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Alexithymia in Parents of Children with Autism Spectrum Disorder

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Abstract Given the recent findings regarding the association between alexithymia and Autism Spectrum Disorder (ASD) and the accumulating evidence for the presence of the Broader Autism Phenotype (BAP) in relatives of individuals with ASD, we further explored the construct of alexithymia in parents of children with ASD as a potential part of the BAP. We hypothesized that (a) parents of children with ASD will demonstrate higher impairment in their emotion processing when compared to controls, and (b) high impairment in emotion processing in parents will be associated with severity of symptoms in children with ASD. Psychometric and diagnostic data were collected on 188 children with a diagnosis of ASD. The Toronto Alexithymia Scale (TAS-20) was completed by 439 parents of children with ASD and a control group of 45 parents of children with Prader Willi syndrome (PW). Results show that ASD parents score higher than controls on the TAS-20 total score. Within the ASD group, children of fathers with *high alexithymia* score higher on repetitive behaviour symptoms compared to children of fathers with *low alexithymia*. The alexithymia trait appears to be one of the many building blocks that make up the BAP.

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Pervasive Developmental Disorders (PDD) or Autism Spectrum Disorders (ASD) are a group of disorders characterized by core impairments in social-communication, persistent inflexibility, and by a preference for repetitive activities (APA 1994). It is believed that individuals with ASD have an impaired ability to express and recognize their own and other people's emotions (Baron-Cohen et al. 2000). Bölte and Poustka (2003) demonstrated that subjects with autism performed worse than schizophrenic and unaffected individuals on facial recognition tests, supporting the proposition that emotion detection deficits are part of the autism phenotype. Ben Shalom et al. (2006) noted that impairments in socio-emotional expression in autism may be related to deficits in perception and/or expression of conscious feelings. Finally, Bölte et al. (2008) reported altered physiological reactivity and affective reporting in individuals with autism, something that may be related to more general impairments in socio-emotional functioning. There is now accumulating evidence that impairments in emotion processing are part of the core mechanism involved in ASD.

A number of family studies (Bailey et al. 1995; Bolton et al. 1994; Piven et al. 1997; Piven 2001; Szatmari et al. 2000) have also supported the idea that within-family transmission of the autism phenotype, may extend to a spectrum of traits that are present in some relatives of individuals with autism but without serious degrees of impairment. Even though these relatives do not meet the standard diagnostic criteria for ASD, they seem to present some ASD-like traits and/or characteristics termed the

"Broader Autism Phenotype" (BAP). Findings on the BAP show that it is potentially associated with social and cognitive deficits, restricted behaviour patterns, as well as with certain personality characteristics and psychiatric difficulties that can be found in parents of autistic children (Bolton et al. 1994; Landa et al. 1992; Piven et al. 1994, 1997; Wolff et al. 1988; Murphy et al. 2000; Yirmiya and Shaked 2005). According to Skuse et al. (2005) autistic traits are widely distributed in the general population, but the boundaries of the autistic spectrum are unclear. Skuse et al. (2005) proposed the use of the Social and Communication Disorders Checklist (SCDC) as a screening tool for measuring heritable dimensional autism-related traits in the general population. Furthermore, Constantino and Todd (2005) and Constantino et al. (2006) using the Social Responsiveness scale (SRS) found familial transmission of common, subsyndromal social impairments that may be related to the causes of categorically defined pervasive developmental disorders. Despite increased interest in the study of the BAP as a dimensional approach to autism, currently, there is no agreement on the definition of the BAP and it is not entirely clear which traits/constructs make up the BAP (Scheeren and Stauder 2008).

Sifneos (1973) coined the word "alexithymia" (derived from the Greek language: a = lack, lexis = word, thymos = emotions) which literally means "having no words for emotions". Alexithymia is not a diagnosis, but a construct useful for characterizing individuals who seem not to understand the emotional feelings they experience, or lack the words to describe these feelings to others. Alexithymia is conceptualized as a personality trait that is normally distributed in the population and is associated with increased risk of psychopathology (Franz et al. 2008). Furthermore, the construct of alexithymia is now considered part of the broader field of emotion research (Taylor and Bagby 2004). Tani et al. (2004) reported that individuals with Asperger's syndrome (AS) were significantly more alexithymic when compared with controls. Hill et al. (2004) also reported higher levels of alexithymia in 27 high-functioning adults with autistic spectrum disorders, when compared with a similar-aged control group. In a second study Berthoz and Hill (2005) demonstrated that these deficits in emotion regulation are stable over time in ASD. Thus alexithymia may be part of the emotion processing difficulties experienced by people with ASD.

Given the recent findings regarding the association between alexithymia and ASD and the accumulating evidence for the presence of the BAP in relatives of individuals with ASD, we further explored the construct of alexithymia in parents of children with ASD as a potential part of the BAP. More specifically, we hypothesized that (a) parents of children with ASD will demonstrate more difficulties in emotion processing when compared to a control group, and (b) high impairment in emotion processing in parents will be associated with severity of autistic symptoms in children with ASD.

Methods

Participants

Sample 1 (ASD vs. PW Parents)

The first sample consisted of 439 parents from families with children affected with ASD and 45 parents of children affected with Prader–Willi Syndrome (PW). The normative data for the Toronto Alexithymia Scale 20 (TAS-20, Bagby et al. 1994a, 1994b) is based on the responses of undergraduate university student volunteers. There is a problem when one compares these scores with those of parents of ASD children since the age of the parents is significantly higher than the age of the students. Furthermore, it is plausible that the stress of raising an atypical child increases alexithymia scores through its effects on depression (Taylor and Bagby 2004).

The group of parents of children with PW was selected as a suitable comparison group for age, and the effect of raising a child with special needs (more so, we would argue, than controls with Down's syndrome). They were ascertained through the local PW parent support group. PW children are also severely impaired, present with many behavioural problems of a quite different nature (they typically have compulsive food-related behaviours, oppositional traits and mild mental retardation). Furthermore, PW is not caused by the familial transmission of a genetic variant with an associated phenotype from parent to child so any evidence for the BAP in PW parents could be attributed to stress alone. All parents were assessed using the TAS-20 (Bagby et al. 1994a, 1994b). The diagnosis of ASD in the children was confirmed by best-estimate (Table 1a). We did not have data on the clinical characteristics of the PW children.

Sample 2 (Children with ASD and Their Parents)

The second sample consisted of 188 children with a diagnosis of ASD that had complete data on all measures of interest. The parents of these children with complete data on the TAS-20 were also included in this sample ($n = 344$; 188 mothers and 156 fathers). Although some families had two children with an ASD diagnosis, only one child (i.e. proband) per family was randomly selected for analyses in order to maintain independence of observations. All probands were initially assessed using the Autism Diagnostic Interview—Revised (ADI-R) and the Autism Diagnostic

Table 1 (a) Sample 1 (ASD vs. PW parents) descriptive statistics. (b) Sample 2 (children with ASD and their parents) descriptive statistics

Characteristics	*N*	Mean (Std. Dev.)	min-max
(a)			
Parent type			
ASD	439		
Male	202		
Female	237		
PW	45		
Male	17		
Female	28		
Age at completion of TAS-20			
ASD		39.23 (6.67)	25–68
PW		44.96 (8.14)	32–63
(b)			
Children	188		
Sex			
Male	145		
Female	43		
Best estimate diagnosis			
Autism	145		
PDD-NOS	20		
Asperger syndrome	23		
Leiter IQ adjusted	188	66.85 (28.97)	23–133
Age at ADI-R interview (in months)	188	113.91 (68.87)	28–482
Parents	344		
Mothers	188		
High alexithymia (TAS-20 total score ≥61)	14		
Low alexithymia (TAS-20 total score <61)	174		
Fathers	156		
High alexithymia (TAS-20 total score ≥61)	22		
Low alexithymia (TAS-20 total score <61)	134		

ASD: Autism Spectrum Disorders; PW: Prader–Willi syndrome; ADI-R: Autism Diagnostic Interview—Revised; TAS-20: Toronto Alexithymia Scale 20

Observation Schedule (ADOS). Further information was collected from a measure of non-verbal IQ, the Leiter International Performance Scale (Leiter). Data from the ADI-R and ADOS, as well as the Leiter and any previous clinical notes (including language, psychological, pediatric/psychiatric, occupational therapy, and school records) were forwarded to three 'expert' raters who had an average of 15 years experience in the diagnosis of ASD. Each rater made a diagnosis independently using DSM-IV criteria. If there was disagreement regarding the diagnosis, the raters discussed the case until a consensus diagnosis was reached (Mahoney et al. 1998). A diagnosis of autism was based on severe impairments in social reciprocity, in verbal and non-verbal communication plus the presence of speech delay (onset of phrases after 36 months). Thus, the children with autism could be differentiated from those with Asperger syndrome (AS) who spoke on time and had normal IQ. Children with Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS) had fewer symptoms than those with either autism or AS. Table 1b describes the characteristics for Sample 2. Further information regarding the diagnostic procedure and its reliability and validity is described in detail in Mahoney et al. (1998).

Assessment Measures

Autism Diagnostic Interview Revised (ADI-R)

The Revised version of the ADI (Lord et al. 1994), the most widely used research measure for the diagnosis of Autistic Disorder, is a standardized semi-structured interview consisting of three major domains: (1) social interaction, (2) nonverbal and/or verbal communication, and (3) restricted, repetitive behaviours and interests. A diagnostic algorithm that discriminates autism from other developmental disorders is based on cut-off points for each of the three domains.

Autism Diagnostic Observation Schedule (ADOS)

The ADOS (Lord et al. 2000) is a semi-structured direct assessment of communication, social interaction and play or imaginative use of materials, for individuals suspected of having autism or other autism spectrum disorders (ASDs). The ADOS consists of four modules, each of which is appropriate for individuals of differing language levels, ranging from nonverbal to verbally fluent.

Leiter International Performance Sales (Levine 1986)

The Leiter is a standard, nonverbal measure of problem solving and learning ability. The Leiter does not require verbal instructions or responses, which makes it appropriate for in individuals with autism, particularly those with low linguistic levels (Tidmarsh and Volkmar 2003).

Toronto Alexithymia Scale—20 (TAS-20)

The TAS-20 (Bagby et al. 1994a, 1994b) is a self-report scale comprised of 20 items. Each item is rated on a five-point Likert scale ranging from 1 (strongly disagree) to 5 (strongly agree). The TAS-20 has been translated and validated into 18 languages, and its efficacy has been evaluated with confirmatory factor-analysis in 19 countries (Taylor et al. 2003). The TAS-20 is a reliable and valid measure of

emotion processing in adults which includes a total score and three subscales: *Difficulty Identifying Feelings (DIF), Difficulty Describing Feelings (DDF), and Externally-Oriented Thinking (EOT)*. The first factor in the three-factor model for the TAS-20 consists of seven items assessing the ability to identify feelings and to distinguish them from the somatic sensations that accompany emotional arousal (e.g., "I am often confused about what emotion I am feeling" and "I have feelings that I can't quite identify"). Factor 2 consists of five items assessing the ability to describe feelings to other people (e.g., "I am able to describe my feelings easily" and "It is difficult for me to reveal my innermost feelings, even to close friends"). Factor 3 consists of eight items assessing externally oriented thinking (e.g., "I prefer to analyze problems rather than just describe them" and "Looking for hidden meanings in movies or plays distracts from their enjoyment"). The empirically derived cutoff score of 61 is used for identifying individuals with "high" or "low" alexithymia. This cut-off point was determined by three methods used in assessing the "goodness" of a diagnostic test—sensitivity, specificity, and predictive value in relation to a consensus decision by three clinicians who used interviews to designate individuals as "alexithymic" or "non-alexithymic" (Taylor et al. 1988; Bagby et al. 1994b; Taylor et al. 1997). Since then, a number of studies reported similar results with approximately 10% of the population exceeding the TAS-20 sum score threshold of 61 (Honkalampi et al. 2001; Franz et al. 2008).

Analyses

Comparison Between ASD and PW Parent Alexithymia Scores

First, due to the large difference in sample size between the ASD and PW parent groups in Sample 1, we tested for homogeneity of variances for TAS-20 scores. Since the hypothesis of homogeneity of variances was rejected ($p = 0.021$) we used a non-parametric method (i.e. *Wilcoxon–Mann–Whitney two-sample rank-sum test)* to compare their scores on the TAS-20. If a significant difference between parents and controls was seen on the total score, we then tested whether those differences were specific to any subscale or was specific to mothers or fathers. Because of multiple testing, a Bonferonni correction was applied to the levels of significance (i.e. from .05 to .015) for these sub-scales.

Association Between ASD Proband Characteristics and Parental Alexithymia Scores

For the purposes of all remaining analyses, Sample 2 comprised only ASD parents and their children. It should be noted that parental TAS-20 scores were normally distributed but child ADI-R scores were not. Using Sample 2, we compared means on maternal/paternal alexithymia scores based on the child's ASD subtype (i.e. Autism, vs. PDD-NOS, vs. Asperger's), family composition (i.e. *multiplex*-more than one child affected with ASD vs. *simplex*-one affected child per family), and language ability (i.e. verbal vs. nonverbal). We further examined potential mean differences in maternal/paternal alexithymia based on child IQ, child sex and a sex X IQ interaction term using analysis of variance (ANOVA).

Next, by applying the empirical total TAS-20 cut-off score of 61, we examined, using the *Wilcoxon–Mann–Whitney two-sample rank-sum test*, whether children of mothers/fathers with *high alexithymia* (i.e. TAS-20 total score ≥ 61) showed more autistic symptoms (as indexed by the ADI-R domains) compared to children of mothers/fathers with *low alexithymia* (i.e. TAS-20 total score < 61). Again, a Bonferonni correction was applied to the levels of significance (i.e. from .05 to .015) for the three sub-scales of the ADI-R.

Results

Comparison Between ASD and PW Parent Alexithymia Scores

The proportions of fathers and mothers were similar in the two groups ($p = 0.346$, Fisher's Exact Test). ASD parents score significantly higher (i.e. demonstrate greater alexithymia) than PW parents on the total score ($p = .05$). On the sub-scales the largest difference was on the subscale "Difficulty Identifying Feelings" ($p = .02$; see Table 2) but this did not reach significance when correcting for multiple testing. Results of analyses using Sample 1 indicate that mothers of ASD children had higher scores than mothers of PW children, and that fathers of ASD children had higher scores than fathers of PW children, although these differences were not significant presumably due to the smaller sample size when sub-grouping occurs.

Association Between ASD Proband Characteristics and Parental Alexithymia Scores

Within the ASD group (i.e. Sample 2), there are no significant differences (not shown here but available upon request; $p > .05$ for all scores) in maternal or paternal alexithymia scores (total or subscale) based on the child's diagnostic subtype (i.e. AD vs. PDD-NOS, vs. AS), family composition (i.e. multiplex vs. simplex), or language level (i.e. verbal vs. nonverbal). We also examined whether there are mean differences in total and/or subscale maternal and/

Table 2 Non-parametric Mann–Whitney test for comparison of mean rank for TAS-20 total and subscale scores, for ASD ($n = 439$) and PW parents ($n = 45$)

	Mean rank	Asymptotic significance
TAS-20 total		
ASD parents	246.46	0.05
PW parents	203.89	
TAS-20 DIF		
ASD parents	243.52	0.02
PW parents	195.63	
TAS-20 DDF		
ASD parents	244.43	0.23
PW parents	218.39	
TAS-20 EOT		
ASD parents	242.46	0.22
PW parents	215.70	

TAS-20: Toronto Alexithymia Scale 20; ASD: Autism Spectrum Disorders; PW: Prader–Willi syndrome

or paternal alexithymia scores based on proband characteristics, such as nonverbal IQ and sex. Results from analyses of variance (ANOVA) indicated that there were no differences in maternal or paternal scores based on these proband characteristics (not shown here but available on request).

We then classified parents by their total alexithymia scores using the empirical TAS-20 cut-off score of 61. Children of mothers with *high alexithymia* scored marginally higher on the ADI-R repetitive behaviours domain when compared to children of mothers with *low alexithymia* ($p = .04$; see Table 3a) but this did not reach statistical significance. However, children of fathers with *high alexithymia* scored significantly higher on the ADI-R repetitive behaviours domain when compared to children of fathers with *low alexithymia* ($p < .001$; see Table 3b). For the other two ADI-R domains (social and communication), there were no significant differences (see Table 3a and b).

Table 3 Non-parametric Mann–Whitney test for comparison of mean rank scores for ADI-R domains for (a) children of mothers with high alexithymia (i.e. TAS-20 ≥ 61, $n = 14$) vs. children of mothers with low alexithymia (i.e. TAS-20 < 61; $n = 174$) (b) children of fathers with high alexithymia (i.e. TAS-20 ≥ 61, $n = 22$) vs. children of fathers with low alexithymia (i.e. TAS-20 < 61; $n = 134$)

	Mean rank	Asymptotic significance
(a)		
ADI-R social domain		
High alexithymia in mother	86.50	0.76
Low alexithymia in mother	95.14	
ADI-R communication domain		
High alexithymia in mother	104.71	0.12
Low alexithymia in mother	93.68	
ADI-R repetitive behaviours domain		
High alexithymia in mother	116.14	0.04
Low alexithymia in mother	92.76	
(b)		
ADI-R social domain		
High alexithymia in father	90.05	0.19
Low alexithymia in father	76.60	
ADI-R communication domain		
High alexithymia in father	73.43	0.57
Low alexithymia in father	79.33	
ADI-R repetitive behaviours domain		
High alexithymia in father	105.77	<0.001
Low alexithymia in father	74.02	

TAS-20: Toronto Alexithymia Scale 20; ADI-R: Autism Diagnostic Interview—Revised

Discussion

Results of the current study provide evidence that the alexithymia trait, a trait similar to difficulties in social–emotional understanding found in the broader autism phenotype, is also seen in parents of children with ASD. These results are somewhat more pronounced using the measure of "Difficulty Identifying Feelings", which is probably closest to the kinds of traits seen in people with ASD. In addition, high alexithymia scores among fathers and perhaps mothers are associated with high scores in repetitive stereotyped behaviours in affected children. Higher scores are not associated with ASD subtype, family composition (i.e. multiplex vs. simplex), language ability, sex or IQ of the proband.

Combined with the published literature, the alexithymia trait appears to have the following properties (a) it is more common in individuals with ASD than in the general population (see Hill et al. 2004), (b) it is more common in parents of individuals with ASD than in parents of individuals with another developmental disability (i.e. PW), and (c) symptom severity in repetitive stereotyped behaviours in individuals with ASD is associated with high father's alexithymia scores. Together, these findings suggest that the alexithymia trait could be viewed as part of the BAP and meets at least some of the criteria for an "intermediate phenotype" (Carlson et al. 2004) or "endophenotype".

Duvall et al. (2007) demonstrated the utility of the teacher version of Social Responsiveness Scale (SRS) as a quantitative endophenotype to detect autism-related genetic loci using quantitative behavioural information from affected children and their "unaffected" siblings (unaffected with ASD that is). It may seem surprising that in the current study, parental alexithymia scores were not

correlated with child scores of autistic symptoms in social reciprocity. In fact, with the exception of the study by Duvall et al. (2007), studies of sibling correlation consistently show that repetitive behaviours are correlated among siblings while impairments in social reciprocity are not (see Szatmari et al. 2006). It is perhaps relevant, in this context, that when linkage analyses are conducted with sub-phenotypes of ASD, it is usually repetitive behaviours for which significant linkage results are reported (Shao et al. 2003). Moreover, Hus et al. (2007) conclude that out of several potential grouping variables for increasing phenotypic homogeneity, only restricted and repetitive behaviours associated with insistence on sameness is independent of age, IQ, and autism severity. One other explanation for the correlation of alexithymia and repetitive behaviours scores could be the already reported association between anxiety and alexithymia and the increased expression of anxiety traits in relatives of autistic probands. More specifically, high alexithymia has been found to be associated with high anxiety sensitivity in a study of undergraduate students (Devine et al., 1999). It is worth noting that this association was stronger with the Difficulty Identifying Feelings sub-scale of the TAS-20. Furthermore, Murphy et al. (2000) reported significantly increased expression of the anxiety trait in relatives of autistic probands compared to relatives of controls (i.e. Down's syndrome probands).

One limitation of this study is the small sub-group size once the analyses focus on mothers or fathers with high alexithymia. A second limitation is the lack of psychometric and clinical data on the children of the control group. Thus, findings from the current study should be interpreted with caution and should be replicated in independent samples. It should also be noted that since this is a correlational study, there is no way to establish a causal link between alexithymia in parents and severity of symptoms in their children. It is conceivable but unlikely that high levels of alexithymia in parents might be a result of raising a child with ASD. In other words, alexithymia in parents might be an outcome rather than a precursor of ASD (an example of reverse causation). We believe that this issue deserves further examination with longitudinal data. Finally, the TAS-20 is a self-report measure and one could argue that individuals that recognize they have difficulty identifying feelings are able to do this better than those who have similar impairments but lack the insight that they do so. In the future, we recommend that one version of the TAS-20 be self-report and another be filled out by an informant familiar with the subject. Even better, an interview version of the TAS-20 is currently being developed and this should provide the most reliable and valid measure of this construct (see Bagby et al. 2006).

Findings from this study suggest that the alexithymia trait appears to be one of the many building blocks that make up the BAP in relatives (i.e. parents) of children with ASD. According to Rutter (2000) it is unclear as to whether the BAP represents a lesser "dose" of genetic liability, a different pattern of susceptibility genes, or some kind of "two-hit" mechanism, in which the BAP is "necessary but not sufficient" to trigger symptoms over the traditional diagnostic threshold for ASD. Careful examination of the BAP composition and boundaries could lead to the discovery of a useful intermediate phenotype of the autism genotype.

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