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Alexithymia, not autism, predicts poor recognition of emotional facial expressions

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ABSTRACT

Considerable research has sought to determine whether face perception is impaired in autism. Clear answers have, however, proved elusive. The present study sought to determine whether comorbid alexithymia (characterized by difficulties interpreting emotional states) may be responsible for face perception deficits previously attributed to autism. Two experiments were conducted to determine the relative contributions of alexithymia and autism to identity and expression recognition using psychophysical procedures. Experiment 1 showed that alexithymia correlates strongly with precision of expression attributions, while autism severity was unrelated to expression recognition ability. Experiment 2 confirmed that alexithymia is not associated with impaired ability to detect expression variation, instead suggesting difficulties interpreting intact sensory descriptions. Neither alexithymia nor autism was associated with biased or imprecise identity attributions. These findings accord with the hypothesis that the 'emotional symptoms' of autism are in fact due to comorbid alexithymia, and that existing diagnostic criteria may need to be revised.

INTRODUCTION

Autism Spectrum Conditions (ASCs) are characterized by abnormalities of social interaction, impaired verbal and non-verbal communication, and a restricted repertoire of interests and activities (APA, 1994). Because of characteristic problems with social interaction, much research has sought to determine whether individuals with autism are impaired in their ability to perceive the most fundamental of all social stimuli: faces. Clear answers have, however, proved surprisingly elusive. Despite the substantial research funding invested, review articles have repeatedly concluded that inconsistency is a consistent feature of the literature on face perception in autism (Harms, Martin, & Wallace, 2010; Simmons et al., 2009; Weigelt, Koldewyn, & Kanwisher, 2012). While several studies suggest that individuals with autism are impaired at recognizing identity from faces (e.g. Boucher & Lewis, 1992; Riby, Doherty-Sneddon, & Bruce, 2009), many others have found no deficit (e.g. Deruelle, Rondan, Gepner, & Tardif, 2004; Ozonoff, Pennington, & Rogers, 1990). An equally incoherent picture has emerged from the study of facial emotion recognition, with different studies finding evidence for (e.g. Ashwin, Chapman, Colle, & Baron-Cohen, 2006; Humphreys, Minshew, Leonard, & Behrmann, 2007) and against (e.g. Adolphs, Sears, & Piven, 2001; Castelli, 2005) an emotion recognition deficit in autism. Indeed, one review recently concluded that "behavioral studies are only slightly more likely to find facial emotion recognition deficits in autism than not" (Harms et al., 2010, p.317).

Several factors have been suggested as potential causes of the inconsistent empirical results. First, the methodology used differs widely across studies (Weigelt et al., 2012). There has been a growing call for the use of more rigorous psychophysical paradigms to be employed, including the use of morph stimuli (Harms et al., 2010) and the modeling of full psychometric functions (Dakin & Frith, 2005). Second, differences in demographic variables such as IQ and age may account for inconsistent results across studies. Some effects may only be evident at a particular range of functioning or at certain developmental stages (Harms et al., 2010). Third, clusters observed within behavioral datasets have prompted some authors to raise the possibility of subgroups within the ASC population (Weigelt et al., 2012). It is this suggestion which forms the focus of the present study. Specifically, we sought to address the possibility that comorbid alexithymia may be responsible for face perception deficits often attributed to ASC.

Trait alexithymia (hereafter ‘alexithymia’) is a subclinical phenomenon characterized by difficulties in recognizing, distinguishing and describing feelings from the bodily sensations of emotional arousal (Nemiah, Freyberger, & Sifneos, 1976). Crucially, while the incidence of alexithymia in the general population is thought to be only 10% (Linden, Wen, & Paulus, 1995; Salminen, Saarijärvi, Äärelä, Toikka, & Kauhanen, 1999), studies suggest severe degrees of alexithymia in at least 50% of individuals with autism (Berthoz & Hill, 2005; Hill, Berthoz, & Frith, 2004; Lombardo, Barnes, Wheelwright, & Baron-Cohen, 2007). Despite this comorbidity, however, alexithymia and autism are fundamentally independent constructs. Alexithymia is neither necessary nor sufficient for an autism diagnosis, while many individuals show severe degrees of alexithymia without demonstrating autistic symptoms.

There is good reason to speculate that comorbid alexithymia may play an important role in understanding face perception deficits in ASC. First, previous research suggests that alexithymia (independent of autism) is associated with impaired recognition of emotional expressions. Although existing studies have employed a variety of methods, a consistent picture has emerged: Greater alexithymia seems to be associated with atypical sorting or classification of emotional facial expressions, particularly those with negative valence (Jessimer & Markham, 1997; Lane et al., 1996; McDonald & Prkachin, 1990; Swart, Kortekaas, & Aleman, 2009). Second, recent findings suggest that several other emotional deficits attributed to autism may instead be due to comorbid alexithymia, including socio-emotional deficits in empathy (Bird et al., 2010), and attention to facial emotion (Bird, Press, & Richardson, 2011). In these studies, the degree of alexithymia, but not autism severity, predicted anterior insula activity when empathizing with the pain of others, and fixations to the eye and mouth area, respectively.

The foregoing results suggest that studies aiming to understand how autism affects face perception, need also to consider the contribution of alexithymia. Here we evaluate the relative contributions of autism and alexithymia to the attribution of facial identity and emotion using rigorous psychophysical methods. In two experiments we compared the performance of an ASC group with a group of alexithymia-matched controls. According to the ‘alexithymia hypothesis’ previous reports of impaired face perception in ASC, in particular deficits of expression recognition, reflect comorbid alexithymia. Consequently, the alexithymia hypothesis predicts no group difference when control groups are matched for alexithymia. Crucially, we therefore

ensured that both the ASC and controls groups contained individuals both with and without alexithymia, allowing us to distinguish the influence of autism and alexithymia. In our first experiment we found that alexithymia and not autism predicted the precision of participants' attributions of emotional expressions. In our second experiment we confirmed that this effect was due to alexithymic individuals' inability to interpret the emotional content of their percept rather than difficulties detecting subtle expression variation.

EXPERIMENT 1

Experiment 1 sought to determine the relative contribution of autism and alexithymia to participants' ability to attribute facial identity and emotion. Stimuli were drawn from morph continua to systematically vary stimulus intensity, and presented according to a 'method-of-constant-stimuli' procedure to estimate participants' psychometric functions for identity and expression attribution.

Methods

Participants: Thirty-two participants completed the experiment, 16 with a clinical diagnosis of ASC (15 males; mean age = 39.2 years) and 16 without (12 males; mean age = 33.4 years). The ASC and control groups did not differ significantly in age [$t(30) = 1.41$, $p > .16$] or gender [$\chi^2(1) = 2.13$, $p > .14$]. All ASC participants received diagnoses of an Autism Spectrum Disorder from an independent clinician. Participants' degree of autism was determined using the Autism Diagnostic Observation Schedule (ADOS-G; Lord et al., 2000). Of the 16 participants, 10 met the criteria for autism, and 5 for autistic spectrum disorders. One participant in the ASC group did not reach the necessary criteria for either of these diagnoses, but reached the criteria for ASC on the Autism Spectrum Quotient (ASQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001). This participant was not an outlier in any analysis, and their exclusion did not alter correlations with ASC severity. Autistic features were assessed in all participants using the ASQ. Full details of the ASC group are provided in Supplementary Table S1. ASQ scores were significantly higher in the ASC group ($M = 33.13$, $SD = 10.09$) than in the control group ($M = 17.88$, $SD = 8.21$) [$t(30) = 4.69$, $p < .001$].

Participants were assessed for alexithymia using the Twenty-Item Toronto Alexithymia Scale (TAS-20; Bagby, Taylor, & Parker, 1994) and the Bermond-Vorst Alexithymia Questionnaire

(BVAQ; Vorst & Bermond, 2001). As expected, scores on these measures were highly correlated (r = .720, p < .001). Because the incidence of alexithymia differs between the ASC and typical populations (Hill et al., 2004), participants were pre-screened using the TAS-20, to ensure equivalent distributions of alexithymia in each group. Of the 32 participants, five in each group met the criteria for alexithymia (TAS-20 score of ≥ 61). TAS-20 scores were used for group matching and in the analyses described below, due to their previous predictive validity (Bird et al., 2011; Bird et al., 2010). Alexithymia levels did not differ between the ASC (M = 55.6, SD = 12.0) and control (M = 46.9, SD = 19.5) groups [$t(30) = 1.51$, $p > .14$]. The IQ of the ASC (M = 121.1, SD = 11.4) and control groups (M = 115.8, SD = 10.3) did not differ significantly [$t(30) = 1.38$, $p > .17$], as measured by the Wechsler Adult Intelligence Scale (Wechsler, 1997) and the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999) respectively.

Stimuli and Procedure: Four morph continua were produced which together constituted two sets of 'cross-morph' stimuli (Figure 1a). Each continuum morphed simultaneously between two expressions (either surprise and fear or disgust and anger) and two identities ('Tracie' and 'Maria' or 'Harold' and 'Felix'). The two cross-morph sets (Disgust-Anger and Surprise-Fear) each comprised two complimentary morph continua. For example, the continuum derived from morphing Harold expressing anger with Felix expressing disgust and the complimentary continuum derived from morphing Harold expressing disgust with Felix expressing anger together comprised the Disgust-Anger cross-morph set. Original greyscale images were taken from Ekman and Friesen (1976) (identities M4, M6, F4 and F5) and were morphed using Morpheus Photo Morpher version 3.11 (Morpheus Software LLC, Indianapolis, USA). All cross-morph stimuli are provided as supplementary materials (Supplementary Figure S1). Surprise was morphed with fear, and disgust with anger, to produce cross-morph sets which emphasized eye- and mouth-region variation respectively.

Figure-1

The experimental program was written in MATLAB with Psychtoolbox (Brainard, 1997; Pelli, 1997). Trials began with a fixation cross (1500ms) and then presented a stimulus drawn from one of the cross-morph sets (800ms). Stimuli were presented for 800ms until replaced by a prompt to attribute either expression or identity. The use of cross-morph stimuli meant that the same

stimulus images could be used to model the psychometric functions for identity and expression attribution. Because i) the same stimuli were used for both attributions and ii) attribution type was interleaved within each block, participants were unaware whether they would be required to attribute emotion or identity during stimulus presentation. Participants therefore needed to attend to sources of identity and expression variance at all times, as is typical when faces are encountered outside the laboratory.

Testing for Experiment 1 consisted of two sessions, one for each cross-morph set. Session order was fully counterbalanced. Sessions comprised 10 blocks of 28 experimental trials. The 14 cross-morph stimuli were presented twice within each block, eliciting each attribution once. Sessions began with an introductory screen showing the two emotions on the two individuals at 80% intensity. These four images were clearly labeled for expression and identity. Thereafter participants completed eight practice trials without feedback. During short breaks between blocks the introductory screen showing the labeled expressions and identities was presented again. Each cross-morph session lasted approximately 25 minutes.

Results and Discussion

The attribution data from Experiment 1 were modeled by fitting cumulative Gaussians to estimate psychometric functions. Function fitting was completed in MATLAB using the Palamedes toolbox (Prins & Kingdom, 2009). Separate functions for each expression and identity dimension were modeled for each participant. Two parameters were estimated: The point of subjective equivalence (PSE) and attribution threshold (Figure 2a). The PSE is a measure of bias and describes the point on the identity or expression dimension where participants are equally likely to make either attribution. The attribution threshold is an index of attribution precision and was inferred from the standard deviation of the Gaussian distribution which best fit the data; lower thresholds indicate better performance.

Consistent with the alexithymia hypothesis, group analyses revealed no differences between the ASC group and the alexithymia-matched control group on any measure of identity or expression attribution (Table 1). To confirm whether the absence of group effects was due to the equivalent levels of alexithymia in the two groups, more detailed analyses of the individual differences were undertaken. ASQ was used as a measure of autism severity, as data were available for all

participants. Its use was validated by the high correlation between the presence of a clinical diagnosis and ASQ (r =.650, p < .001). Simple correlations (Figure 2b) revealed that alexithymia was significantly correlated with the precision of participants' attributions of Disgust-Anger (r = .522, p < .01) and Surprise-Fear (r = .392, p < .05). Autism, however, was not significantly correlated with attribution precision for Disgust-Anger (r = .296, p > .10) or Surprise-Fear (r = .097, p > .50). Neither alexithymia nor autism was correlated with any measure of bias or identity attribution precision (see supplementary Table S2).

Table-1

Despite the significant simple correlation between alexithymia and expression attribution, it is possible that this relationship is not robust once the effects of IQ, gender and age are considered (Harms et al., 2010). Moreover, it is possible that autism accounts for a significant proportion of unique variance once these demographic factors and alexithymia are taken into account. To consider these possibilities, additional hierarchical regression analyses were performed.

Figure-2

The regressions of principal interest model the variance in Disgust-Anger and Surprise-Fear attribution precision (Table 2). Demographic variables (Gender, Age and IQ) were entered into the first step of each model, and alexithymia and autism were entered into the second and third steps respectively. When added to the demographic variables (step 2), alexithymia was a significant predictor of both Disgust-Anger (β = .548, t = 3.27, p < .01) and Surprise-Fear (β = .363, t = 2.07, p < .05) precision. The addition of alexithymia scores significantly improved both models, increasing the variance accounted for by 26.7% [$F(1,27)$ = 10.68, p < .01] and 11.7% [$F(1,27)$ = 4.28, p < .05] in the Disgust-Anger and Surprise-Fear models. In contrast, adding autism (step 3) led to non-significant changes in R^2 of 0.8% and 5.1% in the Disgust-Anger and Surprise-Fear models.

Table-2

The analyses described above suggest that autism accounts for very little variance in expression attribution precision, once alexithymia has been accounted for. However, our autism (ASQ) and alexithymia (TAS-20) measures are correlated (r = .640, p < .001). Consequently, when entered into a multiple regression simultaneously, autism may not be a significant predictor due to multicollinearity. We therefore ran two further hierarchical regressions, again modeling Disgust-Anger and Surprise-Fear attribution precision, but now entering autism in step 2 and alexithymia in step 3. Importantly, when added to the demographic predictors (step 2), autism failed to significantly improve either model, only accounting for an additional 7.0% and 0.2% of the variance in Disgust-Anger and Surprise-Fear precision. Despite the correlation with autism, alexithymia was again a significant predictor of both Disgust-Anger (β = .624, t = 2.82, p < .01) and Surprise-Fear (β = .562, t = 2.50, p < .025) precision when added in step 3. Adding alexithymia led to significant changes in R^2, increasing the variance accounted for by 20.5% [$F(1,26) = 7.96$, p < .01] and 16.6% [$F(1,26) = 6.24$, p < .025] in the Disgust-Anger and Surprise-Fear models.

Together these analyses strongly argue that alexithymia, and not autism, is associated with impaired expression recognition. Autism did not correlate with attribution precision and failed to account for significant variance in the regression analyses. In contrast, alexithymia correlated with expression attribution precision and remained a highly significant predictor, after the influence of demographic variables and autism had been accounted for. Tellingly, this pattern was replicated across both the Disgust-Anger and Surprise-Fear tasks, despite the differing emphasis on eye and mouth variation. However, while it is clear that high alexithymic individuals have difficulties attributing facial emotion, neither Experiment 1 nor previous studies of expression recognition in alexithymia, reveal whether this reflects a problem interpreting an intact sensory description or whether individuals are less able to detect subtle differences between facial expressions. We address this possibility in our second experiment.

EXPERIMENT 2

Experiment 2 sought to determine whether autism or alexithymia are correlated with participants' ability to detect physical differences present in morphed facial stimuli. Participants completed a sequential matching task to estimate their ability to detect the presence of a 20% difference in either identity or expression intensity. Unlike the attribution task employed in Experiment 1,

matching tasks do not require participants to label a percept; simply to decide whether two stimuli are identical. If alexithymia is correlated with detection of expression variation it would argue against a higher-level percept interpretation account.

Methods

Participants: The 32 participants who completed Experiment 1 also completed Experiment 2. The order in which participants completed the experiments was fully counterbalanced.

Stimuli and Procedure: The stimuli used in Experiment 1 morphed simultaneously between different identities and different expressions; therefore participants could distinguish adjacent stimuli based on variation in either expression or identity dimensions. In order to derive separate estimates of ability to detect identity and expression differences, it was necessary to morph expression and identity independently (Figure 1b). Four novel continua were derived from the same face images as those morphed in Experiment 1. Each continuum comprised seven stimuli morphing between 20% and 80% intensities in equidistant intervals of 10% (Supplementary Figure S2).

Experimental trials began with a fixation cross present for 1000ms. Two stimuli drawn from one of the identity or expression continua were then presented sequentially for 800ms each. During an 800ms inter-stimulus-interval a mask was displayed, constructed by phase scrambling one of the morph stimuli. Experiment 2 comprised 200 trials, divided equally into 5 blocks. On 50% of trials, the first and second stimuli were identical. On the remaining 50% the stimuli were two steps apart on the morph continua, representing an inter-stimulus intensity difference of 20%. Participants judged whether or not the two stimuli were ‘same’ or ‘different’ and made key press responses accordingly. Participants took short breaks between blocks to prevent fatigue. Before commencing the experiment participants completed eight practice trials.

Results and Discussion

The data were analyzed by calculating separate *d*-prime statistics (Macmillan & Creelman, 1991) to estimate detection ability on each of the four continua: Disgust-Anger (M = 1.13, SD = .45); Surprise-Fear (M = .36, SD = .52); Harold-Felix (M = 1.61, SD = .78); and Tracie-Maria (M = 1.20, SD = .48). One-sample *t*-tests confirmed that both groups could detect a morph difference

of 20% on all dimensions (all $p < .025$). However, consistent with the alexithymia hypothesis, no significant differences were revealed between the ASC and alexithymia-matched control groups (Table 1b).

As in Experiment 1, correlational and regression analyses were undertaken to compliment the group analyses. Simple correlations revealed no relationships between either autism or alexithymia and the two expression detection measures. Ability to detect Disgust-Anger variation was significantly correlated with IQ ($r = .383$, $p < .05$). Alexithymia was also significantly correlated with detection of Harold-Felix identity variation ($r = -.390$, $p < .05$) but not with detection of Tracie-Maria differences ($r = -.072$, $p > .60$).

The same hierarchical regression analyses used to model expression attribution precision in Experiment 1 were used to model the four detection measures calculated in Experiment 2 (see Supplementary Table S3). IQ continued to significantly predict detection of Disgust-Anger variation when entered with gender and age in step 1 ($\beta = .389$, $t = 2.29$, $p < .05$), but this fell below significance when alexithymia was added to the regression model. Neither alexithymia nor autism was a significant predictor of any of the four measures when the variance accounted for by the demographic variables was taken into account, irrespective of the order in which they were entered into the regression.

Neither alexithymia nor ASC significantly predicted participants' ability to detect physical differences between the morphed facial expressions or identities. These findings suggest that the association between alexithymia and imprecise attribution of expressions observed in Experiment 1 is unlikely to reflect inability to detect physical differences between stimuli. Rather, it appears that severe alexithymia may impair participants' ability to interpret the emotional content of an intact sensory description.

GENERAL DISCUSSION

The present study evaluated the relative contributions of autism and alexithymia to the recognition of facial identity and emotional expressions. Experiment 1 showed that an ASC and an alexithymia-matched control group showed equivalent ability to recognize emotional expressions and identity. Regression analyses revealed that alexithymia, and not autism,

predicted expression attribution precision. Experiment 2 sought to determine whether the influence of alexithymia on expression recognition reflects ability to detect differences between morphed facial stimuli. Neither alexithymia nor autism, however, predicted ability to detect identity or expression variation, after accounting for effects of IQ, gender and age. This second finding suggests that high-alexithymic individuals are able to form an intact sensory description, but thereafter have difficulties interpreting its emotional content. This impairment does not reflect systematic attribution biases for particular emotions; such a tendency would have resulted in correlations between alexithymia severity and PSE estimates. Rather, alexithymia predicts imprecise, but unbiased attributions of emotion.

These results represent a significant step towards disambiguating the inconsistent literature on expression recognition in autism. Where expression recognition deficits have been reported previously (e.g. Ashwin et al., 2006; Humphreys et al., 2007), group differences may reflect greater proportions of severely alexithymic individuals in ASC samples. Due to the higher incidence of alexithymia in the ASC population, ASC samples are likely to contain higher levels of alexithymia than control samples, unless steps are taken to ensure matching. It is of particular interest that, where reported, expression recognition deficits in ASC are often restricted to negative emotions (Harms et al., 2010); a similar pattern to that seen in alexithymia. Those studies which found no evidence of impaired expression recognition (e.g. Adolphs et al., 2001; Castelli, 2005) may have used control samples matched, either explicitly or inadvertently, for alexithymia. This conclusion parallels findings with empathic brain activity (Bird et al., 2010) and gaze fixations to emotional social stimuli (Bird et al., 2011). In both cases, alexithymia was found to be a better predictor than the presence or severity of autism. These findings, together with the present results, suggest that the characterization of autism as a disorder with emotional symptoms (e.g. APA, 1994) may be inappropriate, necessitating the development of novel diagnostic criteria for autism spectrum conditions which do not include emotional impairment.

That alexithymic individuals show atypical patterns of fixations when viewing faces (Bird et al., 2011) may be cited as a potential cause of imprecise expression attribution. However, under this interpretation it is hard to explain why alexithymia does not also predict impaired identity recognition. Instead, we propose the reverse pattern of causality: underlying problems interpreting the emotional expressions of others may give rise to atypical patterns of social gaze

fixations. It is widely thought that those systems responsible for the experience of particular emotions contribute to the recognition of the corresponding emotions in others (Adolphs, Tranel, Damasio, & Damasio, 1994; Calder, Lawrence, & Young, 2001; Calder & Young, 2005). A population with atypical development of (or connectivity with) limbic structures (e.g. amygdala, insula) might therefore be expected to have difficulties interpreting both their own emotions and those of others.

Interestingly, we found no relationship between ability to discriminate or attribute identity and the presence of autism. Moreover, neither identity attribution (Experiment 1) nor detection (Experiment 2) was predicted by alexithymia once the effects of IQ, gender and age were accounted for. These findings are consistent with a recent review, which concluded that identity deficits are most likely to be seen in ASC groups when face stimuli remain unfamiliar and experimental paradigms place a demand on short-term perceptual memory for faces (Weigelt et al., 2012). Both of our paradigms repeatedly presented stimuli derived from the same four individuals, and therefore gave participants opportunity to learn these identities. Moreover, the use of a single-stimulus procedure used in Experiment 1 minimized the perceptual memory load. It remains to be seen whether the alexithymia hypothesis proves useful in understanding apparent face memory deficits in autism (e.g. Boucher & Lewis, 1992).

Having employed rigorous psychophysical methods, we found that degree of alexithymia, and not autism, was predictive of expression attribution precision. These results go a long way toward disambiguating the equivocal literature on expression recognition in autism. Specifically, expression recognition deficits in ASC samples may only be seen when ASC and control groups are not matched for alexithymia. Our results suggest that matching ASC and control groups for alexithymia should be adopted as routine practice by researchers studying emotional processing in autism. The present findings also reaffirm the clinical and theoretical significance of alexithymia. These results add to the growing literature suggesting that a higher incidence of alexithymia within the population of individuals with autism, rather than autism *per se*, may be responsible for the emotional impairments currently considered a feature of autism. Developing a more sophisticated understanding of this intriguing condition should be a priority for cognitive scientists.

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FIGURES LEGENDS:

Figure 1:

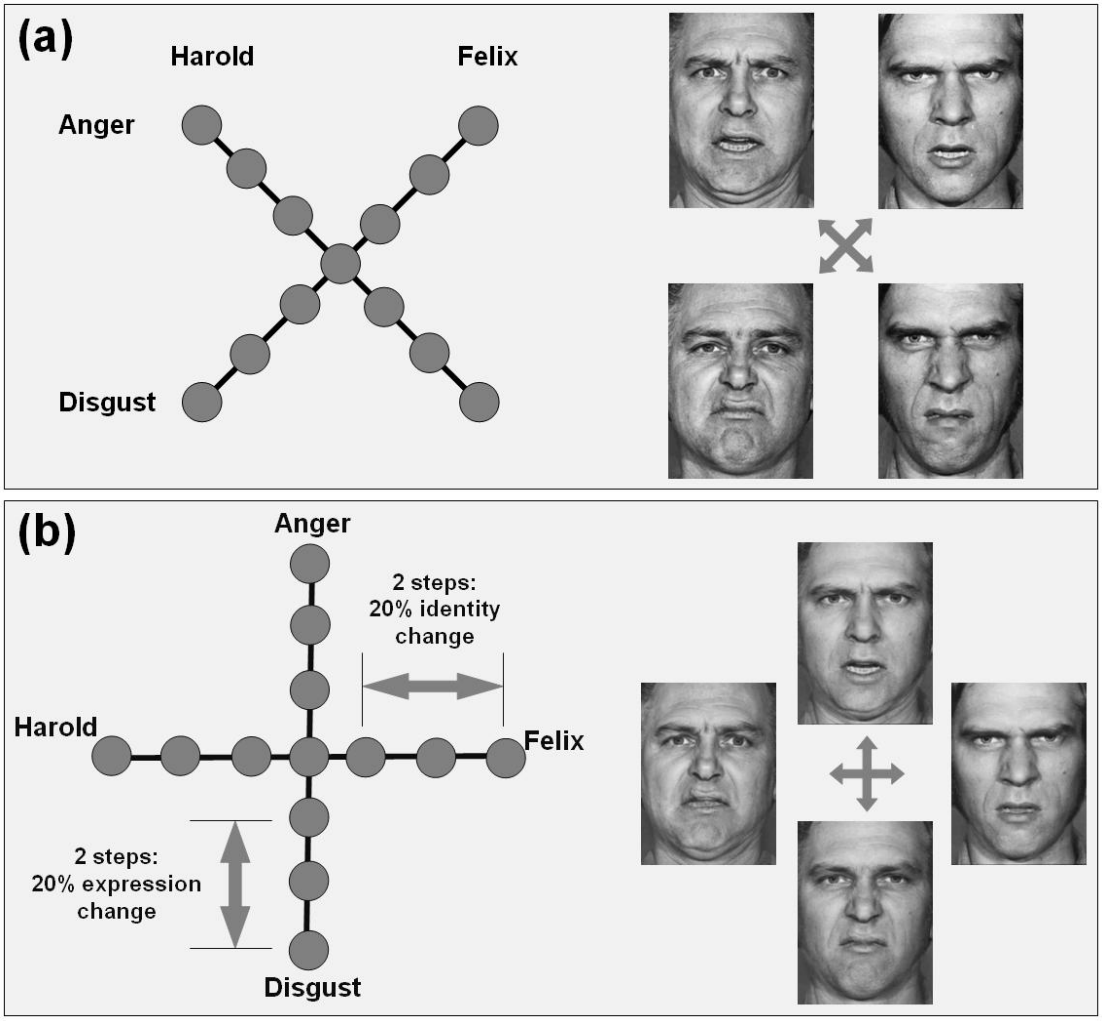

Figure 1: (a) Schematic illustrations of the cross-morph continua (left) and the end points of each continuum (right). These two identities were arbitrarily assigned the names 'Harold' and 'Felix' for purposes of the attribution judgments. Each 'arm' of a cross-morph set comprised seven morph stimuli which varied in stimulus intensity between 20% and 80% of each attribute in equidistant 10% increments. (b) Schematic illustration of the morph continua used in Experiment 2 (left) and the continua end points (right).

Figure 2:

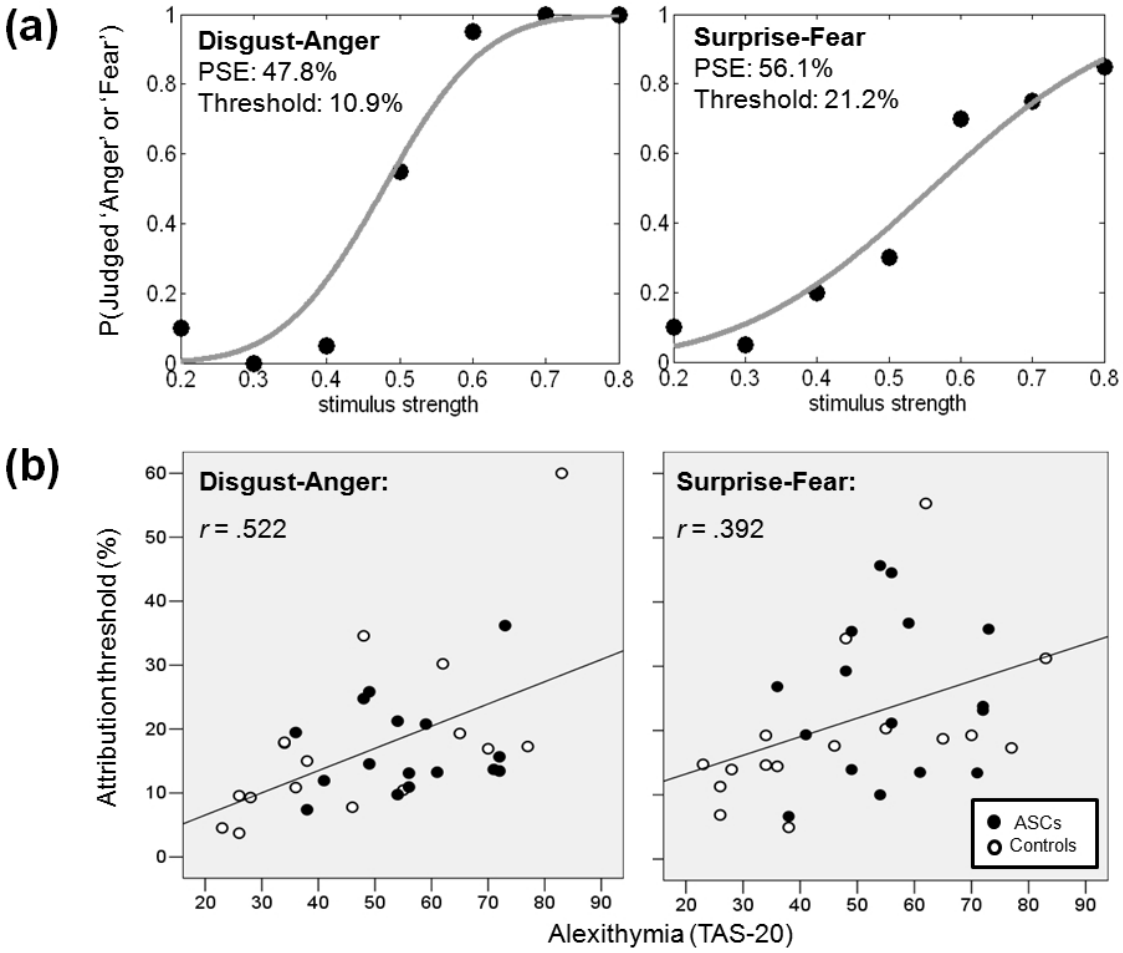

Figure 2: (a) Examples of the psychometric functions modeled by fitting cumulative Gaussian functions. This participant demonstrated more precise attributions of Disgust-Anger relative to Surprise-Fear as indicated by the steeper slope of the function. Attribution threshold is the standard deviation of the Gaussian distribution that best modeled participants' responses. The wider the Gaussian, the less precise participants' attributions; lower thresholds therefore indicate better performance. This participant's Surprise-Fear attributions were both less precise and subject to greater bias than their Disgust-Anger attributions. A tendency to attribute 'Surprise' meant that a stimulus had to contain 56.1% Fear to be equally likely to be judged Surprise or Fear. (b) Simple correlations between scores on the Twenty-Item Toronto Alexithymia Scale (TAS-20) and participants' expression attribution ability. A greater score on the TAS-20 indicates the presence of more severe alexithymia. Significant Disgust-Anger correlations were observed both with (r = .522; p < .001) and without (r = .413; p < .025) the outlying threshold evident in the left panel.

TABLES:

Table 1: Summary of the group analyses conducted during Experiment 1 and Experiment 2. The *t*-statistics and associated probabilities are reported for independent samples t-tests. Consistent with the alexithymia hypothesis, no differences were observed between the ASC and alexithymia-matched control groups in either experiment.

Face Perception Measure	*ASCs Mean (SD)*	*Controls Mean (SD)*	*t*	*P*
(a) Experiment 1				
Disgust-Anger bias	.50 *(.05)*	.52 *(.06)*	1.253	.220
Surprise-Fear bias	.54 *(.11)*	.55 *(.07)*	.285	.777
Disgust-Anger precision	.17 *(.07)*	.18 *(.14)*	.210	.835
Surprise-Fear precision	.25 *(.12)*	.20 *(.12)*	-1.242	.224
Harold-Felix bias	.54 *(.07)*	.53 *(.04)*	-.336	.739
Tracie-Maria bias	.52 *(.05)*	.53 *(.07)*	.376	.717
Harold-Felix precision	.16 *(.13)*	.09 *(.04)*	-1.983	.063
Tracie-Maria precision	.18 *(.16)*	.12 *(.03)*	1.341	.199
(b) Experiment 2				
Disgust-Anger detection	1.08 *(.48)*	1.18 *(.44)*	.614	.544
Surprise-Fear detection	.38 *(.59)*	.35 *(.46)*	-.132	.896
Harold-Felix detection	1.50 *(.77)*	1.72 *(.80)*	.812	.423
Tracie-Maria detection	1.18 *(.52)*	1.22 *(.45)*	.181	.858

Table 2: Summaries of the hierarchical regressions used to model Disgust-Anger and Surprise-Fear attribution precision. Alexithymia was, and Autism was not, a significant predictor of expression attribution precision, irrespective of the order they were entered into the regression model. Cook's distance (cut-off > 1) and leverage (cut-off > .5) statistics were calculated in order to ensure these results were not dependent on outliers. None of the data points, however, showed a value greater than .2 (Cook's distance) or .4 (leverage value). * denotes significance at p <.05; ** denotes significance at p <.001.

		Disgust-Anger Precision			**Surprise-Fear Precision**		
Step	**Predictor**	**β**	**R^2**	**ΔR^2**	**β**	**R^2**	**ΔR^2**
Step 1	Age	.053	5.7%	5.7%	-.051	14.1%	14.1%
	Gender	-.125			-.229		
	IQ	-.186			-.294		
Step 2	Age	.123	32.4%	**26.7%****	-.005	25.9%	**11.7%***
	Gender	-.173			-.226		
	IQ	-.027			-.189		
	Alexithymia	**.548****			**.363***		
Step 3	Age	.136	33.2%	0.8%	.030	30.9%	5.1%
	Gender	-.180			-.278		
	IQ	-.024			-.181		
	Alexithymia	**.624****			**.562***		
	Autism	-.115			-.297		

SUPPLEMENTARY FIGURES:

Figure S1:

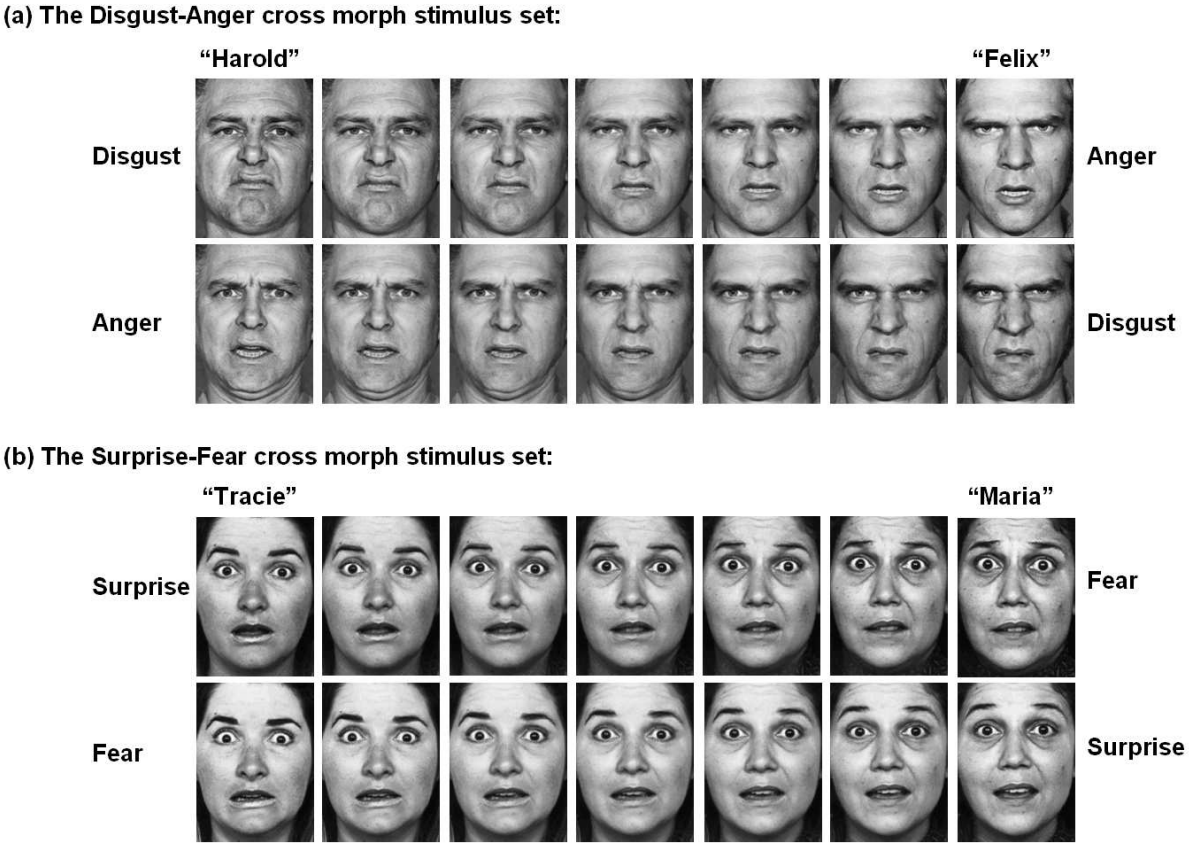

Figure S1: The sets of cross-morph stimuli used in Experiment 1.

Figure S2:

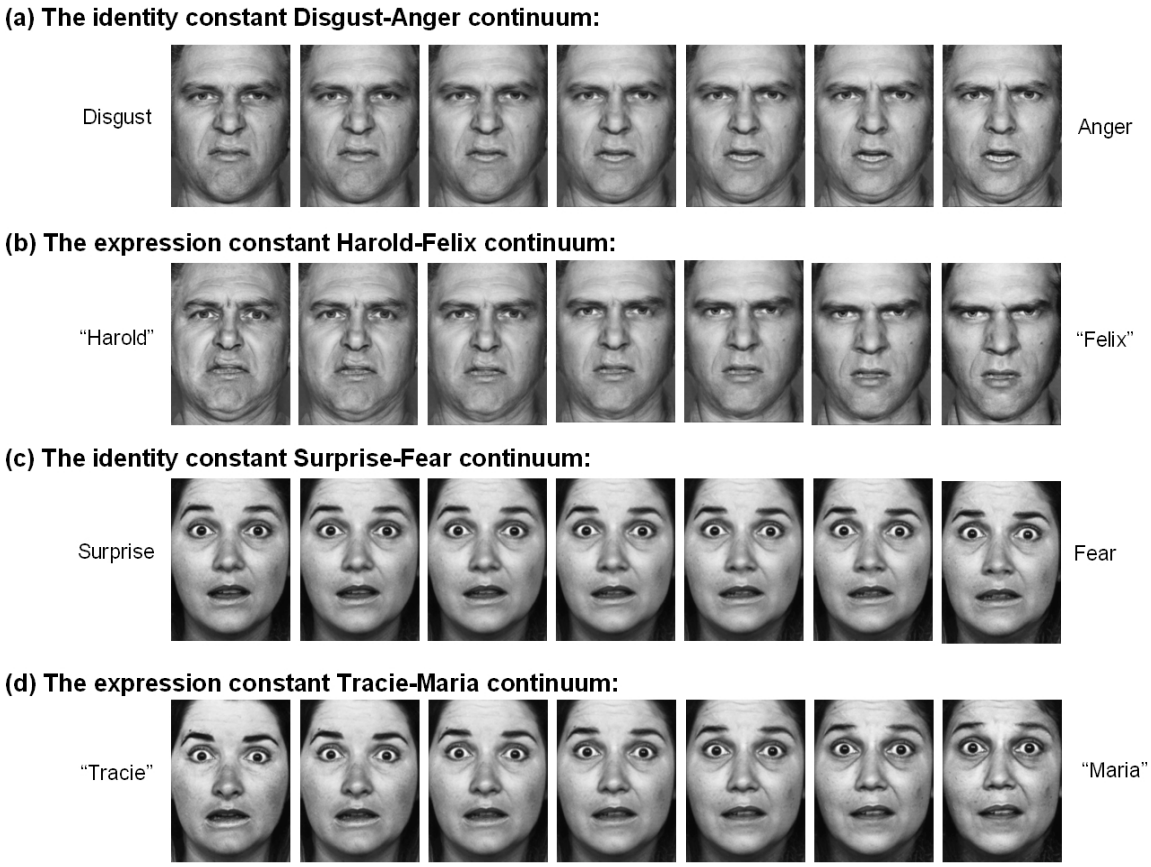

Figure S2: The morph stimuli used in Experiment 2

SUPPLEMENTARY TABLES:

Table S1: ADOS classification, Autism Diagnostic Observational Schedule (ADOS), Toronto Alexithymia Scale (TAS-20), Autism Spectrum Quotient (ASQ), and IQ scores for the ASC group. The IQ of ASC participants was assessed with the Wechsler Adult Intelligence Scale (WAIS). The IQ of the control participants was assessed using the Wechsler Abbreviated Scale of Intelligence (WASI-I).

Participant	ADOS Classification	ADOS Total[a] (*cut-off ≥ 7*)	ASQ	TAS-20[b] (*cut-off ≥ 61*)	Full-scale IQ
1	Autism	10	28	59	125
2	Autism Spectrum	7	46	61	132
3	Autism Spectrum	9	41	72	103
4	Autism Spectrum	8	31	72	108
5	Autism	10	38	56	124
6	Autism	7	27	48	125
7	Autism	10	26	54	102
8	Autism Spectrum	7	42	71	116
9	None	6	37	73	127
10	Autism	11	37	38	118
11	Autism	15	29	41	118
12	Autism	10	42	54	112
13	Autism Spectrum	7	36	49	124
14	Autism	11	19	56	105
15	Autism	15	18	49	97
16	Autism	11	33	36	117

Note. [a]ADOS-G scores are derived from a diagnostic algorithm (Lord et al., 2000) and represent the behavior of the participant at the time of the study. Higher scores represent a higher degree of autism. [b]TAS-20 scores represent the degree of alexithymia, with higher scores indicating more severe alexithymia.

Table S2: Simple correlations between the predictors and the measures of identity and expression recognition estimated in Experiment 1. Alexithymia was correlated with the precision of attribution on both expression continua. Autism failed to correlate significantly with any of the measures of expression or identity attribution. * denotes significance at $p < .05$; ** denotes significance at $p < .001$.

	Disgust-Anger		Surprise-Fear		Harold-Felix		Tracie-Maria	
	Precision	Bias	Precision	Bias	Precision	Bias	Precision	Bias
Age	.077	-.312	-.008	-.057	.080	-.159	.039	.101
IQ	-.191	.238	-.300	.343	-.262	-.153	.048	.125
Gender	-.139	-.024	-.229	.178	-.113	.135	-.065	.289
Autism	.296	.011	.097	-.164	.106	.149	.092	-.323
Alexithymia	**.522****	.183	**.392***	-.009	.271	.062	.279	-.217

Table S3: (a) Summaries of the hierarchical regressions used to model ability to detect differences in the Disgust-Anger and Surprise-Fear expression continua. (b) Summaries of the hierarchical regressions used to model ability to detect differences in the Harold-Felix and Tracie-Maria identity continua. * denotes significance at $p < .05$.

(a)

		Disgust-Anger Detection			Surprise-Fear Detection		
Step	**Predictor**	**β**	**R^2**	**ΔR^2**	**β**	**R^2**	**ΔR^2**
Step 1	Age	.069	.192	.192	-.087	.029	.029
	Gender	-.191			.078		
	IQ	**.389***			-.120		
Step 2	Age	.056	.201	.009	-.113	.066	.036
	Gender	-.182			.095		
	IQ	.360			-.178		
	Alexithymia	-.101			-.202		
Step 3	Age	.044	.208	.007	-.144	.107	.042
	Gender	-.176			.112		
	IQ	.357			-.186		
	Alexithymia	-.174			-.382		
	Autism	.108			.270		

(b)

		Harold-Felix Detection			Tracie-Maria Detection		
Step	**Predictor**	**β**	**R^2**	**ΔR^2**	**β**	**R^2**	**ΔR^2**
Step 1	Age	-.096	.064	.064	-.046	.026	.026
	Gender	-.053			-.048		
	IQ	.232			.151		
Step 2	Age	-.143	.188	.123	-.050	.027	.001
	Gender	-.021			-.045		
	IQ	.124			.142		
	Alexithymia	-.372			-.034		
Step 3	Age	-.174	.227	.039	-.051	.028	.000
	Gender	-.005			-.045		
	IQ	.117			.141		
	Alexithymia	**-.574***			-.039		
	Autism	.262			.008		