; Elizur and Perednik, 2003). Extant data suggest that SM usually begins in early childhood, often during the preschool years when a child is first required to speak in formal settings such as school and daycare. Little is known about the naturalistic course of SM. The few studies that do exist suggest that even though mutism may frequently remit over time (Steinhausen et al., 2006) rates of "talking" behaviors remain lower than average (Bergman et al., 2002) and residual psychopathology such as social phobia and other anxiety disorders often persists (Steinhausen et al., 2006).

The etiology of selective mutism is not well understood. Previous explanations offered that overcontrolling or hostile parenting, intrapsychic conflicts, or past trauma contributed to the onset of selective mutism; however limited data exist to support any of these positions (Anstendig, 1999; Black and Uhde, 1995). Other studies suggest that child oppositionality may contribute to the "refusal to speak" yet data are mixed in this regard (Cunningham et al., 2006; Yeganeh et al., 2003; 2006). To date, most research supports the position that SM is related to social anxiety disorder (SAD) and that they share common etiologies. Cross-sectional comorbidity rates between SM and SAD range from 70-95% (Black and Uhde, 1995; Dummitt et al., 1997) and characteristics such as shy,

anxious, withdrawn and serious are used to describe both selective mutism and social anxiety alike (Kumpulainen et al., 1998; Steinhausen and Juzi, 1996). Findings from family history studies also support a relationship between SM and SAD. In a study of personality characteristics, as assessed with the Millon Clinical Multiaxial Inventory-II (MCMI-II) (Millon, 1987), 39% of mothers and 32% of fathers of SM children were classified as shy/socially anxious versus 4% of mothers and 1% of fathers of controls. The avoidant and schizoid scales of the MCMI also predicted membership in the SM index group for mothers and fathers, respectively (Kristensen and Torgensen, 2001). Using a different assessment of temperament, parents of children with SM (n = 38) reported greater taciturnity in 1st, 2nd, and 3rd degree relatives when compared to parents of control children (n =31) (Steinhausen and Adamek, 1997). In the only family study that included a diagnostic assessment (N = 30 families with a child diagnosed with SM), 37% of the first degree relatives had SM and 70% had social phobia. In that study, information was initially gathered by checklist format and then followed up by unstructured clinical interviews; a control group was not included (Black & Uhde, 1995). While findings are not conclusive, in general, data support a relationship between social anxiety and SM.

Aims of Current Study

The current study builds on past findings in its assessment of personality traits and psychiatric disorders among parents of children with and without a SM diagnosis. Several methodological improvements have been made to improve the validity of the findings. For example: 1) A control group is included to provide appropriate comparisons; 2) Well-established semi-structured diagnostic interviews are used rather than informal

assessments and 3) Multiple clinicians, blind to proband status, serve to minimize diagnostic bias.

METHOD

Study procedures

This study is part of a larger project which includes the collection of DNA samples from families of children with SM. A nationwide sample has been recruited by means of two sources: 1) a website sponsored by a non-profit organization for children with selective mutism (the Selective Mutism Group~Child Anxiety Network), and 2) parent oriented conferences organized by this same non-profit group. Control families have been recruited through community advertisements and a website advertising participation in research studies.

Interested parents were sent a consent form. A child assent form was also included as this study is part of a larger study, where genetic samples were collected from both children and parents. Those families who returned their consent forms were screened over the telephone with the Selective Mutism module of the Anxiety Disorders Schedule for Children–Parent Report (Silverman and Albano, 1996) and the Selective Mutism Questionnaire (Bergman et al., 2001) to determine if their child did or did not have an SM diagnosis. A series of screening questions assessing developmental delays and communication difficulties was also included and a portion of families provided videotapes of their children speaking at home. Families in the SM group were eligible if the proband screened positive for SM and did not screen positive for psychotic, developmental, or communication disorders. Control families were eligible if they did not screen positive for SM, psychotic, developmental, or communication disorders.

Appointments were scheduled by a study coordinator (who was not blind to proband diagnostic status) who assigned interviewers (who were blind to proband diagnostic status) to conduct the parent interviews by telephone. Self-report questionnaires were returned by mail. All study procedures were approved by the Institutional Review Board at our institution.

Participants

This study included 70 mother-father dyads (n = 140) with a proband child who had an SM diagnosis and 31 control mother-father dyads (n = 62) where an SM diagnosis was not present. The participation of both biological parents and having a child between the ages of 3-11 were initial requirements for this study. The mean age of the proband in both groups was 6.8 years (SD = 2.8 years for controls and SD = 3.7 years for SM).

Analyses

Chi square analyses and one way ANOVAs were conducted to compare the demographic characteristics across groups. Omnibus chi square analyses were used to compare the distribution of DSM-IV disorders (lifetime) across parents of SM and control children. Analyses were also run separately for mothers and fathers and logistic regressions were used to test for an interaction between proband status (SM or control parents) and parent gender for each psychiatric disorder. Multivariate analyses of variance were conducted with personality dimensions and facets as the dependent variables and proband status (SM or control) as the independent variable.

Screening Measures

The Anxiety Disorders Interview Schedule-Parent Report (ADIS-C/P) (Silverman and Albano, 1996) is a semi-structured diagnostic interview designed to assess DSM-IV

childhood anxiety disorders as well as depressive and behavioral disorders. Published kappa coefficients for the ADIS-C/P disorders are 0.88 for separation anxiety, 0.86 for social anxiety disorder, 0.65 for specific phobia, 0.72 for generalized anxiety disorder, and 1.00 for ADHD (Silverman et al., 2001). The selective mutism module was administered as part of the screening procedure to gather information about the proband's SM symptoms. This module was also modified to be administered to parents to inquire about whether they ever had SM symptoms in their lifetime.

The Selective Mutism Questionnaire (SMQ) (Bergman et al., 2001) is a parent report measure of child SM behaviors and SM related impairment. It queries speaking behaviors in three domains; school, home/family, and public settings. Data from 576 parents have revealed a meaningful factor structure with adequate psychometric properties (Bergman et al., 2001). This measure was used as an additional assessment to confirm the presence or absence of "talking" behaviors.

Family History Measures

The Structured Clinical Interview for DSM-IV Disorders (First et al., 1997) was used to assess various Axis I disorders. For the current study, we included the depressive, manic, psychotic, and anxiety disorders modules. Questions were phrased in terms of "ever in your life". The SCID is a widely used semi-structured diagnostic interview and its reliability and validity have usually been in the fair to good range (First et al., 2000; Williams et al., 1992).

In this study, diagnostic reliability was conducted on 15% of the SCID interviews. Kappa statistics were used to calculate reliabilities and ranged from .50-1.00, suggesting a moderate to acceptable range for most diagnoses. The kappa statistics for social

phobia, generalized social anxiety disorder (GSAD), nongeneralized social anxiety disorder, or any social anxiety disorder were .65, .50, and 0.75, respectively. Kappas were .60 for major depression, .65 for dysthmia, .60 for generalized anxiety disorder, .88 for specific phobia, and .65 for past history of SM. Kappas for childhood separation anxiety disorder, PTSD, and panic with agoraphobia were all 1.0.

The Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II) (First et al., 1997) is a semi-structured interview used to obtain diagnoses for the Axis II disorders of the DSM-IV. In this study, only the Avoidant Personality Disorder and Schizoid Personality Disorder modules were administered. Fair-good median interrater kappas have been found for the more commonly occurring axis II disorders (Renneberg et al., 1992; Zanarini et al., 2000), and test-retest kappas have also been found to be in the fair-good range (First et al., 1995; Zanarini et al., 2000).

The NEO Personality Inventory Revised (Costa and McCrae, 1992) (NEO-PI-R) is a widely used measure of personality with well-established psychometric properties (Costa and McCrae, 1988; Costa et al., 1991). It includes 240 items which assess personality domains that are consistent with a five factor model of personality: 1) Neuroticism (N); 2) Extraversion (E); 3) Openness (O); 4) Agreeableness; 5) Conscientiousness (C). Each of the five domains also has six, lower level facets. Gender normed T-scores are calculated. According to gender specific norms developed by Costa and McCrae (1992), T scores ranging from 45-54 are “average”, scores between 55-65 are “high”, and scores above 65 are considered “very high.”

Consensus Meetings. All interviews performed in this study were made by phone and audiotaped with the participant’s permission. Interviewers received approximately 8

weeks of training on the diagnostic instruments and were required to meet gold standard criteria (at least 80% agreement on 3 mock cases) prior to conducting actual interviews. All interviewers had prior experience in research and/or clinical settings and had a masters, or doctoral degree. Weekly consensus meetings, at which each and every case was reviewed in detail, were held for the purposes of determining final diagnoses. DSM-IV guidelines, clinical judgment, review of audiotapes, and ultimately majority votes (when necessary) were used to establish final diagnoses.

RESULTS

Parent Demographics

The demographic characteristics of parents of SM children and controls are presented in Table 1. As shown in Table 1, there were no differences in age of parent, education, or ethnicity across the SM and control groups; X^2 analyses and ANOVAs were used.

Psychiatric Disorders

The distribution of lifetime Axis I and Axis II psychiatric disorders is presented in Table 2. Parents of SM children had significantly higher lifetime rates of generalized social anxiety disorder (GSAD) (OR = 3.6, CI = 1.6-7.9) and avoidant personality disorder (AVPD) (OR = 4.3, CI = 1.3-14.9) than the parents of the control children. Rates of nongeneralized social anxiety disorder (mostly public speaking phobias) as well as other psychiatric disorders were similar across groups. Additional chi-square analyses were conducted to examine the additive effect of both parents having GSAD compared to only one parent. There was no association between proband status and both parents having GSAD compared to only one parent having GSAD (X^2 (1, n = 60) = .14, p =.71),

or for both parents having AVPD compared to only one parent having AVPD (X^2 (1, n = 27) = .49, p = .48).

Gender: Simple main and moderating effects

When the distribution of psychiatric disorders was compared in mothers and fathers separately, significant relationships emerged for GSAD and AVPD. Fathers of SM children had significantly higher rates of GSAD (X^2 (1, n = 101) = 7.9, p = .005) and AVPD than their control counterparts (X^2 (1, n = 101) = 3.97, p = .046). The pattern was different for mothers. There was a trend toward significance for GSAD (X^2 (1, n = 101) = 3.62, p = .057), but not for AVPD (X^2 (1, n = 101) = 2.45, p = .13). Logistic regression analyses were conducted to test for an interaction between gender and proband status predicting the various psychiatric disorders, with gender, ethnicity, and the interaction term entered as the independent variables. Neither the effect of gender nor the proband status by gender interactions were significant.

NEO-PI-R domains and facets

MANOVA analyses revealed a main effect of proband status on the NEO-PI-R domains *Wilks F* (5, 165) = 2.16, p = .005. As shown in Figure 1, parents of SM children had significantly higher mean T-scores on the Neuroticism and lower mean T-scores on the Openness domains than parents of control children. There were no significant differences for Extraversion, Agreeableness, or Conscientiousness. Two MANOVAs were conducted on the six lower level Neuroticism facets (anxiety, angry hostility, depression, self-consciousness, impulsiveness, and vulnerability) and the six Openness facets (fantasy, aesthetics, feelings, actions, ideas, values); *Wilks F* (6, 164 = 2.16, p = .05) and *Wilks F* (6, 164) = 2.56, p = .02), respectively. As shown in Table 3, the parents

of the SM children scored higher than control parents on the anxiety, depression, self-consciousness and vulnerability facets of Neuroticism. Parents of the SM children scored lower than parents on control children on the aesthetics and ideas facets of Openness.

DISCUSSION

Our data support a strong phenomenological relationship between SM and generalized social anxiety disorder (GSAD). In this study, parental GSAD and AVPD were three-to-four-fold more common in the SM group than in the control group. GSAD has often been characterized as a more severe form of social anxiety and it is possible that SM may be an early onset form of the disorder. Similarly, AVPD, which shares much in common with GSAD (Holt et al., 1992; Ralevski et al., 2005), may also lie on this same continuum of social anxiety, and represent a more severe form of the disorder during adulthood. Without additional longitudinal research, it is difficult to know whether such a continuum actually exists or whether these relationships are a product of an imperfect diagnostic classification system where criteria are overlapping. At minimum, data support the conclusion that SM is related to GSAD, and like GSAD, has a familial and, likely, heritable component (Mannuzza 1995; Stein et al., 1998).

Quantitative assessments of dispositional characteristics may be most informative when trying to understand the heritable component of mental disorders. In a family history study of GSAD, probands scored significantly higher than first-degree relatives of comparison subjects on measures of trait and social anxiety, as well as on the anxiety-related personality trait known as harm avoidance (Stein et al., 1998). In a community sample of 4,564 pairs of 4 –year old twins, findings support some degree of genetic influence for various anxiety related behaviors however the contributions for general

distress, separation anxiety, and fear were modest, while the contributions for obsessive-compulsive behaviors and shyness/inhibition (heritability estimate was 62%) were substantial (Eley et al., 2003). If SM has a heritable component, it is likely that dimensional traits such as neuroticism or temperamental characteristics (e.g., behavioral inhibition, shyness) are those variables which are transmitted. In the current study, parents of children with SM reported higher levels of neuroticism, which was further characterized as higher levels of anxiety, self-consciousness, depression and vulnerability. In general such individuals are more susceptible to psychological distress, irrational ideas, and less effective coping strategies. Previous research suggests a possible relationship between certain candidate genes, in particular the serotonin transporter promoter polymorphism, and neuroticism (Schinka et al., 2004; Sen et al., 2004; Stein and Bienvenu, 2004). It is possible that such genetic factors and associated vulnerabilities may be present in children with SM. However given that only a portion of children with such characteristics (e.g., behavioral inhibition) develop later anxiety disorders (i.e., social anxiety disorder) (Schwartz et al., 1999) the etiology of SM is likely complex, incorporating environmental factors and likely multiple genes. Traumatic conditioning experiences, family environment, and parenting styles may facilitate the actual expression of SM (Elizur and Perednik, 2003; Kumplainen et al., 1998; Yeganeh et al., 2006) however additional research is necessary in this regard. In this study, parents of SM children also had lower Openness scores than control parents. Openness to Experience can represent a willingness to entertain novel ideas and unconventional values; those who score low on Openness often prefer the familiar to the novel and tend to be more conventional in behavior and conservative in outlook. The enrichment of

these attributes in parents of SM children is understandable, particularly in the presence of elevated rates of social anxiety disorder in this group.

The variability in the SM phenotype also warrants mention at this point. As has been suggested, it is possible that different developmental pathways to SM exist (Cohan et al., 2006; Kristensen and Torgensen, 2002). For example, in a study of SM children with and without communication disorders, significant differences were found in the temperamental characteristics of both the children and the parents (Kristensen and Torgensen, 2002). The SM children with communication disorders (COD) were rated as more social than SM children without COD although both groups were rated as more shy than controls. Similarly, the mother and fathers of the SM children with COD did not differ from the controls on the various temperament scales while parents of SM children without communication disorders had temperaments that were similar to their children. According to the authors, there may be two different developmental pathways to SM, where in the presence of a communication disorder, shyness and social anxiety directly concern the language impairment, whereas in the absence of such deficits, the shyness and social anxiety may be a more temperamental characteristic. In either case, it is likely that both genetic and environmental factors are involved. Additional research is necessary to identify whether phenotypic subtypes exist and whether genetic factors differentiate these groups.

Limitations

This study is limited by our use of telephone interviews and reliance on parents' report. Telephone interviews were used to facilitate nationwide recruitment and are associated with the standard limitations of not conducting interviews in person. Given the

low base rates of selective mutism however, it would have been difficult to recruit a large sample size by only ascertaining subjects from one geographic region. The use of telephone interviews as well as the young age of our probands led us to rely on parent report of their child's selective mutism. It is possible that such reports may have been inconsistent with the child's report however given the visibility of such behaviors (i.e., not talking) it is likely that ascertainment of SM is less prone to parent-child discrepancies than some other disorders. Another limitation is the fact that we did not include a formal assessment of communication disorders and pervasive developmental disorder. A clinical psychologist with experience in working with SM families used a validated diagnostic instrument to assess for SM and an SM severity questionnaire was also included. An informal clinical interview and a series of questions on a screening form were used to query developmental delays, language deficits, psychotic symptoms, and past psychiatric history. In-person standardized assessments to comprehensively assess for the presence of these disorders would have been optimal, yet in this case not feasible.

Lastly, there is the possibility of a sampling bias. We recruited families from a website as well as from national conferences which suggests that these families may have had more motivation to educate themselves about SM than other parents of children with SM. Perhaps this motivation may have stemmed from parents themselves having more social anxiety symptoms. It is also possible however, that parents with more social anxiety would have been less likely to engage such services, at least the in-person conferences, which could have led to a bias in the opposite direction. Lastly, in this study we excluded children with significant language impairments and perhaps in doing so,

captured a sample of families with a more temperamentally influenced form of social anxiety.

Clinical Implications

Taken together, these findings suggest that SM is diagnostically related to generalized social anxiety disorder and may be a familial phenomenon. At this point, it is not possible to disentangle genetic contributions from environmental forces (e.g., social learning). Future twin studies, adoption studies, and genetic marker designs will further inform questions of this nature. As suggested by others, SM may be an indicator of underlying psychopathology that has a more protracted course than the mere not talking symptoms that are the hallmark of SM (Steinhausen et al., 2006). More broadly, SM may act as a risk factor for later phobic and anxiety disorders and therefore in the presence of persistent SM, early intervention is warranted. Parents who suffer from social anxiety and their children may benefit from this knowledge, particularly in the presence of validated treatments for child social anxiety disorder (Beidel et al., 2004; Kendall et al., 1997; Wagner et al., 2004) and new treatments for selective mutism are emerging. Furthermore, if varying subtypes of SM exist (e.g., SM children with and without communication deficits, SM children with severe social anxiety, SM children with oppositional behaviors), then a one size fits all approach to treatment may be insufficient for such a varied group and interventions may need to be modified to meet the diverse needs of children in this diagnostic category.

TABLE 1. Parent Demographics for SM and Controls

	SM Fathers N = 70	Control Fathers N = 31	SM Mothers N = 70	Control Mothers N = 31
Parent Age	M = 40.4 SD = 6.4	M = 41.7 SD = 6.6	M = 38 SD = 5.5	M = 40.23 SD = 7.4
	F = 2.9, p = .09		F = .78, p = .38	
Education				
High school	17.1%	12.9%	11.4%	3.3%
Some college	15.7%	19.4%	21.4%	13.3%
College degree	31.4%	16.1%	38.6%	40%
Graduate degree	35.7%	51.6%	28.6%	43.3%
	X^2 (3) = 3.63, p = .30		X^2 (3) = 3.65, p = .31	
Ethnicity				
Caucasian	91.4	74.2	94.3	90.3
Latino	4.3	9.7	2.9	6.5
African American	--	3.2	--	3.2
Asian	1.4	3.2	1.4	--
Filipino/Pacific Islander	0	0	1.4	--
Mixed Race	2.9	9.7	--	--
	X^2 (4) = 6.42, p = .17		X^2 (4) = 3.89, p = .42	

TABLE 2. Distributions of psychiatric disorders across parents of SM Probands and Controls

	SM		Controls			
	N	%	N	%	X^2	p
MDD	40	29%	17	26.6%	.12	.72
Mania	1	.7%	0	0	.47	.50
Hypomania	3	2.2%	0	0	1.41	.50
Dysthymia	5	3.6%	1	1.6%	.64	.42
OCD	5	3.6%	2	3.1%	.03	.86
PTSD	6	4.3%	3	4.7%	.01	.91
Panic/Agor	4	2.9%	2	3.1%	.02	.93
Specific Phobia	13	9.5%	6	9.4%	.001	.98
GSAD	**51**	**37%**	**9**	**14.1%**	**10.98**	**.001**
NGSAD	9	6.6%	4	6.3%	.005	.94
GAD	14	10.2%	2	3.1%	2.99	.08
AVPD	**24**	**17.5%**	**3**	**4.7%**	**6.18**	**.013**
SPD	0	0	0	0	0	0
SM	6	4.4%	2	3.1%	.18	.67
SEP	8	5.8%	1	1.6%	1.84	.18

Note: MDD = Major Depressive Disorder, OCD = Obsessive Compulsive Disorder, PTSD = Post-traumatic Stress Disorder, Panic/Agora = Panic Disorder with Agoraphobia, GSAD = Generalized Social Anxiety Disorder, NGSAD = Non-generalized Social Anxiety Disorder, GAD = Generalized Anxiety Disorder, AVPD = Avoidant Personality Disorder, SPD = Schizoid Personality Disorder, SM = Selective Mutism, SEP = Separation Anxiety Disorder

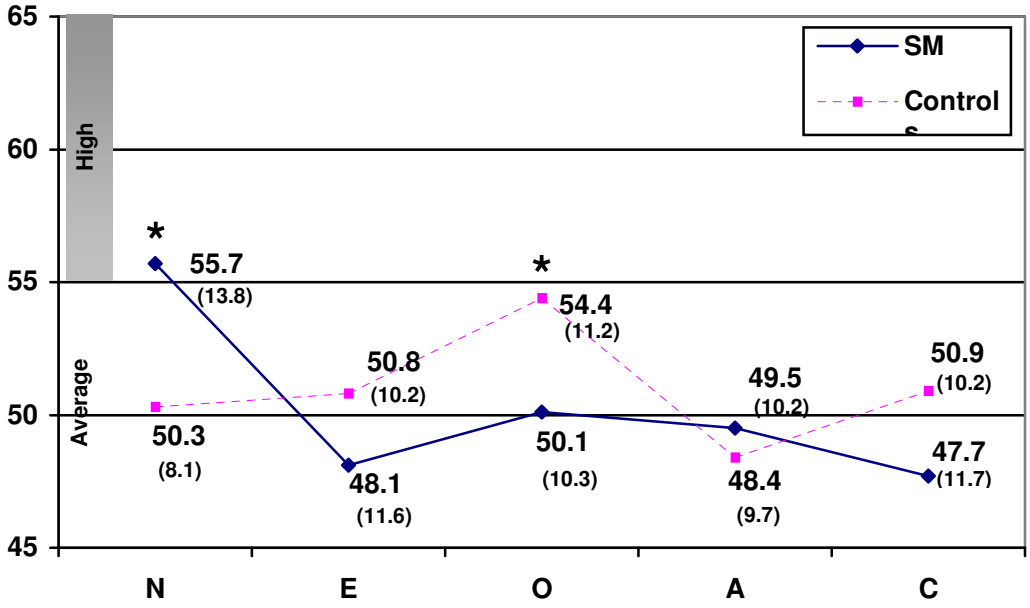

FIGURE 1. NEO-PI-R Domain Mean (T-scores) and Standard Deviations (in parentheses) across the SM and Control Groups.

Table 3. Neuroticism and Openness to Experience Facets across SM and Control groups

	SM		Controls			
	X	SD	X	SD	F	p
Neuroticism Facets						
Anxiety (N1)	**53.8**	**11.6**	**50.1**	**9.4**	**4.13**	**.04**
Angry Hostility (N2)	53.7	12.7	50.6	9.1	2.63	.11
Depression (N3)	**53**	**12.4**	**49**	**8.5**	**4.49**	**.04**
Self-Consciousness (N4)	**52.9**	**13**	**46**	**9.8**	**11.8**	**.001**
Impulsiveness (N5)	51.6	11.9	49.6	10.8	1.14	.29
Vulnerability (N6)	**61.5**	**12.3**	**56.9**	**7.8**	**6.15**	**.01**
Openness Facets						
Fantasy (O1)	52.9	10.2	53	11	.003	.95
Aesthetics (O2)	**47.3**	**10.3**	**52.6**	**11.4**	**9.03**	**.003**
Feelings (O3)	51.3	9.9	52.8	9.3	.89	.35
Actions (O4)	48.2	11.7	49.9	9.4	.89	.35
Ideas (O5)	**51**	**10.5**	**55**	**10.6**	**5.21**	**.02**
Values (O6)	49.7	11.1	52.8	9.3	3.10	.08

Acknowledgements: Many thanks to our interviewers and interview schedulers, Kelly Bailey MS, Bonnie Bethel MA, Laura Campbell-Sills PhD, Adrienne Means Christensen PhD, Shadha Hami MS, Teresa Marcotte BA, Jack Maser PhD, Sonya Norman PhD, Ryan Pepin BA. Also many thanks to the Selective Mutism Group-Child Anxiety Network and all the families who participated in this study.

REFERENCES

American Psychiatric Association (1994), *Diagnostic and Statistical Manual of Mental Disorders, 4th ed.* Washington, DC: American Psychiatric Association

Anstendig KD (1999), Is selective mutism an anxiety disorder? Rethinking its DSM-IV classification. *J Anxiety Disord* 13:417-434

Beidel DC, Turner SM, Young B, Paulson A (2005), Social effectiveness therapy for children: three-year follow-up. *J Consult Clin Psychol* 73:721-725

Bergman RL, Keller M, Wood J, Piacentini J, McCraken J (2001), Selective mutism questionnaire (SMQ): development and findings. Poster session presented at the meeting of the American Academy of Child and Adolescent Psychiatry, Honolulu, October

Bergman RL, Piacentini J, McCracken JT (2002), Prevalence and description of selective mutism in a school-based sample. *J Am Acad Child Adolesc Psychiatry* 41:938-946

Black B, Uhde TW (1995), Psychiatric characteristics of children with selective mutism: a pilot study. *J Am Acad Child Adolesc Psychiatry* 34:847-856

Brown JB, Lloyd H (1975), A controlled study of children not speaking at school. *J Association of Workers for Maladjusted Children* 3:49-63

Cohan SL, Price JM, Stein MB (2006), Suffering in silence: why a developmental psychopathology perspective on selective mutism is needed. *J Dev Behav Pediatr* 27:341-55.

Costa PT, McCrae RR (1988), From catalog to classification: Murray's needs and the five factor model. *J of Personality and Social Psychology*, 55: 258-265.

Costa PT, McCrae RR (1992), Revised NEO Personality Inventory (NEO-PI-R)and the NEO Five-Factor Inventory (NEO-FFI) professional manual. Odessa, FL: Psychological Assessment Resources.

Costa PT, McCrae RR, & Dye DA (1991), Facet scale for agreeableness and conscientiousness: A revision of the NEO Personality Inventory. *Personality and Individual Differences*, 12: 887-898

Cunningham CE, McHolm A, Boyle MH (2006), Social phobia, anxiety, oppositional behavior, social skills, and self-concept in children with specific selective mutism, generalized selective mutism, and community controls. *Eur Child Adolesc Psychiatry* 15:245-55

Elizur Y, Perednik R (2003), Prevalence and description of selective mutism in immigrant and native families: a controlled study. *J Am Acad Child Adolesc Psychiatry* 42:1451-9

Dummit ES, Klein RG, Tancer NK, Asche B (1997), Systematic assessment of 50 children with selective mutism. *J Am Acad Child Adolesc Psychiatry* 36:653-660

Eley TC, Bolton D, O'Connor TG, Perrin S, Smith P, Plomin R (2003), A twin study of anxiety-related behaviours in pre-school children. *J Child Psychol Psychiatry* 44:945-960

First MB, Spitzer RL, Gibbon M, Williams JBW et al., (1995), The structured clinical interview for DSM-III-R personality disorders (SCID II): II. Multi-site test retest reliability study. *J of Personality Disorders*, 9:92-104.

First MB, Spitzer RL, Gibbon M, Williams JBW (1997), User's guide for the Structured Clinical Interview for the DSM-IV Axis I Disorders-Clinician Version *(*SCID-CV*)*. Washington, DC, American Psychiatric Press

First MB, Gibbon M, Spitzer RL, Williams JBW, Benjamin LS (1997), Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II*)*. Washington, DC, American Psychiatric Press

Holt CS, Heimberg RG, Hope DA (1992), Avoidant personality disorder and the generalized subtype of social phobia. *J Abnorm Psychol* 101:318-325

Kendall PC, Flannery-Schroeder E, Panichelli-Mindel SM, Southam-Gerow M, Henin A, Warman M (1997), Therapy for youths with anxiety disorders: a second randomized clinical trial. *J Consult Clin Psychol* 65:366-380

Kristensen H, Torgersen S (2001), MCMI-II personality traits and symptom traits in parents of children with selective mutism: a case-control study. *J Abnorm Psychol* 110:648-652

Kristensen H, Torgersen S (2002), A case-control study of EAS child and parental temperaments in selectively mute children with and without a co-morbid communication disorder. *Nord J Psychiatry* 56:347-353

Kumpulainen K, Räsänen E, Raaska H, Somppi V (1998), Selective mutism among second-graders in elementary school. *Eur Child Adolesc Psychiatry* 7:24-29

Mannuzza S, Schneier FR, Chapman TF, Liebowitz MR, Klein DF, Fryer AJ (1995), Generalized social phobia: Reliability and validity. *Arch Gen Psychiatry* 52:230-237

Millon T (1987), *Manual for the MCMI-II (2nd ed*). Minneapolis, MN: National Computer system

Ralevski E, Sanislow CA, Grilo CM, Skodol AE, Gunderson JG, Tracie Shea M, Yen S, Bender DS, Zanarini MC, McGlashan TH (2005), Avoidant personality disorder and social phobia: distinct enough to be separate disorders? *Acta Psychiatr Scand* 112:208-14

Renneberg B, Chambless DL, Dowdall DJ, Fauerbach JA, et al. (1992), The structured clinical interview for DSM-III—R, Axis II and the million clinical multiaxial inventory: A concurrent validity study of personality disorders among anxious outpatients. *J Pers Disord* 6:117-124

Schinka JA, Busch RM, Robichaux-Keene N (2004), A meta-analysis of the association between the serotonin transporter gene polymorphism (5-HTTLPR) and trait anxiety. *Mol Psychiatry* 9:197-202

Schwartz CE, Snidman N, Kagan J. (1999). Adolescent social anxiety as an outcome of inhibited temperament in childhood. J Am Acad Child Adolesc Psychiatry, 38, 1008-1015.

Sen S, Burmeister M, Ghosh D (2004), Meta-analysis of the association between a serotonin transporter promoter polymorphism (5-HTTLPR) and anxiety-related personality traits. *Am J Med Genet* 127:85-89

Silverman W, Albano AM (1996), Anxiety Disorders Interview Schedule for Children using DSM-IV, Psychological Corporation, San Antonio, TX

Silverman W, Saavedra LM, Pina AA (2001), Test-retest reliability of anxiety symptoms and diagnoses with the Anxiety Disorders Interview Schedule for DSM-IV: child and parent versions. *J Am Acad Child Adolesc Psychiatry*, 40:937-44.

Stein MB, Chartier MJ, Hazen AL, Kozak MV, Tancer ME, Lander S, Furer P, Chubaty D Walker JR (1998), A direct-interview family study of generalized social phobia. *Am J Psychiatry* 155:90-97

Stein MB, Bienvenu OJ (2004), Diagnostic classification of anxiety disorders: DSM-V and beyond. In: Charney DS, Nestler EJ (eds). *The Neurobiology of Mental Illness*, 2nd edn. Oxford University Press: New York, NY. pp 525-534

Steinhausen HC, Juzi C (1996), Elective mutism: an analysis of 100 cases. *J Am Acad Child Adolesc Psychiatry* 35:606-614

Steinhausen HC, Adamek R (1997), The family history of children with elective mutism: a research report. *Eur Child Adolesc Psychiatry* 6:107-111

Steinhausen HC, Wachter M, Laimbock K, Metzke CW (2006), A long-term outcome study of selective mutism in childhood. *J Child Psychol Psychiatry* 47:751-756

Wagner KD, Berard R, Stein MB, Wetherhold E, Carpenter DJ, Perera P, Gee M, Davy K, Machin A (2004), A multicenter, randomized, double-blind, placebo-controlled trial of paroxetine in children and adolescents with social anxiety disorder. *Arch Gen Psychiatry* 61:1153-1162

Williams JB, Gibbon M, First MB, Spitzer RL, et al. (1992), The Structured Clinical Interview for DSM-III—R (SCID): II. Multisite testetest reliability. *Arch Gen Psychiatry* 49:630-636

Yeganeh R, Beidel DC, Turner SM, Pina AA, Silverman WK (2003), Clinical distinctions between selective mutism and social phobia: an investigation of childhood psychopathology. *J Am Acad Child Adolesc Psychiatry* 42:1069-1075

Yeganeh R, Beidel DC, Turner SM (2006), Selective mutism: more than social anxiety? *Depress Anxiety* 23:117-23

Zanarini MC, Skodol AE, Bender D, Dolan R, Sanislow C, Schaefer E, Morey LC, Grilo CM, Shae MT, McGlashan TH, Gunderson JG (2000), The collaborative longitudinal personality disorders study: Reliability of axis I and II diagnoses. *J Pers Disord* 14:291-299