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## **The psychophysiological mechanisms of Alexithymia in Autism Spectrum Disorder**

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**Abstract**

Accumulating evidence indicates that co-occurring alexithymia underlies several facets of the social-emotional difficulties common in individuals with Autism Spectrum Disorder (ASD). The mechanisms involved, however, remain poorly understood because measuring alexithymia relies heavily on self-report. To address this issue, carefully matched groups of individuals with ASD and comparison participants rated 70 emotion-inducing pictures on subjectively experienced arousal while skin conductance responses were monitored objectively. The results demonstrated reliable correlations between these subjective and objective measures, and in both groups around 25% of individual differences in this correlation (i.e. in emotion-relevant interoception) were accounted for by self-reported alexithymia. In the context of the wider literature, this suggests that alexithymia involves a disruption in how physiological arousal modulates the subjective experience of feelings in those with and without a diagnosis of ASD. Since mindfulness based therapies foster greater awareness of thoughts, feelings and bodily sensations, the findings also have implications for how the symptoms and consequences of alexithymia (e.g., anxiety) might be ameliorated.

**Key Words:** Alexithymia; Autism; Emotion; Arousal; Interoception

## The psychophysiological mechanisms of Alexithymia in ASD

### Introduction

Recent evidence indicates that several facets of the social-emotional difficulties commonly associated with Autism Spectrum Disorder (ASD) are the consequence of co-morbid alexithymia – a difficulty in identifying and describing one’s own emotions that affects an estimated 40-50% of individuals with ASD (Berthoz & Hill, 2005). For example, alexithymia has been shown to account for the difficulties many individuals with ASD experience in fixating the eye-region of faces and in recognising facial, vocal and musical expressions of emotions (Bird & Cook, 2013). Empathic brain responses to the pain of others is also predicted by alexithymia rather than ASD symptoms (Bird, et al., 2010) and in general population samples alexithymia mediates the relationship between sub-clinical autistic traits and certain social reward and empathic processes (Aaron, Benson & Park, 2015). Such findings are important because they resolve inconsistencies in the literature concerning social-emotional processes in ASD by showing that certain difficulties in this domain are not a feature of ASD *per se* but a feature of co-morbid alexithymia (see Bird & Cook, 2013; Brewer, Happé, Cook & Bird, 2015; Gaigg, 2012 for further discussion).

An important caveat of the above literature is that the measurement of alexithymia relies almost exclusively on self-report questionnaires that require participants to reflect on the difficulties they have in reflecting on their own emotions (Vorst & Bermond, 2001). This is not only counterintuitive, it also makes it difficult to specify the mechanisms through which alexithymia impacts social-emotional processes and that could serve as targets for treatment. One possibility is that alexithymia in the context of ASD is a reflection of the broader impairments in mental-

state reasoning that characterise the disorder (Moriguchi et al., 2006). This, however, seems unlikely because alexithymia and mental-state reasoning are subserved by distinct neural mechanisms (Bernhardt, et al., 2013; Silani et al., 2008). The question therefore remains what it is that makes it difficult for those with alexithymia to identify and describe their own emotions.

From the perspective of models that consider the perception of physiological arousal (interoception) to play a central role in the subjective experience of feelings (Craig, 2009), those with alexithymia might either fail to gain awareness of otherwise typical physiological arousal, or demonstrate atypical arousal with consequences for the subjective experience of emotions. Based on evidence that physiological arousal to emotive stimuli is often preserved in ASD, whilst subjectively reported feelings are not (Ben Shalom et al., 2006), the present paper tests the prediction that alexithymia represents an impairment in the awareness of otherwise intact physiological facets of emotional experiences in ASD. If this is true, self-reported alexithymia should predict the degree to which objectively measured physiological arousal is associated with subjective reports of feeling aroused.

## **Methods**

### *Subjects:*

Thirteen adults with ASD (1 female, 12 male) and thirteen typically developing (TD) adults (all male) were recruited from an existing database to achieve groups that were closely matched on chronological age, intellectual functioning (WAIS-III<sup>UK</sup>; The Psychological Corporation, 2000) and critically also alexithymia (BVAQ<sup>1</sup>; Vorst & Bermond, 2001). Care was also taken to ensure

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<sup>1</sup>The 20-item Bermond Vorst Alexitymia Questionnaire (BVAQ) form B was used in this study because it has been validated in previous studies of ASD (Berthoz & Hill, 2005).

that the range of alexithymia within each group was representative of the range generally experienced by individuals with and without ASD. For comparison with Table 1 below, the average BVAQ score in the general population is 41, with scores below 43 thought to indicate the absence, and scores of above 53 the presence, of alexithymia (Deborde et al., 2008). ASD participants were diagnosed according to DSM-IV criteria by experienced clinicians through the UK health services, and administration of the Autism Diagnostic Observation Schedule (ADOS-G; Lord, et al., 2000) confirmed difficulties in reciprocal social and communicative behaviours that are the hallmark clinical feature of the disorder. The Autism-Spectrum Questionnaire (AQ; Baron-Cohen et al., 2001) provided further support for the diagnosis and confirmed that none of the TD participants experienced difficulties commensurate with ASD. All study procedures received ethical approval by the host institutions' ethics committee and all participants provided informed consent in line with the declaration of Helsinki.

### *Materials & Design:*

For the experimental task fifty images were chosen from the International Affective Picture System (IAPS; Lang, Bradley & Cuthbert, 2008)<sup>2</sup> to cover as wide a range as possible on the nominal valence ( $M = 4.59$ ;  $SD = 2.16$ ; Range = 1.51 – 8.03) and arousal ( $M = 5.60$ ;  $SD = 0.96$ ; Range = 3.56 – 7.15) ratings that comprise this stimulus database. An additional twenty images were drawn from various web sites to generate a final pool of 70 images that varied widely in content and emotional salience. Images were presented for 6 seconds each in one of two fixed pseudo-random orders.

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<sup>2</sup> The following images were chosen: 1050, 1160, 1201, 1205, 1300, 1722, 1731, 1947, 1999, 2092, 2095, 2151, 2158, 2811, 3120, 3190, 3302, 3350, 4006, 4008, 4130, 4534, 4538, 4604, 4642, 4677, 4694, 5300, 5725, 5825, 5829, 5910, 6520, 6940, 7033, 8160, 8179, 8485, 9040, 9042, 9043, 9183, 9410, 9412, 9413, 9414, 9440, 9561, 9582, 9599, 9940.

**Table 1***Psychometric characteristics of the ASD and TD groups.*

Measure	ASD ( <i>n</i> = 13)			TD ( <i>n</i> = 13)			Cohen's <i>d</i>
	<i>M</i>	<i>Range</i>	<i>SD</i>	<i>M</i>	<i>Range</i>	<i>SD</i>	
					19.2 –		
Age (years)	38.8	25.3 – 61.6	11.9	40.8	57.5	10.9	0.17
VIQ <sup>a</sup>	108.5	81 – 121	11.3	110.3	88 – 138	15.5	0.13
PIQ <sup>a</sup>	106.2	74 – 131	16.5	103.5	72 – 124	13.6	0.17
FIQ <sup>a</sup>	108.5	81 – 128	14.2	108.0	74 – 135	14.6	0.03
BVAQ Total	47.77	36 – 64	9.08	44.62	34 – 61	7.89	0.37
ADOS							
Com <sup>c</sup>	2.9	1 – 5	1.5	-	-	-	
RSI <sup>c</sup>	6.8	3 – 12	2.9	-	-	-	
Tot <sup>c</sup>	9.6	5 – 17	3.6	-	-	-	
Autism Quotient	37.1	26 – 45	6.4	16.9	4 – 23	6.0	3.3

<sup>a</sup>Verbal (VIQ), Performance (PIQ) and Full-Scale (FIQ) WAIS-III<sup>UK</sup> Intelligence Quotients.

<sup>c</sup>ADOS-G Communication (Com.), Reciprocal Social Interaction (RSI) and Total (Tot.) algorithm scores.

Throughout the experiment, skin conductance responses (SCR) were monitored at 1 kHz using an ADInstruments PowerLab System through stainless steel bipolar SCR electrodes attached to the medial phalanges of the index and ring fingers of the non-dominant hand. LabChart 7

(ADInstruments, 1994-2011) was used for the recording and off-line processing of the data. Following standard procedures (Bradley, Codispoti, Cuthbert & Lang, 2001) SCRs were quantified as the peak response within the six second picture presentation window, applying a log transformation ( $\log[\text{SCR}+1]$ ) to normalise the distribution of the data. It is worth noting that the magnitude and range of SCR responses reported in the current study are comparable to those of previous studies using similar materials and procedures (e.g., Bradley et al., 2001; Lang, Bradley & Cuthbert, 1998).

#### *Procedure:*

Participants rated each of the 70 images on how positive or negative it made them feel (the *valence* dimension) and how intense this emotional experience was (the *arousal* dimension). Instructions emphasised that ratings should reflect the participants' actual feelings at that moment rather than what they thought an appropriate feeling would be. Ratings were given through a keyboard on a 9-point scale, where valence ranged from very negative (1), through neutral (5) to very positive (9) and arousal ranged from calm and peaceful (1) to highly aroused and excited (9). After explaining the task in full participants were asked to give their written consent.

#### **Results**

Prior to testing the principle prediction that self reported alexithymia would predict the degree of association between objectively measured and subjectively reported levels of arousal in both groups, it was important to establish that groups did not differ overall with respect to these dependant variables. In terms of the average responses across all images, there were no group

differences with respect to the arousal ratings (TD:  $M = 5.52$ ,  $SD = 0.63$ ; ASD:  $M = 5.22$ ,  $SD = 1.24$ ;  $t = 0.77$ ,  $df = 24$ ,  $p = .45$ ), valence ratings (TD:  $M = 4.58$ ,  $SD = 0.51$ ; ASD:  $M = 4.43$ ,  $SD = 0.63$ ;  $t = 0.66$ ,  $df = 24$ ,  $p = .51$ ) or SCRs (TD:  $M = 0.089$ ,  $SD = 0.053$ ; ASD:  $M = 0.075$ ,  $SD = 0.081$ ;  $t = 0.54$ ,  $df = 24$ ,  $p = .59$ ). In addition, an item analysis confirmed that the average arousal and valence ratings for each image within the ASD group was highly correlated with the average image ratings of the TD group (valence:  $r = .95$ ,  $p < .001$ ; arousal:  $r = .82$ ,  $p < .001$ ) and average SCRs were also correlated between groups ( $r = .50$ ,  $p < .001$ ). Thus, there was no indication of substantive group differences with respect to the key dependant variables.

Turing to the inter-correlations between variables, in line with previous studies (Bradley et al., 2001) arousal and valence ratings were inversely correlated in both the TD ( $r = -.63$ ,  $p < .001$ ) and ASD groups ( $r = -.62$ ;  $p < .001$ ). Partial correlations, controlling for valence ratings, furthermore confirmed that SCRs were highly associated with arousal ratings in both groups (TD:  $r = .55$ ;  $p < .001$ ; ASD  $r = .51$ ;  $p < .001$ ), demonstrating that SCRs represent a reliable physiological correlate of participants' subjective experience of arousal<sup>3</sup>. Most importantly, as illustrated in Figure 1, individual differences in participants' BVAQ scores were inversely correlated with their overall SCR responses (Figure 1A  $r = -.46$ ;  $p < .05$ ), indicating that alexithymia is associated with reduced levels of peripheral arousal. In addition, however, BVAQ scores were also inversely correlated with individual differences in the association between subjectively reported and objectively measured levels of arousal (Figure 1B;  $r = -.57$ ;  $p < .005$ ), suggesting that alexithymia may also be associated with a reduced awareness of peripheral arousal. To maintain this latter conclusion, however, it is important to rule out the possibility that

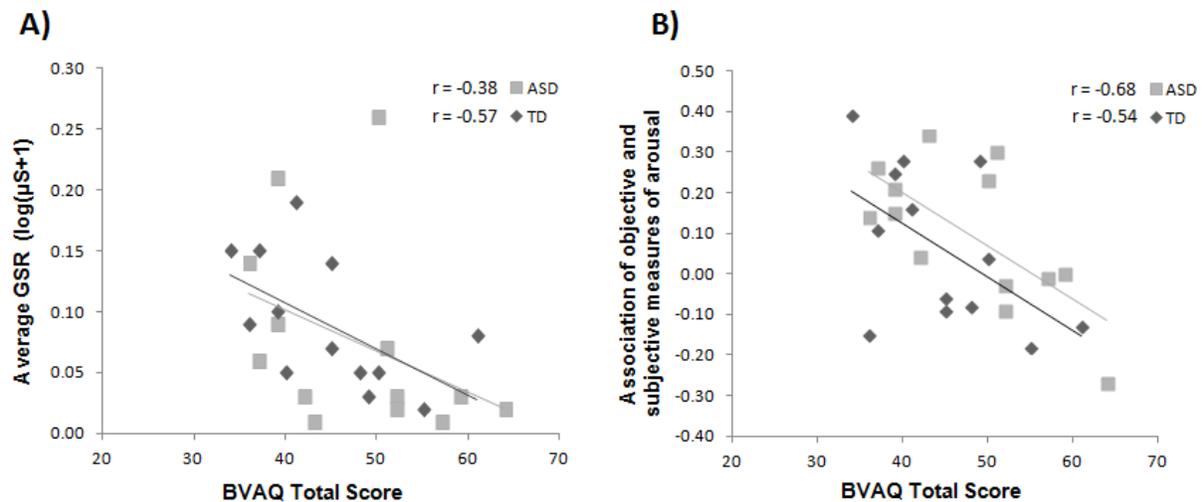
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<sup>3</sup> The 0-order correlations between SCRs and arousal ratings were also significant for the ASD ( $r = .43$ ;  $p < .001$ ) and marginally the TD group ( $r = .23$ ;  $p = .056$ ).

reduced *awareness* of arousal is not simply the consequence of reduced *levels* of arousal and therefore the last correlation analysis was repeated whilst controlling for overall SCR levels. Importantly, the association between BVAQ scores and the association between subjectively reported and objectively measured arousal held ( $r = -.50$ ;  $p < .05$ ). Thus, over and above attenuated levels of arousal, alexithymia is associated with reduced awareness of arousal. Importantly, Fishers  $r$  to  $z$  transformations indicated that there were no significant group differences in any of these correlations ( $z < 0.56$ ;  $p > .58$ ) and power calculations indicated that at least 300 participants would be required in each group for an 80% chance of detecting reliable group differences.

**Figure 1.**

*Associations between the participants' BVAQ alexithymia scores and average SCR responses across all images (A) and between BVAQ alexithymia scores and the magnitude of the correlation between SCR responses and arousal ratings (B).*



## Discussion

The present results make three important contributions to the literature. First, they elucidate the psychophysiological mechanisms that underlie the emotional interoception difficulties of alexithymia. The data show that self-reported difficulties in identifying and describing own emotions are associated with reduced SCR and independently also with a reduced concordance between subjectively reported and objectively measured levels of arousal in both groups. This confirms that at least two distinct processes can contribute to alexithymia – one involving a blunting of emotional experiences (*Type 1 alexithymia*), the other involving reduced awareness and cognitive processing of otherwise preserved emotional experiences (*Type 2 alexithymia*) (see Vorst & Bermond, 2001 for further discussion).

The second contribution is that the present findings confirm that self-report alexithymia questionnaires identify qualitatively very similar emotional interoception difficulties in those with and without a diagnosis of ASD. Specifically, the pattern of correlations between alexithymia questionnaire scores and subjective and objective measures of arousal were very similar in both groups. This is consistent with the observation that self-report levels of alexithymia also predict neural activity in the insula cortex to the same extent in those with and without ASD during emotional interoception and empathy tasks (Silani et al., 2008; Bird et al., 2010). It is important to remember, however, that both in the current and in these previous studies, care was taken to compare ASD and non-ASD samples who were closely matched on self-reported alexithymia. Such sampling ensures that the analysis of within group correlations is not confounded by between group differences in critical measures. However, it also means that samples are not necessarily representative of the populations from which they are drawn because

high levels of alexithymia are rare in the general population but common in ASD. In representative samples we would expect to find significantly lower or no correspondences between subjective and objective measures of arousal in ASD, which is precisely what previous studies have indicated when groups are not purposefully matched on alexithymia (Ben Shalom et al., 2006). We would also expect attenuated levels of arousal in ASD but the evidence here is less consistent (see Gaigg, 2012; Rogers & Ozonoff, 2005), which may reflect a greater prevalence of Type II rather than Type I alexithymia in this population (Berthoz & Hill, 2005).

It is important to acknowledge the relatively small sample size as a possible limitation of the current study. This was, unfortunately, inevitable given a very careful sampling procedure that ensured that groups were closely matched in terms of age, IQ as well as alexithymia, whilst also ensuring a representative range of alexithymia within each group. Importantly, the inclusion of a substantial number of experimental trials and the fact that robust correlations were observed where expected, suggests that the sample size was sufficient for the aims of this study. Moreover, a power analysis suggested that over 300 participants would be needed in each group to observe reliable differences between ASD and TD participants in the correlations of key interest. Although this further suggests that differences between groups are negligible, the present findings should be considered preliminary and further studies are necessary to establish how far the current observations generalise to the broader autism spectrum.

Assuming that the findings do generalise, it is interesting to consider the wider implications for how we might support individuals with ASD who experience co-morbid alexithymia. It is well known that alexithymia negatively impacts psychotherapeutic treatment efficacy (Lumley, Neely

& Burger, 2007), and that many individuals with ASD seek treatment for mental health difficulties such as depression and anxiety (Simonoff et al., 2008). Given that the current observations suggest that alexithymia involves a difficulty in gaining awareness of the body's state of arousal it follows that mindfulness based therapies, which foster greater awareness of inner moment-to-moment experiences of thoughts, feelings and bodily sensations (Hölzel et al., 2011), may prove useful in unlocking the full treatment potential of primary therapies for individuals with ASD and alexithymia. Evidence for a close link between alexithymia and anxiety in ASD (Maisel et al., 2016) and of the impact of mindfulness on alexithymia levels (Baer, et al., 2006) lends support to this suggestion but systematic studies are necessary to thoroughly examine this issue.

### **References:**

- Aaron RG, Benson TL and Park S (2015) Investigating the role of alexithymia on the empathic deficits found in schizotypy and autism spectrum traits. *Personality and Individual Differences* 77: 215-220.
- Allen R, Davis R and Hill E (2013) The effects of autism and alexithymia on physiological and verbal responsiveness to music. *Journal of Autism and Developmental Disorders* 43: 432-44.
- Baer RA, Smith GT, Hopkins J, Krietemeyer J and Toney L (2006) Using self-report assessment methods to explore facets of mindfulness. *Assessment* 13, 27-45.
- Baron-Cohen S, Wheelwright S, Skinner R, Martin J and Clubley E (2001) The Autism-Spectrum Quotient (AQ): evidence from Asperger Syndrome/high-functioning autism,

- males and females, scientists and mathematicians. *Journal of Autism and Developmental Disorders* 31: 5-17.
- Ben Shalom D, Mostofsky SH, Hazlett RL, Goldberg MC, Landa RJ, Faran Y, McLeod DR and Hoehn-Saric R (2006) Normal physiological emotions but differences in expression of conscious feelings in children with high-functioning autism. *Journal of Autism and Developmental Disorders* 36: 395-400.
- Bernhardt BC, Valk SL, Silani G, Bird G, Frith U and Singer T (2013) Selective disruption of sociocognitive structural brain networks in autism and alexithymia. *Cerebral Cortex* 24: 3258-67.
- Berthoz S and Hill EL (2005) The validity of using self-reports to assess emotion regulation abilities in adults with autism spectrum disorder. *European Psychiatry* 20: 291-8.
- Bird G and Cook R (2013) Mixed emotions: the contribution of alexithymia to the emotional symptoms of autism. *Translational Psychiatry* 3: e285.
- Bird G, Silani G, Brindley R, White S, Frith U and Singer T (2010) Empathic brain responses in insula are modulated by levels of alexithymia but not autism. *Brain* 133: 1515-25.
- Bradley MM, Codispoti M, Cuthbert B and Lang PJ (2001) Emotion and Motivation I: Defensive and Appetitive Reactions in Picture Processing. *Emotion* 3: 276-98.
- Brewer R, Happé F, Cook R and Bird G (2015) Commentary on “Autism, oxytocin and interoception.”: Alexithymia, not Autism Spectrum Disorders, is the consequence of interoceptive failure. *Neuroscience and Biobehavioural Reviews* 56: 348-353.
- Craig AD (Bud) (2009) How do you feel – now? The anterior insula and human awareness. *Nature Reviews Neuroscience* 10: 59-70.

- Deborde AS, Berthoz S, Wallier JM, Fermanian J, Falissard B, Jeammet P and Corcos M (2008) The Bermond-Vorst Alexithymia Questionnaire Cutoff Scores: A Study in Eating-Disordered and Control Subjects. *Psychopathology*, 41: 43-49.
- Gaigg SB (2012) The interplay between emotion and cognition in autism spectrum disorder: Implications for developmental theory. *Frontiers in Integrative Neuroscience* 4: 113.
- Hölzel BK, Lazar SW, Gard T, Schuman-Olivier Z, Vago DR and Ott U (2011) How does mindfulness meditation work? Proposing mechanisms of action from a conceptual and neural perspective. *Perspectives on Psychological Science* 6: 537-559.
- Lang PJ, Bradley MM and Cuthbert BN (1998) Emotion, Motivation, and Anxiety: Brain Mechanisms and Psychophysiology, *Biological Psychiatry* 48: 1248-1263.
- Lang PJ, Bradley MM and Cuthbert BN (2008) *International affective picture system (IAPS): Affective ratings of pictures and instruction manual. Technical Report A-8*. University of Florida: Gainesville.
- Lord C, Risi S, Lambrecht L, Cook EH, Leventhal BL, DiLavore PC, Pickles A and Rutter M (2000) The Autism Diagnostic Observation Schedule-Generic: A standard measure of social and communication deficits associated with the spectrum of autism. *Journal of Autism and Developmental Disorders* 30: 205-23.
- Lumley MA, Neely LC and Burger AJ (2007) The assessment of alexithymia in medical settings: Implications for understanding and treating health problems. *Journal of Personality Assessment* 88: 230-46.
- Maisel ME, Stephenson KG, South M, Rodgers J, Freeston, MH and Gaigg SB (2016) Modeling the cognitive mechanisms linking autism symptoms and anxiety in adults. *Journal of Abnormal Psychology*, Advance online publication. <http://dx.doi.org/10.1037/abn0000168>

Moriguchi Y, Ohnishi T, Lane RD, Maeda M, Mori T, Nemoto K, Matsuda H and Komaki G

(2006) Impaired self-awareness and theory of mind: An fMRI study of mentalising in alexithymia. *Neuroimage* 32: 1472-82.

Rogers SJ and Ozonoff S (2005) Annotation: what do we know about sensory dysfunction in

autism? A critical review of the empirical evidence. *Journal of Child Psychology and Psychiatry* 46: 1255-68.

Silani G, Bird G, Brindley R, Singer T, Frith C and Frith U (2008) Levels of emotional

awareness in autism: An fMRI study. *Social Neuroscience*, 3: 97-112.

Simonoff E, Pickles A, Charman T, Chandler S, Loucas T and Bird G (2008) Psychiatric

disorders in children with autism spectrum disorders: Prevalence, Comorbidity, and associated factors in a population-derived sample. *Journal of the American Academy of Child and Adolescent Psychiatry* 47: 921-9.

The Psychological Corporation (2000) *Wechsler Adult Intelligence Scale III UK edition (WAIS*

*III<sup>UK</sup>) (3<sup>rd</sup> ed.)*. The Psychological Corporation: London.

Vorst HCM and Bermond B (2001) Validity and reliability of the Bermond-Vorst Alexithymia

Questionnaire. *Personality and Individual Differences* 30: 413-34.