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BRIEF REPORT

Brief Report: Do the Nature of Communication Impairments in Autism Spectrum Disorders Relate to the Broader Autism Phenotype in Parents?

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Abstract Extensive empirical evidence indicates that the lesser variant of Autism Spectrum Disorders (ASD) involves a communication impairment that is similar to, but milder than, the deficit in clinical ASD. This research explored the relationship between the broader autism phenotype (BAP) among parents, an index of genetic liability for ASD, and proband communication difficulties. ASD probands with at least one BAP parent (identified using the Autism Spectrum Quotient) had greater structural and pragmatic language difficulties (assessed using the Children's Communication Checklist-2) than ASD probands with no BAP parent. This finding provides support for the position that genetic liability for ASD is associated with increased communication difficulties across structural and pragmatic domains.

Keywords Heritable language phenotype · Broader autism phenotype · Communication impairment

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Autism spectrum disorders (ASDs) are highly heritable conditions. Siblings of a child with an ASD are at increased risk of having an ASD and there is higher concordance for monozygotic relative to dizygotic twins (Bailey et al. 1995; Folstein and Rutter 1977a, b). One aspect of the heritable ASD phenotype that has attracted interest in the past decade has been the communication difficulties experienced by this population. Pragmatic language impairments—difficulties with the use of language—are pervasive in the ASD population (Baltaxe 1977; Eisenmajer et al. 1998; Rapin and Allen 1983; Tager-Flusberg 1981). However, there is considerable variability in the linguistic capabilities, with some individuals with ASD never developing functional verbal language, while others have precocious vocabularies, and often progress to develop fluent and complex language.

The broader autism phenotype (BAP) provides a means for examining the heritability of pragmatic and structural language impairments in families of children with ASD. A proportion of non-affected biological relatives of ASD probands exhibit sub-threshold levels of ASD symptomatology (Szatmari et al. 2000). It is hypothesized that the same genetic risk factors responsible for ASD may also be responsible for the milder difficulties observed in relatives (Bailey et al. 1998). Studies of ASD probands that incorporate knowledge of their relatives' BAP status will help reveal which deficits are genetically transmitted in ASD.

Early studies indicated that the lesser variant of ASD included pragmatic language difficulties that were milder than, but similar to the deficits observed in ASD (Bolton et al. 1994; Landa et al. 1992; Piven et al. 1997a, b). Several family studies have also found increased rates of language and literacy difficulties among relatives of children with ASD (Bailey et al. 1998; Folstein et al. 1999; Ruser et al. 2007; Tomblin et al. 2003), providing further

support for the presence of communication impairments in the BAP. Other studies of language in ASD families have reported that some relatives of children with ASD perform poorly on tests of nonword and sentence repetition, which are purported markers for heritable structural language impairment (Lindgren et al. 2009). In contrast, several studies have found that the communication impairments that characterize the BAP may be restricted to the pragmatic domain. For example, Whitehouse et al. (2007) reported that parents of children with ASD had impaired pragmatic, but not structural language skills. These findings were replicated in a larger study that included cohorts in the UK and US, which found that the predominant communication difficulty experienced by parents of a child with ASD was in social engagement rather than language structure (Whitehouse et al. 2010). More recent studies have provided further evidence to support the claim that social communication deficits are part of the heritable ASD phenotype (Bernier et al. 2012; Gerdts et al. 2012). Interestingly, these results indicate that parents from multiplex ASD families demonstrate a greater degree of ASD characteristics relative to parents from simplex ASD families, particularly in social and communication domains. Overall, these data suggest that impairments in social communication rather than structural language comprise the heritable communication deficit in ASD.

The majority of research to date has examined the effect of proband phenotype (ASD vs. non-ASD) on relative BAP status, with far less research investigating the reverse. By investigating proband characteristics as a function of parental BAP status, we can start to determine which aspects of communication may be part of the heritable ASD phenotype. One previous study has examined possible links between parent BAP status and offspring communication. Bishop et al. (2006) assessed communication in the siblings of ASD children using the Children's Communication Checklist-2 (CCC-2; Bishop 2003a), a parent-report scale designed to screen for pragmatic and structural language difficulties and behavioural characteristics of ASD. Bishop et al. (2006) identified the siblings with low CCC-2 scores and then examined their parents' autistic-like traits using the Autism Spectrum Quotient (AQ; Baron-Cohen et al. 2001). While the mothers of the low CCC-2 subgroup were not atypical on the AQ, 80 % of the fathers had high scores on the social and communication subscales. Interestingly, in an earlier study, Bishop et al. (2004a) found no increased rate of language and literacy difficulties in ASD parents. However, when the AQ was used to classify parents as BAP or non-BAP, BAP parents were more likely to report a personal history of language or literacy impairment than non-BAP parents. These studies are limited by small numbers (e.g. Bishop et al. 2006, had only five fathers of children with low CCC-2 scores) and have not explored associations between parent BAP status and communication in the ASD child. Nonetheless, the limited evidence of this kind suggests that genetic liability for ASD, as indexed by the BAP, may be associated with structural language difficulties.

The current study explored relationships between parent BAP status and the communication characteristics of ASD probands using the CCC-2. We hypothesised that if structural language deficits are part of the heritable ASD phenotype, then the children of BAP parents would have more severe structural and pragmatic language impairments than the children of non-BAP parents. Conversely, if pragmatic, but not structural language difficulties are part of the heritable ASD phenotype, then children would have similar structural language abilities regardless of the BAP status of their parents.

Method

Participants

Participants were part of the Western Australian Autism Biological Registry (WAABR), which is an ongoing study of children with ASD and their families taking place at the Telethon Institute for Child Health Research in Perth, Western Australia. Participants were recruited via newspaper advertisements and children with a clinical diagnosis of autistic disorder, Asperger's disorder or pervasive developmental disorder-not otherwise specified (PDD-NOS) were included in the study. In Western Australia, diagnosis of ASD is obtained by consensus following a multidisciplinary assessment by a team comprising a paediatrician, clinical psychologist and speech pathologist. We sought to verify diagnoses using the Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord et al. 2000). Forty-seven of the ASD probands reached the ADOS-G cut-off for autism; 18 met criteria for the 'autism spectrum' and 17 did not meet the ASD threshold based solely on the ADOS-G scores. Of the 17 children who did not meet ADOS-G criteria for ASD, 13 had received a clinical diagnosis of Autistic Disorder, two had been diagnosed with PDD-NOS, and two with Asperger's disorder. Clinical judgment remains the 'gold-standard' for diagnosis of ASD (Lord et al. 2012), with ADOS-G often used as a confirmatory measure. Given that all children in this study had received a consensus multidisciplinary clinical diagnosis, and that the majority had also reached at least ASD threshold on the ADOS-G, none of the children recruited were excluded from the study.

Eighty-two families provided a completed CCC-2 for the proband and the AQ for both parents (see Tables 1, 2 for participant characteristics). All children with ASD were between 4 and 17 years of age, and were speaking in

Table 1 ASD diagnosis, gender and mean (and SD) chronological age for ASD probands

	Autistic disorder	Asperger's disorder	PDD-NOS
N	68	7	7
Male: female	56:12	5:2	6:1
Age range	4:1–17:10	5:4–13:8	4:6–16:5
Years: months			
M	8:7	10:1	8:6
SD	3:8	2:9	4:4

Table 2 Means (and SDs) of age and AQ scores for parents and proband ADOS-G social and communication scores as a function of sex and BAP status

	Mother		Father	
	BAP	Non-BAP	BAP	Non-BAP
N	21	61	22	60
Age				
M	39.43	40.80	43.41	42.67
SD	4.34	6.27	5.00	7.44
AQ				
Range	26–43	4–24	26–42	3–25
M	33.43	12.36	31.73	16.25
SD	6.69	5.40	4.56	5.64
ADOS communication				
M	4.57	4.26	4.14	4.43
SD	1.72	2.20	1.91	2.15
ADOS social				
M	7.23	6.70	7.18	6.82
SD	3.33	2.81	2.20	3.17

sentences, which is a requirement for the CCC-2. Woodbury-Smith et al. (2005) evaluated the AQ for its use as a clinical screening questionnaire and found that a score of 26 and above was a useful cut-off to identify high levels of autistic-like traits which may require further clinical assessment. This threshold was used to identify parents with the BAP in the current study.

Procedure and Measures

Participants were mailed the CCC-2 and AQ as part of a larger battery of questionnaires, which they completed at home. Families attended the Telethon Institute where the ADOS-G was administered by a trained assessor.

The Children's Communication Checklist (CCC-2; Bishop 2003a) is a 70-item parent-report questionnaire designed to screen for communication difficulties in children with phrase speech. The scale is comprised of ten subscales that measure general communication difficulties (speech, syntax, semantics and coherence), pragmatic language (inappropriate initiation, stereotyped language, use of context and nonverbal communication) and behaviours commonly associated with ASD (social behaviour and interests). Standard scores with a mean of 10 can be derived for each subscale. Two composite scores can be computed: the Global Communication Composite (GCC) provides a measure of overall communication ability and the Social Interaction Deviance Composite (SIDC) identifies children who have pragmatic impairments disproportionate to their structural language abilities.

The Autism Spectrum Quotient (AQ; Baron-Cohen et al. 2001) is a 50-item self-report questionnaire that measures mild levels of autistic-like traits. Scores range from 0 to 50 and higher scores indicate a greater degree of autistic-like characteristics.

Results

The means and standard deviations for parent age, AQ scores and proband ADOS-G social and communication scores are presented in Table 2. While fathers were found to be significantly older than mothers, $F(1, 163) = 5.90$, $p < .05$, $\eta_p^2 = .035$, there was no age difference between the BAP and non-BAP groups for mothers, $F(1, 81) = .864$, n.s., $\eta_p^2 = .011$, or for fathers, $F(1, 81) = .187$, n.s., $\eta_p^2 = .002$. As expected, given the way the groups were formed, the BAP group had higher AQ scores than the non-BAP group for mothers, $F(1, 81) = 209.55$, $p < .001$, $\eta_p^2 = .72$, and for fathers, $F(1, 81) = 134.35$, $p < .001$, $\eta_p^2 = .63$ (see Table 2). There was no difference in proband ADOS-G severity (indexed as the social and communication total score) between the BAP and non-BAP groups for mothers, $F(1, 81) = .614$, n.s., $\eta_p^2 = .088$, or for fathers, $F(1, 81) = .004$, n.s., $\eta_p^2 = 0$.

In order to explore whether proband communication difficulties increased with higher genetic liability for ASD, we partitioned the probands into three groups based on whether neither parent met the criteria for the BAP, one parent was BAP, or both parents were BAP. Prior to partitioning the groups in this manner, we investigated whether proband CCC-2 scores differed according to the sex of the BAP parent for those families where just one parent met the BAP criterion. Univariate ANOVAs with BAP group as the between subjects factor (mother only BAP, father only BAP) showed no differences on any of the CCC-2 subscales (all p values >.05). As no differences between the mother only BAP and father only BAP groups were apparent, we collapsed these two groups together to form a single 'one parent BAP' group.

Table 3 Means (and SDs) standard scores for each subscale of the CCC-2 as a function of parent BAP status

Subscale	Parent BAP status						
	Neither (N) N = 45	One (O) N = 31	Both (B) N = 6	*F*	*p*	η_p^2	Post hoc tests (LSD)
A: Speech	6.00 (3.89)	3.55 (2.99)	4.17 (4.71)	4.32	.017	.099	N > O; N = B; B = O
B: Syntax	4.31 (3.65)	2.97 (3.38)	4.33 (3.93)	1.37	.26	.034	No group difference
C: Semantic	5.64 (3.18)	4.09 (2.57)	4.33 (1.21)	2.82	.066	.067	No group difference
D: Coherence	4.04 (2.06)	2.81 (1.96)	2.00 (1.41)	5.25	.007	.117	N > O; N > B; B = O
E: Inappropriate Initiation	5.64 (2.18)	4.16 (2.76)	4.17 (1.47)	3.94	.023	.091	N > O; N = B; B = O
F: Stereotyped	4.67 (2.62)	3.42 (2.42)	1.50 (1.05)	5.55	.006	.123	N > B; N > O; B = O
G: Use of Context	3.71 (2.40)	1.90 (2.43)	1.50 (1.52)	6.45	.003	.140	N > O; N > B; B = O
H: Non-Verbal	2.93 (1.71)	2.06 (1.77)	1.33 (1.51)	3.80	.027	.088	N > O; N > B; B = O
I: Social	2.69 (2.02)	2.26 (2.31)	.17 (.41)	3.95	.023	.091	N = O; N > B; B < O
J: Interests	5.33 (2.37)	4.32 (2.88)	3.00 (1.67)	3.04	.054	.071	No group difference
GCC	36.96 (15.27)	24.97 (12.77)	23.33 (11.11)	7.70	.001	.163	N > O; N > B; B = O
SIDC	−3.40 (8.99)	−.61 (10.07)	−6.17 (10.52)	1.26	.290	.031	No group difference

Descriptive statistics for proband standard scores on each subscale of the CCC-2 are presented in Table 3. Univariate ANOVAs with BAP group (neither, one, both) as a between-subjects factor revealed significant group differences for all subscales except for syntax, semantic and interests[1]. While a significant group difference was also present for the GCC, no difference was observed for the SIDC (see Table 3). Post hoc tests (LSD) were used to follow-up significant group differences, in most cases showing significantly higher standard scores for probands with no BAP parent compared to probands with one or two BAP parents (see Table 3). Scores for probands with two BAP parents were similar to scores for probands with one BAP parent across all subscales, except for social, where probands with both BAP parents had significantly lower scores than probands with only one BAP parent.

Discussion

The current study explored the relationship between parent BAP status and proband communication difficulties. The results showed that probands with at least one BAP parent had lower (worse) scores on both structural and pragmatic CCC-2 subscales compared to probands with no BAP parent. These findings are consistent with other studies that have used the BAP to assess the heritability of offspring communication characteristics. For example, Bishop et al. (2004b) found that BAP parents were more likely to report a personal history of language and literacy difficulties than non-BAP parents. More recently, Bishop et al. (2006) found that the BAP status of ASD fathers was associated with poor offspring communication across structural (speech, semantic, coherence) and pragmatic domains. Taken together, these findings provide some support for the position that both pragmatic and structural deficits may be part of the heritable ASD phenotype.

There are several possible reasons for the observed communication difficulties in the probands with BAP parents. It is plausible that there is a gene-environment interaction, such that ASD probands with a BAP parent have both an increased genetic liability for a social communication impairment and also less opportunity to learn important aspects of communication from parents who themselves may have structural or pragmatic language difficulties. A further possibility is that families with greater genetic liability for ASD have children with more severe difficulties across developmental domains, including structural and pragmatic language. Certainly, associations have been reported from research of a complementary design, whereby parents from multiple-incidence autism families, thought to represent higher genetic liability for ASD, have more pragmatic and speech difficulties than parents from single incidence autism families (Bernier et al. 2012; Gerdts et al. 2012; Losh et al. 2008; Piven et al. 1997a, b). Finally, there may be an interrelationship between pragmatic and structural difficulties in the ASD probands with BAP parents, whereby the increased genetic liability for ASD leads to greater pragmatic impairments, which result in increased difficulty learning aspects of

[1] We re-ran the univariate ANOVAs without the children who did not meet ADOS-G cut-offs for ASD. While the *p*-values were slightly higher than for the original analysis, the overall pattern of results was unchanged. Significant effects were retained across the Coherence, Inappropriate Initiation, Stereotyped Language and Use of Context subscales as well as for the Global Communication Composite. There was also a trend towards significance for the Social subscale ($p = .062$)

structural language. Importantly, while the current study reports associations between communicative characteristics of ASD probands and their parents, the heritability of linguistic and pragmatic capabilities can only be determined through twin studies in which environmental influences on behaviour can be assessed. In these studies, it may be useful to obtain more extensive data on the ASD characteristics of the parents, by including measures of parent language as well as behavioural assessments of the ASD phenotype. The inclusion of more detailed information about the parental phenotypes, as well as child language and ASD characteristics, will help to determine whether the communication characteristics of children with ASD are inherited or alternatively, learned from parents who themselves may have structural or pragmatic difficulties.

In this study, the AQ and CCC-2 were completed by parents who are knowledgeable about and possibly sensitive to the characteristics of ASD, which may result in biased interpretations of the child's communicative difficulties. In addition, having a child with ASD could lead some parents to interpret personality characteristics that they share with their child as features of ASD, resulting in overestimations of autistic-like tendencies on self-report measures such as the AQ. However, parent completed CCC-2 questionnaires have been recognised as a valid tool to identify and discriminate between different communication disorders and scores are consistent with clinical diagnoses that have been assigned based on direct measures of language ability (Norbury et al. 2004). Nonetheless, in future studies, it may be useful to include an additional language measure that is rated by a relatively impartial third party.

Interestingly, the current results indicate that the parent BAP may be associated with impairments in some aspects of structural language, suggesting that genetic liability for ASD may confer risk for a language impairment that resembles Specific Language Impairment (SLI). SLI is defined by a language delay in the absence of neurological, intellectual, sensory, or other developmental difficulties (Bishop 2003b; Stark and Tallal 1981). While the structural language impairment in SLI contrasts with the pragmatic impairment in ASD, recent findings indicate that the conditions share some linguistic characteristics (Kjelgaard and Tager-Flusberg 2001; Lewis et al. 2007; Rapin et al. 2009). The current results, which indicate that probands with a BAP parent have more structural language difficulties than probands with no BAP parent could represent superficial overlap in the ASD and SLI phenotypes, resulting from phenomimicry (Bishop 2010). Nonetheless, while the current findings suggest that there could be a degree of overlap in the heritable communication impairment in ASD and SLI, future investigations that compare the broader phenotypes of ASD and SLI in parents will make an important contribution to theoretical arguments about overlap in these conditions.

This research reports novel associations between parent BAP status and proband communication using the CCC-2. The CCC-2 is useful in explorations of the heritable ASD phenotype as it measures structural and pragmatic language, thereby allowing us to potentially distinguish between the heritable phenotype of communication disorders such as SLI and the heritable ASD phenotype. However, the children with ASD in the current study were at least 4 years of age and had functional verbal language. Further insight into the heritable language phenotype of ASD could be provided by investigating whether parents' BAP status is associated with early language development, with the development of non-verbal communication, and with whether functional verbal language is achieved in the child with ASD.

The current study reports associations between parent BAP status and proband communication characteristics. The findings make a unique contribution to research concerning the heritable language impairment in ASD and may have implications for theories of overlap between ASD and other heritable communication disorders, such as SLI.

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