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Using self-report to identify the broad phenotype in parents of children with autistic spectrum disorders: a study using the Autism-Spectrum Quotient

Dorothy V.M. Bishop,[1,2] Murray Maybery,[2] Alana Maley,[2] Dana Wong,[2] Wayne Hill,[2] and Joachim Hallmayer[2,3]

[1]Oxford University, UK; [2]University of Western Australia; [3]Stanford University, USA

Background: The concept of the 'broad phenotype' of autism refers to the finding that relatives of people with autism often have mild forms of autistic-like characteristics, such as social and communicative difficulties. This study used the Autism Spectrum Quotient (AQ), a questionnaire devised to assess features of the broad phenotype in adults, with parents of people with autism, to see whether they would be more likely to obtain extreme scores than a control group. **Methods:** The AQ was administered to parents of 69 people with an autism spectrum disorder and parents of 52 controls. **Results:** On two of the five subscales of the AQ, social skills and communication, parents of people with autism obtained higher scores than control parents. The other three scales, attention to detail, attention switching, and imagination, did not differentiate groups. The correlation between social skills and communication scales was .663. The scales can be combined to give an index of broad phenotype. **Conclusions:** The AQ appears to be sensitive to the broad phenotype, provided attention is restricted to the social skills and communication scales. **Keywords:** Autism, genetics, family, questionnaire, broad phenotype. **Abbreviations:** ADI-R: Autism Diagnostic Interview – Revised; AQ: Autism-Spectrum Quotient; DZ: dizygotic; MZ: monozygotic; PDDNOS: pervasive developmental disorder not otherwise specified; SCQ: Social Communication Questionnaire; WAFSASD: Western Australia Family Study of Autistic Spectrum Disorders.

For many years the notion of a genetic basis to autism was dismissed because the disorder did not appear to run in families. In a genetic disorder, we expect to find evidence of the same condition in parents and their children, and in affected cases and their siblings, yet in autism it is unusual to find families where more than one individual is affected. Indeed, people with autism are unlikely to have children, because their social impairments make it difficult to establish close relationships with others. Nevertheless, over the years there have been repeated suggestions that parents of children with autism may themselves show unusual traits reminiscent of autism, such as difficulties with social interaction and communication. In the past, there was a tendency to assume that abnormalities in parents were either direct causes of a child's autistic behaviour (e.g., the notion of the 'refrigerator parent' who is unable to engage emotionally with a child), or were reactions to the stress of rearing a child with a serious developmental disorder. However, over the last decade new evidence has emerged to indicate that associations between unusual characteristics in a parent and autism in a child are likely to be genetically mediated.

In a recent review, Rutter (2000) pointed out that, despite the low familiality, genes appear to play a major role in the risk for autism. This shows up most clearly in the study of identical (monozygotic or MZ) and nonidentical (dizygotic or DZ twins). If one MZ twin has autism, not only is the likelihood of autism in the co-twin greatly increased, but also the likelihood of other neurodevelopmental difficulties affecting language and social interaction (Folstein & Rutter, 1977; Bailey et al., 1995). Twin concordance was considerably lower in non-identical same sex twin pairs, though here too, there was evidence that some co-twins of affected children had neurodevelopmental difficulties that fell short of core autism. The twin studies thus led to the idea that the same genetic variants that put the child at risk for autism may also lead to a broad phenotype that can be seen in close relatives of people with autism.

The notion of a broad phenotype was given further impetus by family studies, in which rates of impairment were assessed in relatives of people with autism. In an early study, Wolff, Narayan, and Moyes (1988) rated parental personality characteristics from an interview, comparing 21 parents of autistic children with 21 control parents, and found an elevated rate of social gaucheness in the former group. Bolton et al. (1994) compared 99 families who had a child with autism with 36 families who had a child with Down syndrome. Parents and siblings were directly assessed. Although rates of core autism or other pervasive developmental disorder (PDD) were substantially higher than general population rates in the siblings of children with autism, in absolute

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terms they were relatively low (3% siblings with autism, a further 6% with PDD). However, a broad phenotype was seen in 20% of siblings of those with autism, compared with only 3% of siblings in the control group.

A better understanding of the broad phenotype in relatives of people with autism is essential if molecular genetic studies are to make progress in uncovering genes associated with risk for autism. As Rutter (2000) noted, 'It is crucial to consider the possibility that the genetic effects operate on components of autism, rather than the syndrome as such.' To make progress in this endeavour, we need to identify which traits characterise the broad phenotype, and find reliable measures of these to use with relatives. If we could achieve these goals, we would be able to use methods of genetic analysis that move away from treating autism as a categorical disorder, and regard it instead as a set of quantitative traits.

Several studies have now been conducted to compare parents of children with autism with control parents, and some differences have been found on psychometric tests. However, these have been rather inconsistent from one study to the next (Bailey, Palferman, Heavey, & Le Couteur, 1998). The clearest indicators of the broad phenotype in parents appear to emerge when family members are interviewed about their social and communicative histories (Fombonne, Bolton, Prior, Jordan, & Rutter, 1997; Piven, Palmer, Jacobi, Childress, & Arndt, 1997; Folstein et al., 1999; Szatmari et al., 2000), or when pragmatic aspects of communication are directly evaluated (Landa et al., 1992). Nevertheless, there is at present no 'gold standard' for identification of the broad phenotype, and it is unclear how these different approaches to assessment relate to one another, and whether one is more valid than another. Many of the features thought to characterise the broad phenotype are rather subtle, and may be difficult to rate reliably in a single assessment. One might hope that more direct assessment of underlying neuropsychological deficits will provide a more objective means of documenting the broad phenotype, but to date, although there have been reports of significant differences between parents of autistic children and control parents on tests measuring aspects of central coherence and executive function, no one measure emerges as a reliable indicator across a range of studies (see review by Bailey et al., 1998). It is noteworthy, too, that for high-functioning individuals with autism or Asperger syndrome, tests of social cognition, executive function or central coherence do not invariably reveal deficits in affected individuals themselves, and so could not be expected to act as infallible markers of the broad phenotype.

In this study we took a different approach to the assessment of the broad phenotype, considering how far a self-report measure might reveal autistic-like characteristics in parents of people with autism. We used the Autism-Spectrum Quotient (AQ), which was developed by Baron-Cohen and colleagues (Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001) as a brief, self-administered instrument for assessing the broader phenotype in adults of normal IQ. Compared with other methods of identifying the broad phenotype, the AQ has the advantage of being extremely quick and easy to administer, which means it has considerable potential for use in genetic studies that would benefit from a means of rapid phenotyping of large samples of individuals. However, in order for it to be valid in this context, it would be necessary to show that the AQ does identify the broad phenotype in a higher proportion of parents of individuals with autism than in other parents.

In a validation study of the AQ, Baron-Cohen et al. (2001) showed that it does discriminate high-functioning people with autism from unaffected adults. It is also sensitive to differences in mild autistic-like features in the normal population, supporting the notion that it is possible to regard autism as the end of a quantitative continuum. Men from the general population fell closer to the autistic end of the continuum than did women. Likewise, those engaged in careers in science and engineering scored closer to the autistic end of the continuum than those working in the arts.

There are 50 items in the AQ, divided into five subscales: social skills, communication, imagination, attention to detail and attention switching. On the whole, the items are not couched in language of disability, but rather ask the respondent to agree or disagree with statements about personal preferences and habits. Sample items from the five scales respectively are: 'I prefer to do things with others, rather than on my own'; 'People often tell me that I keep going on and on about the same thing'; 'I don't particularly enjoy reading fiction'; 'I am fascinated by numbers'; and 'I enjoy doing things spontaneously'. A negative response to the first and last of these items, and a positive response to the remaining items, would lead to a high score (closer to the autistic end of the continuum).

Methods

Participants

The data reported here comes from parent participants in the Western Australia Family Study of Autistic Spectrum Disorders (WAFSASD). Families with a child who had autism or a related pervasive developmental disorder (index families) were recruited via newsletters for special interest groups (e.g., Asperger's support group) and brochures sent to centres where children with autism or PDDNOS were diagnosed or educated. Staff were asked to mail copies of the brochure with a cover letter to appropriate families, inviting them to take part. Families were excluded from the study if the proband had a medical condition that could be causally related to autism, such as Fragile X syndrome. All

referred children had been given a clinical diagnosis of autism spectrum disorder and were designated the index probands. All were assessed with the Autism Diagnostic Interview – Revised (Lord, Rutter, & Le Couteur, 1994). Fifty-nine probands met ADI-R algorithm criteria for autism. The remaining 21 probands scored above the cut-off point on only one or two ADI-R scales and these formed our PDD sub-group. Two potential probands who did not score above threshold on any domain of the ADI-R were excluded from the study.

In order to identify multiplex families (i.e., those with more than one affected child), all siblings of index probands were assessed with the Social Communication Questionnaire (SCQ: Berument, Rutter, Lord, Pickles, & Bailey, 1999), which is a 40-item parental questionnaire that covers the same areas of functioning as the ADI-R, but in much briefer format. The recommended cut-off for identifying PDD is 14, and for autism is 21. To ensure we did not miss any cases of PDD, those scoring above 10 were also given the ADI-R. In two families of index cases, a sibling also met the full ADI-R criteria for autism; in six families one sibling met our criteria for PDD but not autistic disorder; and in one family two siblings had PDD as we defined it.

Control families were recruited by advertisements sent to schools in the Perth Metropolitan Region, with the goal of recruiting control probands of similar age, sex and family size as the index probands, with parents comparable in age and social background. Control probands were screened for autistic symptomatology using the SCQ. The ADI-R was administered to those who scored above 10 points, and those scoring above the algorithm criterion on any domain ($N = 5$) were excluded from the control sample. This gave a final sample of 59 control families.

Parental assessment

Probands and their first degree relatives were seen at home for a neuropsychological examination, except for siblings aged 3.5 years or under. Parents were given a short form of the Wechsler Adult Intelligence Scale (1997), consisting of the subtests Vocabulary, Similarities, Picture Completion and Object Assembly, which were prorated to give short form estimates of Verbal and Performance IQ. Parents were sent the AQ for completion prior to a home visit. Those who had not completed it were asked to mail it back to the research team. Fully completed AQs were obtained from at least one parent for 50 of the 59 families in the autism group, 19 of the 21 families in the PDD group, and 52 of the 59 control families. In all, AQ data were available for 65 mothers and 46 fathers of index families, and 48 mothers and 37 fathers of control families. IQ data were obtained from all these mothers and from 52 of the index fathers and 37 of the control fathers.

Results

Index and control parents were well matched in terms of age and estimated IQ (see Table 1; All differences between index and control parents gave nonsignificant F-ratios of less than one on Anova).

Mean scores on the AQ subscales are shown in Table 2. High scores indicate behaviour characteristic of autism. In general, the means for the control parents are in a similar range to those seen in the control adults of Baron-Cohen et al. (2001). One exception is the attention to detail scale, where the means obtained by Baron-Cohen et al. were significantly higher: for fathers 5.2, SD 2.3, and for

Table 1 Mean (SD) age and IQ for parents of index and control probands

	Index		Control	
	Mother $N = 69$	Father $N = 52$	Mother $N = 52$	Father $N = 37$
age (yr)	40.0 (4.59)	43.0 (6.43)	39.9 (4.71)	42.2 (6.17)
PIQ	105.6 (16.22)	106.1 (17.99)	107.3 (17.02)	108.9 (13.36)
VIQ	102.9 (14.35)	105.0 (16.30)	106.0 (16.40)	101.8 (13.93)

Table 2 Mean (SD) scores on AQ subscales for parents, in relation to gender and proband diagnosis

	Index		Control							
	Mother	Father	Mother	Father	Gender			Proband diagnosis		
Subscale	$N = 65$	$N = 46$	$N = 48$	$N = 37$	F	η^2	p	F	η^2	p
Social skills	2.22 (2.52)	3.80 (2.81)	1.75 (1.47)	2.27 (2.21)	9.7	.048	.002	8.71	.043	.004
Attention switching	3.55 (2.43)	3.93 (2.03)	3.25 (1.85)	4.32 (2.04)	5.48	.028	.020	.019	0	.891
Attention to detail	3.89 (2.16)	3.83 (2.29)	4.54 (2.24)	3.57 (1.94)	2.7	.014	.102	.38	.002	.538
Communication	2.12 (2.05)	3.39 (2.02)	1.75 (1.62)	2.43 (1.57)	12.95	.063	<.001	6.04	.030	.015
Imagination	2.02 (2.13)	3.15 (2.09)	1.88 (1.55)	3.54 (1.89)	24.3	.112	<.001	.19	.001	.663

mothers mean 5.4, SD 2.3. *T*-tests for data with unequal variances were applied and these differences were shown to be reliable: for fathers, t (108.9) = 4.48, $p < .001$; for mothers t (96.7) = 2.16, $p = .03$.

Our data were subjected to two-way Anova, with parent gender as one factor, and proband status (index or control) as the other. *F*-ratios, effect sizes (η^2), and *p*-values for main effects of gender and proband status are also shown in Table 2. Interaction terms are not shown as none approached significance.

There were significant main effects for gender in the predicted direction, with men scoring higher than women, for all scales except attention to detail, where, contrary to prediction, the trend was for women to obtain higher scores than men. The main effect of proband diagnosis was significant for two of the scales: social skills and communication, but did not approach significance for the other scales.

In the original AQ study by Baron-Cohen et al. (2001), two items on the attention to detail scale were identified as poor because normal controls obtained higher scores than people with autism. Accordingly, results from this scale were recomputed with these two items omitted. This did not, however, materially affect the pattern of results.

The two subscales that differentiated the proband groups were significantly intercorrelated (Pearson $r = .663$, $N = 196$, $p < .001$). A combined score was therefore derived using principal components analysis, and the distribution of this score for the four parent groups is shown in Table 3. Parents of index cases were subdivided according to this score, using a cut-off of 1.0, so that those with factor scores above this value were placed in the 'broad phenotype' group, and the remainder in the 'no symptoms' group. Given that low IQ is associated with autism, one might ask whether parents in the broad phenotype group have evidence of lower IQ than other parents. As can be seen from Table 4, this was clearly not the case. The two subgroups of index parents did not differ in terms of their IQ scores, though there was a nonsignificant trend for higher performance IQ in fathers with the broad phenotype. A subsidiary analysis was conducted to see whether the broad phenotype was more common in multiplex families, who might be expected to have a particularly high genetic loading for autism. There was no association between presence of the broad phenotype in a parent, and presence of a second affected child (with autism or PDD) in the family. AQ data were available for mothers from seven of the nine multiplex families, of whom only one (14%) had the broad phenotype, compared with 9 of 62 (14%) mothers of a single autistic child. For fathers, one in four (25%) from multiplex families had the broad phenotype, compared with 17 of 48 (35%) fathers of a single affected child.

Szatmari et al. (2000), using an interview to identify cases of the broad phenotype, reported that social-communicative impairments in biological relatives were significantly more common for PDD probands with an IQ above 60 than for those of lower

Table 3 Number (%) of cases with scores in a given range on the Social Skills/Communication factor

	Index		Control	
	Mother	Father	Mother	Father
−1.00 to −1.49	13 (20%)	5 (10%)	7 (14%)	2 (5%)
−.50 to −.99	18 (27%)	6 (13%)	17 (35%)	9 (24%)
0 to −.49	14 (21%)	3 (6%)	12 (25%)	10 (27%)
.01 to.49	4 (6%)	5 (10%)	5 (10%)	8 (21%)
.50 to.99	6 (9%)	10 (21%)	5 (10%)	3 (8%)
1.00 to 1.49	3 (4%)	6 (13%)	1 (2%)	3 (8%)
1.50 to 1.99	4 (6%)	7 (15%)	1 (2%)	1 (2%)
2.00 to 2.49	1 (1%)	1 (2%)	0 –	1 (2%)
2.50 to 2.99	1 (1%)	3 (6%)	0 –	0 –
3.00 to 3.49	1 (1%)	0 –	0 –	0 –
N	65	46	48	37

Table 4 IQ of parents in index group, subdivided into 'broad phenotype' and unaffected cases on the basis of scores on the Social Skills and Communication factor of the AQ

			t-test	
	Broad phenotype	Unaffected	*t*	*p*
Mothers, *N*	10	55		
Performance IQ	100 (11.35)	107.4 (16.66)	1.34	.184
Verbal IQ	102.1 (15.25)	103.9 (14.45)	.35	.727
Fathers, *N*	17	29		
Performance IQ	113.6 (15.91)	104.6 (17.72)	1.73	.092
Verbal IQ	106.9 (11.9)	104.6 (19.13)	.46	.645

IQ. This was not found in the current sample: rates of broad phenotype did not differ significantly in relation to proband IQ for either mothers or fathers. For mothers, 3 of 22 with proband VIQ of 60 or less (12%) and 7 of 44 with proband VIQ of 61 or more (16%) had the broad phenotype, $\chi^2(1) = .19$, $p = .657$. For fathers, 7 of 17 with proband VIQ of 60 or less (41%) and 11 of 35 with proband VIQ of 61 or more (31%) had the broad phenotype, $\chi^2(1) = .48$, $p = .488$.

Finally, we considered whether families where a parent had the broad phenotype were more likely than other index families to have a child meeting ADI-R algorithmic criteria for autism, as opposed to PDD. Families with no data on the father and an unaffected mother were excluded from this computation, because the status of these families in terms of the presence of an affected parent was unknown. This left 49 index families: eight with a broad phenotype mother, 15 with a broad phenotype father, and two where both parents had the broad phenotype. In 18 of the 25 (72%) index families with a broad phenotype parent, the proband had a diagnosis of core autism, as compared with 17 of the 24 (71%) index families where neither parent had the broad phenotype. Clearly, this is not a meaningful difference.

The choice of cut-off for categorising cases of the broad phenotype is bound to be somewhat arbitrary: the value of 1.0 was picked as it selected a minority of control parents, but sufficient index parents for meaningful comparisons between broad phenotype cases and the remainder. However, the percentage of parents selected using this cut-off, 24% overall, was higher than the proportion of parents affected with the broad phenotype in the study by Bolton et al. (1994), which was 11%. All analyses were re-run using the more extreme cut-off of 1.5 to subdivide index parents into broad phenotype and no symptom groups, giving a rate of broad phenotype, 10%, comparable to that of Bolton et al. (1994). However, the pattern of results was unchanged.

Discussion

Our control data largely supported the idea that the traits measured by the Autism Spectrum Quotient are ones that are differentiated in non-autistic men and women. An exception was the attention to detail scale, in which control women obtained slightly higher scores than men. Although Baron-Cohen (2002) would predict higher attention to detail scores in men, he too failed to find such a difference in the adult control sample of Baron-Cohen et al. (2001).

The AQ identified relatively high rates of autistic-like traits in parents of children with an autistic spectrum disorder. It is noteworthy, however, that differences between parents in the index and control groups were seen only on the social skills and communication scales. Furthermore, these two scales were significantly intercorrelated, suggesting that a single underlying cognitive dimension may be implicated in high scores on both sets of questions. This pattern of results has some commonalities with that observed by Wolff et al. (1988), who failed to find any differences between parents of children with autism and control parents on ratings of special interest patterns, obsessionality, or empathy, but who did find differences in communicative style and social gaucheness.

Baron-Cohen et al. (2001) did not report data on intercorrelations between subscales of the AQ, but subsequent work by Baron-Cohen (2002) is consistent with the idea that social and communicative aspects of autistic behaviour may constitute a separate dimension from other aspects of autism such as rigidity of thought and focussed attention. Baron-Cohen, Richler, Bisarya, Gurunathan, and Wheelwright (2003) have drawn a distinction between two ways in which humans make sense of and predict events, empathising and systemising. Empathising is a mode of thought that is usually best developed in females, and involves predicting other people's behaviour by identifying their emotions and thoughts. It thus has some conceptual overlap with the social and communicative aspects of the AQ. Systemising is a more typically male pattern of cognition, and involves a drive to analyse the variables in a system and uncover the underlying rules that govern its behaviour. It requires the ability to attend to fine details. Baron-Cohen et al. (2003), using a new questionnaire specifically designed to assess these traits, found a nonsignificant correlation between systematising and empathising. Viewed from this perspective, our data may be interpreted as showing that parents of people with autism have relatively low levels of 'empathising', but do not differ from the control group in terms of 'systemising'. Such data suggest that there may be genetic fractionation of different components of the autistic cognitive profile, with only 'empathising' skills showing high heritability. It will be of interest to see whether more direct assessment of behaviours such as attention to detail, attention switching, or local versus global processing differentiates between parents of probands with autism and control parents.

Although parents of probands with an autistic spectrum disorder obtained relatively high scores (indicative of a more autistic-like profile) on the communication scale, they did not differ from other parents in language abilities, as assessed by a short form verbal IQ. This suggests that, like high-functioning people with autism, they show a dissociation between ability to learn words and their meanings, and their ability to use their language skills to communicate and socialise effectively.

We need to be alert to the possibility that self-report of autistic-like characteristics in parents could in part reflect their familiarity with the symptoms of

autism. To help rule out a role for response bias on AQ responding, future studies are needed to assess how the self-reported traits identified by the AQ relate to behaviours and cognitive styles identified using other means of assessment. However, there are two reasons why it seems unlikely that such bias could explain the findings we report here. First, in designing the AQ, Baron-Cohen et al. (2001) aimed to minimize such biased responding by having test items that asked about people's preferences, rather than ability or disability. Second, someone who was over-sensitised to autistic behaviours might be expected to score high on all five scales of the AQ, rather than selectively on two scales. We suggest that evidence presented here is strong enough to merit use of the AQ social and communication scales as a quick and efficient means for screening for cases of the broad phenotype in genetic studies of autism. The ultimate test of its usefulness in such a context will be if it helps identify risk genes for autism.

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Correspondence to

Dorothy V.M. Bishop, Department of Experimental Psychology, University of Oxford, South Parks Road, Oxford, OX1 3UD, UK; Tel: 44 +1865 271369; Fax 44 +1865 281255; Email: dorothy.bishop@psy.ox.ac.uk

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