Genetic and Environmental Covariation Between Autistic Traits and Behavioral Problems

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Our objective was to examine the overlap between autistic traits and other behavioral problems in a general population sample, and explore the extent to which this overlap is due to genetic or environmental factors. Youth Self Report (YSR) data were collected in a general population sample of 424 twin pairs at 18 years of age, and their nontwin siblings. In 197 of these twin families, self-report ratings on the Autism-spectrum Quotient (AQ) were collected. Stepwise backward regression analyses revealed that of all 8 YSR syndrome scales, the Withdrawn Behavior (WB) and Social Problems (SOC) scale were the most important predictors of AQ scores, and together with sex, explained 23% of the variance in AQ scores. Genetic structural equation modeling showed that the overlap between AQ and WB and SOC was mainly due to genetic effects. About half of the genetic variance in AQ scores was specific to the AQ, with the remaining half shared with genetic variance in WB and SOC. Endorsement of autistic traits in a general population sample is associated with social and withdrawn behavioral problems and these problems partly share a common genetic etiology with autistic traits. However, most of the variance in AQ scores remains unexplained by YSR scores, and half of the genetic variance in AQ is unshared with WB and SOC. These results indicate that autistic traits have specific characteristics that are substantially genetically independent from other common but related behavioral domains such as social problems and withdrawn behavior.

Autism is one of the most heritable disorders in psychopathology, with heritability estimates exceeding 90% (Freitag, 2007). Twin and family studies have shown that having an autistic relative not only increases the risk for clinical autism, but also affects the expression of milder, but qualitatively similar autistic traits, such as difficulties with social interaction and communication, and a preference for routines (Bailey et al., 1998; Bolton et al., 1994; Landa et al., 1992; Piven et al., 1997). These findings suggest that the same genetic variants that affect the risk for autism may influence the expression of a 'broader autism phenotype' in relatives of autistic probands (Piven et al., 1997; Spiker et al., 2002). Rather than treating autism as a distinct disorder, recent twin and family studies incorporated a dimensional approach to study the etiology of autistic traits and showed that genetic effects also explain a substantial proportion of the variance in autistic traits in the general population (Constantino & Todd, 2000; Constantino & Todd, 2003; Hoekstra et al., 2007b; Ronald et al., 2005; Ronald et al., 2006).

Individuals with a clinical diagnosis for autism frequently show additional behavioral problems. Affective disorders are common both in autistic individuals with intellectual disability (Lainhart & Folstein, 1994; Matson & Nebel-Schwalm, 2006) and in high functioning individuals (Howlin, 2000). A high prevalence of specific phobia, obsessive compulsive disorder, attention-deficit hyperactivity disorder (ADHD; Leyfer et al., 2006) and ADHD-like symptoms, such as inattention, hyperactivity and impulsivity (Goldstein & Schwebach, 2004; Sturm et al., 2004; Yoshida & Uchiyama, 2004) is observed in children with autism or other pervasive developmental disorders. Family studies suggest that relatives of autistic individuals also have increased risk for psychopathology other than autism. Elevated rates of major depression, anxiety, social phobia, and obsessive compulsive disorders are reported in relatives of children with autism (Bolton et al., 1998; Micali et al., 2004; Piven & Palmer, 1999; Smalley et al., 1995). However, it remains unclear whether these elevated risks can be attributed to the burden of caring

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for an autistic child, or are due to genetic risk factors associated with the risk for autism.

The dimensional approach to autistic traits enables the study of the etiology of the overlap between autistic symptoms and other domains of psychopathology in general population samples. One previous study examined the overlap between deficits in social reciprocal behavior and other behavioral problems in 7- to 15-year-old male twins (Constantino et al., 2003), and found significant covariation between autistic traits and attention problems and social problems. So far, no studies have examined the overlap between autistic traits and other behavioral problems in late adolescence, and none have included females and nontwin siblings. The current study wishes to address these issues and aims to examine the genetic and environmental covariation between quantitative autistic traits, measured with the Autism-Spectrum Quotient (AQ; Baron-Cohen et al., 2001; Hoekstra et al., 2007b), and behavioral problems (indexed by Youth Self Report [YSR] scores; Achenbach & Rescorla, 2001; Verhulst et al., 1997), in a general population sample of 18-year-old twins and their siblings.

Materials and methods

Participants and Procedures

All participants were contacted via the Netherlands Twin Register (NTR), kept by the Department of Biological Psychology at the VU University in Amsterdam (Bartels et al., 2007; Boomsma et al., 2006). From 1986 onwards, the NTR has recruited families with multiples a few weeks or months after birth. When the twins are 1, 2, 3, 5, 7, 10, and 12 years old, the parents are asked to provide information about the physical and behavioral development of their twins via mailed surveys. At age 14, 16, and 18 parents are asked for permission to contact the twins and additional siblings in the family. These offspring are invited to register with the NTR and are asked to fill out self-report questionnaires. The current study includes data from twins born from 1986 to 1988 (mean age = 18.44, *SD* = .39), and their siblings (mean age = 19.00, *SD* = 4.19). Youth Self Report data were collected in 424 families. These included 65 monozygotic male pairs (MZM), 60 pairs were dizygotic males (DZM), 106 were monozygotic females (MZF), 88 were dizygotic females (DZF), and 105 were dizygotic twin pairs of opposite sex (DOS). Data for an additional sibling were available for approximately half of the families (206 siblings, of which 96 were male, 110 were female). Zygosity of the same sex twin pairs was determined using DNA analysis (178 pairs), blood group polymorphisms (48 pairs), or longitudinally collected questionnaire items (Rietveld et al., 2000; 93 pairs). In a subset of this sample (197 families), AQ data were collected. These twin families participated in a longitudinal study into the development of cognition and problem behavior, and were selected based on age and zygosity of the twins, and their place of residence. The subset of families encompassed 37 MZM, 34 DZM, 47 MZF, 40 DZF, and 39 DOS twin pairs, and 104 siblings (52 brothers and 52 sisters). All subjects filled out the YSR at home. Most twin families from the subset (n = 186) filled out the AQ at the VU University as part of an extensive test protocol. Eleven families filled out the AQ at home (AQ scores in these families were not different from the participants who visited the VU University). Complete data on both the YSR and the AQ were available for 452 subjects (all from the subset of 197 families). The YSR scores in this subset were similar to the YSR scores of subjects who did not fill out the AQ (multivariate analysis of variance F (942, 8) = 1.867, p = .06; univariate analyses of each YSR scale yields p values between .18 and .96). Written informed consent was obtained from all subjects who were 18 years or older and from the parents for the underage participants. The study was approved by the Central Committee on Research

Table 1

Multiple Linear Regression Results: Associations Between AQ Scores and YSR Syndrome Scales

Test	Model	–2LL	*df*	Versus	χ^2	*p*	b^2*Var_{pred}
1	Model including all YSR scales + sex	3137.92	421				
2	Drop ATT	3137.93	422	1	.01	.94	.00
3	Drop ANX	3137.96	423	2	.04	.85	.02
4	Drop AGG	3138.05	424	3	.09	.76	.03
5	Drop SOM	3139.59	425	4	1.54	.21	.40
6	Drop RB	3141.75	426	5	2.15	.14	.49
7	Drop THO	3146.84	427	6	5.09	.024	1.09
8	Drop SOC	3162.53	428	7	15.69	< .001	4.09
9	Drop WB	3237.76	429	8	75.23	< .001	17.02

Note: –2LL = –2 log likelihood; vs. = compared to model; b^2*Var_{pred} = explained variance in AQ due to the predictor; ATT = YSR syndrome scale Attention Problems; ANX = Anxious/Depressed; AGG = Aggressive Behavior; SOM = Somatic Complaints; RB = Rule-Breaking Behavior; THO = Thought Problems; SOC = Social Problems; WB = Withdrawn Behavior.

Table 2

Sample Sizes, Means, and Standard Deviations for Scores on the AQ, YSR Withdrawn Behavior (WB), and YSR Social Problems (SOC)

	N	Mean	*SD*
AQ male	218	103.99	10.48
AQ female	252	100.69	10.61
AQ all[a]	470	102.22	10.67
WB male	397	2.36	2.15
WB female	569	2.67	2.17
WB all[bc]	966	2.54	2.16
SOC male	398	1.91	1.74
SOC female	570	1.88	1.72
SOC all	968	1.90	1.72

Note: [a]sex effect significant $p < .01$; [b]sex effect significant $p < .05$; [c]age effect significant $p < .01$

Involving Human Subjects and the institutional review board of the VU University Amsterdam.

Measures

The AQ is a self-administered questionnaire developed to quantify autistic traits in individuals with normal intelligence (Baron-Cohen et al., 2001). The AQ consists of 50 items, selected from the domains of the 'triad of impairment' in autism (impaired social skills, impaired communication, and restricted interests), and from demonstrated areas of cognitive abnormality in autism (e.g. lack of imagination and great attention to detail). Participants rate to what extent they agree or disagree with the statements on a 4-point Likert scale, with answer categories: 1 = definitely agree, 2 = slightly agree, 3 = slightly disagree, and 4 = definitely disagree. For items in which an the 'agree' response is characteristic for autism (24 out of the 50 items), the scoring was reversed. AQ scores were calculated as the sum of the item scores, with a minimum AQ score (50) indicating no autistic traits, and a maximum score (200) indicating full endorsement of all autistic traits. High self-report AQ scores are specific to subjects with an autism spectrum diagnosis, both in the original English version of the test (Baron-Cohen et al., 2001) and in the Dutch translation (Hoekstra et al., 2007a). The Dutch translation of the AQ shows good test–retest reliability ($r = .78$ in a group of 75 general population subjects with a 1–6 month time interval) and internal consistency (Cronbach's alpha = .79 in a previous study [Hoekstra et al., 2007a]; Cronbach's alpha = .77 in the current sample). Complete AQ's were available for 470 subjects. If five or fewer answers were missing, the AQ score was corrected for the number of missing items by adding the mean item score times the number of missing items to the total AQ score (one missing answer $n = 22$, two missing answers $n = 3$).

The YSR is a self-report questionnaire designed to assess emotional and behavioral problems in 11- to 18-year-old children (Achenbach & Rescorla, 2001; Verhulst et al., 1997). The adolescent is asked about the occurrence of problem behavior in the preceding 6 months and to rate the behavior on a 3-point scale (0 if the problem item is not true, 1 if the item is somewhat or sometimes true, and 2 if it is very true or often true). The YSR generates scores for eight syndrome scales: Anxious/Depressed, Withdrawn Behavior, Somatic Complaints, Social Problems, Thought Problems, Attention Problems, Aggressive Behavior, and Rule-Breaking Behavior. Similar to the original version (Achenbach & Rescorla, 2001) the Dutch version of the YSR shows good reliability and validity (Verhulst et al., 1997).

Data analysis

To examine the extent to which the eight YSR syndrome scales predicted AQ scores, multiple regression analysis was conducted. Since the inclusion of twin and twin-sibling data violated the assumption of independent observations, the regression analysis was carried out using structural equation modelling in Mx (Neale et al., 2006), by allowing the data from family members to be correlated. The YSR syndrome scales were included in the regression analysis as so-called definition variables (Neale et al., 2006). As previous studies showed significant sex differences in mean AQ scores (Baron-Cohen et al., 2001; Hoekstra et al., 2007b; Wakabayashi et al., 2006), sex was added as an additional predictor in the analysis. A procedure similar to the backward stepwise regression method was employed. Firstly, all predictors were entered in the regression equation. Next, the predictor explaining the least variance in AQ scores (as reflected in the squared product of the regression coefficient multiplied by the variance of the predictor [i.e., $b^2 * Var_{pred)}$]) was dropped from the model. This procedure was repeated until the significance of each YSR syndrome scale was tested. Those YSR scales that were significant predictors of AQ scores were included in the multivariate genetic analyses.

Phenotypic correlations among the scores on the AQ and YSR scales were estimated in a saturated model in Mx. The saturated model was also used to estimate twin and twin-sibling correlations for each variable, and to estimate the twin and twin-sibling cross-correlations (e.g., the correlation between AQ score of the oldest of the twin and YSR syndrome score of the youngest of the twin). All data were used, including YSR data of subjects without information on the AQ, and including data from families for whom information of one of the twins was missing, or for whom no sibling data were available. Genetic model fitting was performed in Mx using standard methods (Neale & Cardon, 1992).

Table 3

Phenotypic Correlation in all Participants, and Twin and Twin-Sibling Correlations and Cross-Correlations for AQ, YSR Withdrawn Behavior and Social Problems in all Zygosity Groups and Twin-Sibling Pairs

	AQ	WB	SOC	AQ	WB	SOC
			Phenotypic			
AQ	—	.45	.40			
WB		—	.53			
SOC			—			
			MZ males			**DZ males**
AQ	.41/.60[a]	.31	.41	.45/.37[b]	.09	.17
WB	.23	.59/.47[a]	.26	.23	.27/.28[b]	.16
SOC	.26	.38	.52/.38[a]	.16	.17	.31/.30[b]
	MZ females			**DZ females**		
			Male twin-sibling		**Opposite sex twin-sibling**	
AQ	.42/.10[c]	.18	.16	.36/.25[d]	.07	.11
WB	.21	10/.17[c]	.13	.16	.29/.09[d]	-.04
SOC	.31	.18	.19/.05[c]	.14	.25	.24/.00[d]
	Female twin-sibling			**DOS**		
			All DZ twins		**All 1st degree relatives**	
AQ	.49/.39[e]	.16	.17	.33	.17	.19
WB	.27	.55/.28[e]	.20		.17	.11
SOC	.33	.34	.46/.29[e]			.14
	All MZ twins					

Note: MZM = monozygotic males; DZM = dizygotic males; MZF = monozygotic females; DZF = dizygotic females; DOS = dizygotic twins of opposite sex
[a]First figure correlation MZF, second figure correlation MZM; [b]correlation DZF/DZM; [c]correlation female twin-sibling pairs/male twin-sibling pairs; [d]correlation DOS/opposite sex twin-sibling pairs; [e]correlation all MZ/all DZ

MZM, DZM, Male twin-sibling, opposite sex twin-sibling, all DZ, all 1st degree relatives above diagonals; MZF, DZF, female twin-sibling, DOS, and all MZ below diagonals.

Results

The AQ scores were continuously distributed. Males obtained significantly higher scores than females (χ_1^2 = 19.71, $p < .001$). To check for possible effects of influential data points on the multiple regression analysis, we created scatter plots of all AQ scores against the YSR syndrome scores and detected 6 outliers. Running the multiple regression analysis without these outliers revealed that the YSR syndrome scales Withdrawn Behavior (WB) and Social Problems (SOC) were both significant predictors of AQ scores (respectively χ_1^2 = 75.23, $p < .001$ and χ_1^2 = 15.69, $p < .001$; see Table 1). To ensure that we would select YSR syndrome scales with a robust predictive effect, explaining a meaningful proportion of the variance in AQ scores, we used a conservative p value of $p < .01$. When the influential data points were included in the regression analysis, similar results were found for the effect of WB (χ_1^2 = 78.21, $p < .001$), and SOC (χ_1^2 = 17.26, $p < .001$), but now YSR Rule-Breaking Behavior and YSR Thought Problems also approached significance (respectively χ_1^2 = 6.24, p = .013 and χ_1^2 = 5.54, p = .019). WB and SOC were found to explain a significant proportion of the variance in AQ scores in both analyses. The regression model, including these two YSR scales and sex as predictors, explained 23% of the observed variance in AQ scores (i.e., R^2 = 23%), of which 21% was due to the YSR scores. Stein's adjusted R^2 for the regression model (Stevens, 1996) was 21.7%. This value is very similar to the observed value of R^2, indicating that the cross-validity of this model is good.

The descriptive statistics of AQ scores, WB and SOC are provided in Table 2. Apart from the sex effect on mean AQ scores, a significant age (χ_1^2 = 7.59, p = .006) and sex effect (χ_1^2 = 4.87, p = .027) was found for mean WB scores, with higher scores in females than males, and increasing WB scores with age. In subsequent modeling, these effects were retained in the model. The variances (χ_3^2 =1.20, p = .75) and within person covariance (χ_3^2 = 1.52, p = .68) could be set equal in male and female twins and across all twins and siblings (respectively χ_3^2 = 1.15, p = .77 and χ_3^2 = 5.20, p = .16), indicating that the phenotypic correlations between the measures are similar in males and females and in twins and their siblings. The phenotypic correlations between AQ scores and WB and SOC scores (first row of table 3) are moderate, with WB explaining 20% ($.45^2$) of the variance in AQ scores, and SOC explaining 16% of the variance in AQ scores. The two YSR syndrome scales are substantially correlated with each other (r = .53).

On the diagonal of Table 3, the twin correlations are presented for all zygosity groups and twin-sibling

combinations. Taken together, the correlations in MZ twins are higher than in DZ twins and twin-siblings, indicating genetic influences. However, MZF and DZF correlations for AQ are similar, and the MZ twin correlations for AQ scores are not twice as high as the correlations in first-degree relatives, suggesting that shared environmental influences could also play a role. The MZ twin cross-correlations (off-diagonal of Table 3) are higher than the cross-correlations in first-degree relatives, suggesting that the overlap between AQ, WB, and SOC is influenced by genetic factors. The MZ twin cross-correlations between AQ and the two YSR scores are not twice as high as the cross-correlations in first-degree relatives, suggesting that shared environmental influences may explain part of the phenotypic overlap between these measures.

Table 4 presents the model fitting statistics for the full Cholesky model, including both additive genetic, shared environmental, and nonshared environmental influences (referred to as the ACE model), and for the more parsimonious submodels. Constraining the parameters that represent the effect of A, C, and E to be the same in males and females did not significantly worsen the model fit (model 2, $\chi_{18}^2 = 18.44$, $p = .43$), suggesting that the relative effects of these components are the same across the sexes. The shared environmental component could be dropped from the model without a significant deterioration of the fit (models 3 to 5). The genetic effects, however, were of significant importance for all measures (models 6 to 8). The best fitting most parsimonious model was an AE model without sex differences in the relative contribution of the variance components (model 5).

The contributions of additive genetic and nonshared environmental effects to the variance in AQ, WB, and SOC are shown on the diagonal of Table 5. Genetic effects explain 53% of the individual differences in AQ scores; the remaining variance is accounted for by nonshared environmental effects. Genetic influences explain about half of the variance in WB, and account for 41% of the variance in SOC. The overlap between AQ scores and scores on the YSR scales (off-diagonal of Table 5) is largely due to genetic effects, explaining 64% of the covariance between AQ and WB, and 82% of the covariance between AQ and SOC. The remaining covariance between the measures is accounted for by nonshared environmental influences. Examination of the individual parameter estimates showed that 51% of the genetic variance in AQ is shared with the variance in YSR scores, the remaining genetic variance is specific to the AQ. Only 11% of the nonshared environmental variance is shared with variance in YSR scores, implying that the greater part (89%) of this variance is specific to the AQ.

Discussion

This study reported on the overlap between autistic traits and behavioral problems in a general population sample in late adolescence. The YSR syndrome scales WB and SOC were found to be significant predictors of endorsement of autistic traits, and could, together with sex as additional variable, explain 23% of the variance in AQ scores. Quantitative genetic analyses showed that individual differences in AQ scores underlie substantial genetic influence. Moreover, the overlap between AQ, WB, and SOC scores was mainly accounted for by genetic effects. Approximately half of the genetic variance in AQ scores was shared with variance in the YSR scales; the nonshared environmental variance in AQ was largely specific to the AQ.

Previous general population studies reported moderate to high genetic influences on individual differences in autistic traits (Constantino & Todd, 2000; Constantino & Todd, 2003; Ronald et al., 2005; Ronald et al., 2006), and the results of the current study are in line with these findings. In a previous report of the same study sample as reported on here, we reported a univariate heritability of the AQ of 57% (Hoekstra et al., 2007b). Shared environmental influences were insignificant, although the power to detect these influences was limited (Posthuma & Boomsma, 2000). The current report incorporated multivariate analysis, which increases the power to

Table 4

Model Fitting Results for Multivariate Analyses of AQ Scores, YSR Withdrawn Behavior, and Social Problems

Test	Model	–2LL	*df*	vs.	χ^2	*p*
1	ACE sex differences	11005.799	2362			
2	ACE no sex differences	11024.243	2380	1	18.444	.427
3	ACE AQ, ACE WB, AE SOC, no sex differences	11024.669	2383	2	.426	.935
4	ACE AQ, AE WB, AE SOC, no sex differences	11025.041	2385	3	.372	.830
5	**AE no sex differences**	11025.765	2386	4	.724	.394
6	CE AQ, ACE WB, ACE SOC, no sex differences	11032.265	2381	2	8.022	.004
7	ACE AQ, CE WB, ACE SOC, no sex differences	11045.508	2382	2	21.265	< .001
8	ACE AQ, ACE WB, CE SOC, no sex differences	11041.222	2383	2	16.979	< .001

Note: –2LL = –2 log likelihood; vs. = compared to model; A = additive genetic influences; C = shared environmental influences; E = nonshared environmental influences; WB = YSR Withdrawn Behavior; SOC = YSR Social Problems. Preferred model in bold.

Table 5

Standardized* and Unstandardized Parameter Estimates for Additive Genetic (A) and Nonshared Environmental (E) Effects to the Variance and Covariance in AQ Scores and in YSR Withdrawn Behavior (WB) and Social Problems (SOC) Scores

	A			E		
	WB	SOC	AQ	WB	SOC	AQ
WB	1.53 (1.31–1.72)	0.78 (0.57–0.98)	4.38 (2.60–6.11)	1.54 (1.39–1.70)	0.51 (0.34–0.69)	2.40 (1.11–3.73)
	.50 (.38–.60)			**.50 (.40-.62)**		
SOC		0.78 (0.57–0.96)	3.35 (1.13–5.67)		1.23 (1.12–1.34)	0.11 (–1.06–1.30)
	.60 (.44–.75)	**.41 (.29–.52)**		**.40 (.25–.56)**	**.59 (.48–.71)**	
AQ			5.44 (3.02–6.93)			6.92 (6.09-7.86)
	.64 (.41–.84)	**.82 (.58–1.03)**	**.53 (.39–.64)**	**.36 (.16–.59)**	**.18 (–.03–.42)**	**.47 (.36–.61)**

Note: *Standardized in bold and below diagonal

95% confidence intervals in parentheses.

detect genetic and shared environmental influences (Schmitz et al., 1998), but the results were virtually the same: substantial genetic influences and no significant shared environmental effects. The current results strengthen the notion that variance in autistic traits has a substantial genetic component in the general population. However, the MZF and DZF twin correlations were similar in our sample, and future studies including larger sample sizes should elucidate whether shared environmental influences may play a more important role in females than in males.

The finding that the YSR scales Withdrawn Behavior and Social Problems were the best predictors of AQ scores is not surprising. The WB syndrome scale captures shy, introvert, and withdrawn behavior, and includes items such as 'Prefer to be alone', 'Secretive', and 'Withdrawn'. The SOC scale contains items such as 'Too dependent', 'Teased a lot', and 'Other boys/girls don't like me'. These items all hint at difficulties in social interactions, one of the main impairments in autism, and similar items (such as 'I prefer to do things with others rather than on my own' and 'I find it hard to make new friends') are also included in the AQ. However, the WB scale also includes items ('Lacks energy' and 'Sad') more indicative of depression. Examination of the raw item scores revealed that these items loaded as strongly on the AQ scores as the other items of the WB scale. The syndrome scales WB and SOC are substantially correlated with the Anxious/Depressed scale of the YSR, both in our sample (r = .66 for WB, and r = .49 for SOC), and in the general population sample from which the Dutch YSR norm scores were derived (r between .46 and.62; Verhulst et al., 1997). The AQ scores in our sample correlated moderately with the Anxious/Depressed scale (r = .31), but this association was not significant once the association with WB and SOC was taken into account. Altogether, these results suggest that social problems related to anxiety and depression may be common in people highly endorsed on autistic traits.

One previous study in 7- to 15-year-old male twins (Constantino et al., 2003) examined the genetic and environmental influences on covariation between scores on the Social Responsiveness Scale (SRS) and syndrome scores of the Child Behavior Checklist (CBCL). In this study, multiple regression analysis indicated that the CBCL syndrome scales Attention Problems and Social Problems were significant predictors of SRS scores. The WB scale was the third most important predictor, but failed to be significant when the Bonferroni correction for multiple testing was applied. In contrast to our study, the Attention Problems scale was found to account for a significant proportion of the variance. This discrepancy could be due to a variety of factors. First, the study by Constantino and colleagues was performed in 7- to 15-year-old males. The association between autistic-like behaviors and attention problems may be stronger in this younger age group. Furthermore, differences in the questionnaires used to assess autistic traits (parent-report SRS vs. self-report AQ) may underlie the different findings. The SRS inquires about problem behaviors directly, while the AQ assesses personal preferences and habits, rather than a judgment of behavior.

In a clinical study evaluating CBCL scores in autistic children, the highest relative scores were found on Attention Problems, Social Problems, and Thought Problems (Bolte et al., 1999). Another study compared parent-reported CBCL scores in children with problems classified as pervasive developmental disorder not otherwise specified (PDD-NOS), children classified as ADHD, and a group of normal functioning controls (Luteijn et al., 2000). Both the PDD-NOS and the ADHD group showed elevated scores on the Attention Problems scale, these groups did not differ from each other. High scores on the WB and SOC scale were specific to the PDD-NOS group. Our study suggests that in the general population, the observed overlap between autistic traits and withdrawn and social behavioral problems is primarily of genetic origin.

This study has some limitations. The sample size for which both data on the AQ and the YSR were

available was relatively small. Therefore, the power to detect sex differences in the genetic and environmental architecture underlying the association between the AQ and the YSR scores was limited. Moreover, the power to detect shared environmental influences and possible sex differences in the relative importance of the variance components was restricted. Future studies should include larger sample sizes to further examine this. We relied on self-report measures of problem behaviors. Previous studies have shown that autistic traits and behavioral problems can reliably be assessed using self-report AQ (Baron-Cohen et al., 2001; Hoekstra et al., 2007a) and YSR data (Verhulst et al., 1997). However, different raters may provide different perspectives and give important additional information (Bartels et al., 2003; Ronald et al., 2005; Van der Ende & Verhulst, 2005). Therefore, it would be valuable to collect multiple informant data in future studies.

Our study suggests that the vulnerability for general social problems is genetically related to endorsement of autistic traits. About half of the genetic variance in AQ scores was shared with genetic variance in WB and SOC scores, indicating that these problems partly share a common genetic etiology. However, as half of the genetic variance is specific to the AQ, and thus unshared with other behavioral problems, these findings also indicate that autistic traits have specific characteristics that are substantially genetically independent from other behavioral problems.

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