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Emotional Modulation of Perception in Asperger's Syndrome

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Abstract Using an attentional blink paradigm, we show that the typical enhancement of perception for emotionally arousing events is significantly reduced in Asperger's syndrome (AS) at short inter-target intervals. Control experiments demonstrate that this finding cannot be attributed to differences in the perceived arousal of the stimuli, or to a global impairment affecting any type of modulation of perceptual encoding. Because a functioning amygdala is critical for emotional modulation of the attentional blink, the findings support a role for the amygdala in the pathophysiology of AS. More specifically, they suggest there is a fundamental failure of the amygdala to modulate processing in cortex, a concept at the heart of some recent theories of amygdala involvement in the aetiology of autistic-spectrum disorders.

Keywords Social cognition · Amygdala · Autism · Fear · Attentional blink · Emotion

Introduction

Deficits in social reciprocity and communication are defining features of autistic-spectrum disorders (ASD), including Asperger's syndrome (American Psychiatry Association 1994). For example, individuals with an ASD often have significant difficulty with social interactions; they tend to be unaware of social norms and struggle to interpret social cues (Volkmar et al. 1994). In the laboratory, impairments are apparent on numerous tests designed to tax social perceptive and social cognitive abilities. Examples include problems recognising basic (Boraston et al. 2007; Howard et al. 2000; Pelphrey et al. 2002) and complex (Baron-Cohen et al. 2001b; Baron-Cohen et al. 1997) emotions from faces and eyes, as well as more abstract reasoning about others' thoughts, beliefs and desires (e.g., Happe 1994; Castelli et al. 2002). An associated finding is that those with an ASD often fail to attend to the most socially meaningful parts of the environment, such as the eyes in faces (Klin et al. 2002; Dalton et al. 2005; Spezio et al. 2006; Corden et al. 2007).

There has been much work attempting to uncover the neuroanatomical substrates of autistic symptoms (Volkmar et al. 2004). One area which has received considerable attention is the amygdala, probably due to its established role in emotional processing and learning (for review, see Phelps and Le Doux 2005). Damage to the amygdala can cause a number of social perceptual and social cognitive deficits that are highly reminiscent of those seen in ASD. Such patients experience problems recognising complex emotions from the eyes (Adolphs et al. 2002), they fail to spontaneously anthropomorphise cartoons of geometrical shapes (Heberlein and Adolphs 2004) and they show reduced attention towards the eyes in faces (Adolphs et al. 2005). Greater performances on tests of fear recognition and theory of mind ability have been associated with greater amygdala activity in normal individuals (Corden et al. 2006). Also using neuroimaging, both Critchley et al. (2000) and Baron-Cohen et al. (1999) show reduced amygdala activity in ASD individuals when processing emotions from faces. Furthermore, a number of structural MRI studies have found amygdala abnormalities in ASD

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(Aylward et al. 1999; Abell et al. 1999; Rojas et al. 2004; Pierce et al. 2001; Nacewicz et al. 2006; Munson et al. 2006), although there have also been a number of negative findings (e.g., Dziobek et al. 2006; Palmen et al. 2006; Haznedar et al. 2000).

Many theorists argue that, in the typically developed brain, the amygdala functions as a 'salience' detector, monitoring the environment for emotionally or socially significant events. It then responds to these events by influencing both current and future behaviour through modulation of other brain areas, such as the hippocampus, the fusiform gyrus and the superior temporal gyrus (for reviews, see Sander et al. 2003; Dolan 2002). Some theories propose that functional abnormalities in the integrity of this system could be a primary cause of social-perceptual impairment in ASD (Grelotti et al. 2002; Schultz 2005; Schultz et al. 2000). For example, Schultz (2005) argues that failure of the amygdala to flag faces as emotionally salient, and to modulate cortical processing of these stimuli appropriately, could cause chronic inattention to faces, contributing over time to the numerous face processing impairments which are so pervasive in ASD. However, few studies have yet explicitly investigated the effect of emotional or social arousal on the modulation of perception in ASD.

A potential method for testing the hypothesis that cortical modulation of emotionally arousing perceptions could be abnormal in people with AS utilises the phenomenon of the emotional modulation of the attentional blink (Anderson 2005). When two masked targets (designated as T1 and T2) are presented within approximately 500 ms of each other, subjects are often unable to report the second of the two targets accurately, even though the first has been reported correctly (Raymond et al. 1992). Allocating attention to T1 is thought to leave less attention available for T2, rendering T2 vulnerable to decay or substitution—hence the transient 'blink' in attention (for review, see Shapiro et al. 1997). However, the size of the attentional blink is greatly attenuated if T2 stimuli are emotionally charged (Anderson and Phelps 2001; Anderson 2005; Ogawa and Suzuki 2004; Arend and Botella 2002). This affective modulation of the attentional blink is most evident when T1 and T2 are close together, which is when attentional resources are most occupied with the processing of the preceding T1. Further experiments have shown that it is the *arousing* quality of the emotional stimuli, rather than their valence, which enhances the ability of T2 to be detected (Anderson 2005).

Furthermore, consistent with its role in enhancing perception for emotional events, amygdala integrity is crucial for emotional modulation of the attentional blink. Patients with bilateral amygdala damage show no significant perceptual benefit for arousing T2 stimuli, despite unaffected comprehension of their affective quality (Anderson et al. 2001). These deficits are most marked when T2 occurs shortly after T1—i.e., when attention is most stretched.

The emotional modulation of the attentional blink therefore provides a paradigm for investigating the integrity of the cognitive system which serves to enhance perception for biologically salient events. Furthermore, it gives an indication of amygdala functioning, specifically in relation to its role as a 'salience detector' and modulator of cortical processing. In this manner, the paradigm offers the chance to test predictions from models of compromised amygdala function in ASD, in particular those that propose a failure in the salience detection system (e.g., Grelotti et al. 2002; Schultz 2005; Schultz et al. 2000). Therefore, using a sample of high-functioning adults with Asperger's syndrome (AS) and matched controls, we aimed to test the following predictions:

(1) Like patients with amygdala damage, individuals with AS will show reduced enhancement of perception for emotionally arousing events in an attentional blink paradigm, compared to matched controls.
(2) This difference will be most apparent when the lag between T1 and T2 is short (<500 ms)—i.e., when attention is most stretched and normal controls typically show the greatest perceptual benefit for arousing stimuli presented as T2 stimuli.
(3) Within the AS group, a larger (i.e., more typical) perceptual benefit for emotionally arousing stimuli will predict less severe autistic symptoms (measured via the autism diagnostic observation schedule) and/or less impaired social perceptual ability (measured by means of a test of facial fear recognition).
(4) Any measured diminution of perceptual benefit to AS individuals of emotionally salient stimuli, presented as T2, cannot be attributed to differences between them and normal controls in the degree to which their consider the stimuli to be arousing, to their attributions of valence, or to a global impairment affecting modulation of perceptual encoding at short inter-target intervals.

Experiment 1

Methods

Participants

A total of 17 adults with a clinical diagnosis of AS were recruited from local ASD support groups and via advertisement in the National Autistic Society newsletter. AS

diagnosis was from a UK psychiatrist or psychologist and was independently confirmed via the autism diagnostic observation schedule (ADOS, Lord et al. 2000); to be included, an individual had to score above cut-off for an autistic-spectrum disorder in terms of both reciprocal social interaction and communication subscales. Ratings of behaviour from a parental diagnostic interview, such as the ADI-R (Lord et al. 1994) or the 3Di (Skuse et al. 2004), was not possible because, of the participants' age (see Table 1); in most cases no reliable parental informant was available. AS subjects were excluded if they had a full-scale IQ < 90 or if they suffered from any neurological or psychological disorder not normally associated with their condition. About 17 gender-matched, healthy controls were also recruited. Age and IQ were matched as closely as possible (see Table 1). All subjects gave written informed consent prior to their inclusion in the study.

Design and Procedure

The experiment was in two parts, the first being the attentional blink experiment itself. The second, which took place at least 1 week later, measured:

(a) subjects' arousal responses to the T2 words, both via self-report and skin conductance responses (SCRs).
(b) subjects' ability to recognise fearful faces. This was intended as a measure of social perceptual skills and was chosen in particular because of its association with amygdala function (e.g., Adolphs et al. 1994; Morris et al. 1996).

Details of the design and procedure for these sections are given separately below.

Emotional modulation of the attentional blink experiment: The first set of target stimuli (T1) comprised 38 neutral words. The second target stimuli (T2) consisted of 19 negative-arousing words (e.g., rape, bastard) and 19 neutral words (e.g., basin, sparrow). The arousing words, with negative valence, were the same as those used in previous experiments, where they have been shown to produce a large attenuation of the attentional blink in normal subjects (Anderson et al. 2001; Anderson 2005). The neutral words were taken from the ANEW 'Balanced Affective Word List' (http://www.sci.sdsu.edu/CAL/wordlist/) and were chosen to have low ratings for arousal and neutral valence. The negative and neutral word lists were matched for average word length and semantic relatedness. Distractor stimuli were 326 neutral words, again taken from the ANEW list, matched in length with the target words.

Each trial consisted of 15 words—2 targets and 13 distractors (see Fig. 1). The subjects' task was to monitor the stream of words and to report the identity of the two target words (T1 and T2) by writing them down at the end of the stimulus sequence. These were designated as targets by appearing in black, whereas the distractor words appeared in a random selection of 4 colours (yellow, red, green and blue; the same colour was not allowed to appear twice in a row)—see Fig. 1. Each item in the stream was presented for 120 ms and was immediately followed by the subsequent item. A random number of distractors (between 3 and 6) appeared before T1. T2 was presented at one of four possible times after T1—120 ms (no intervening items), 360 ms (two intervening items), 600 ms (four intervening items) or 840 ms (six intervening items). The shorter two of these four T1–T2 lags lie within the time window during which T2 is susceptible to an attentional blink, whereas the later two lie beyond this window (Shapiro et al. 1997). There were 19 trials for each factor combination of lag (1–4) and word type (neutral versus arousing), resulting in 152 trials. Trials where subjects failed to correctly identify T1 were excluded from analysis (this occurred on <5% of cases). The dependent measure was the number of T2 stimuli correctly identified.

Arousal measurements: To assess perceived arousal to the T2 stimuli, subjects viewed all 38 T2 words (19 neutral and 19 arousing) sequentially on a computer screen (Geneva font, 30 point, viewing distance approximately 40 cm), whilst their SCRs were recorded. Each word was shown for 4.5 s. During this time subjects were instructed to read the word silently but to remain still. Following the presentation of each word, there was a blank screen for 0.5 s followed by the question, "How emotionally arousing did you find that word?" This was the cue for subjects to rate the arousal quality of the word on a scale of 1–5 via a keyboard press. On this scale, '1' represented, 'not at all arousing' and '5' represented, 'very arousing'. There was

Table 1 Group characteristics

	Age (years)	Gender	Verbal IQ	Non-verbal IQ	Full IQ	ADOS Total
AS Mean (SD)	34.2 (11.99)	15 M, 2 F	109.4 (14.27)	112.1 (15.25)	112.9 (13.99)	11.3 (3.89)
Controls Mean (SD)	32.3 (13.26)	15 M, 2 F	108.7 (10.99)	108.7 (8.18)	109.9 (8.57)	NA

The groups did not differ significantly on any of these variables—all $t < .81$, all $p > .42$

ADOS = Autism Diagnostic Observation Schedule (Lord et al. 2000)

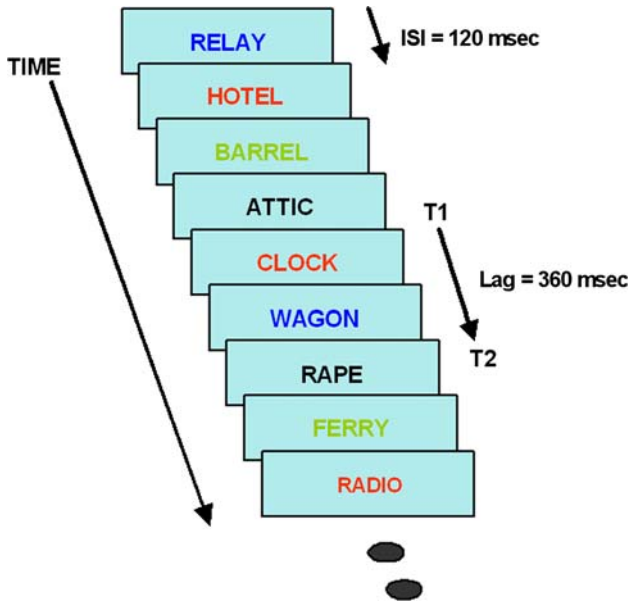

Fig. 1 Diagram of the attentional blink task. Words were briefly presented sequentially in an identical central location and observers were instructed to ignore the coloured words (distractors) and indicate the identity of two target words appearing in black. The temporal lag between the first (T1) and second target (T2) was varied. We used two short lags (120 or 360 ms), during which T2 is susceptible to the attentional blink, and two long lags (600 and 840 ms) at which T2 is less susceptible to the attentional blink. The emotional quality of the T2 stimuli was also varied—half the T2s were 'arousing', as in the example above, and half were 'neutral'. The dependent measure was the percentage of T2s correctly identified

an unlimited time to answer. To allow the skin conductance trace to return to baseline, there was a gap of at least 16 s between words, during which time a blank screen was presented.

SCRs were collected via a pair of silver-silver chloride electrodes, approximately 0.8 cm^2 in contact area and filled with 0.05 M sodium chloride gel, placed on the volar surfaces of the distal phalanges of digits II and III of the non-dominant hand after the skin was wiped with an alcohol swab. Data were recorded and analysed using a system provided by Psylab (http://www.psylab.com). MatLab (Mathworks Inc., Nantick MA) interfaced with the Psylab system via the parallel port, allowing synchronisation of SCR recording with the experimental paradigm. A SCR was considered to be in response to a word if it began between 1 and 3 s post stimulus onset (Dawson et al. 2000). Only the first response occurring in this time window was analysed. The amplitude of each SCR was recorded and the mean SCR amplitude was calculated for each word type (neutral or arousing), separately for each group.

Fear recognition ability: The Ekman–Friesen Test of Facial Affect Recognition (Ekman and Friesen 1976) was administered. Subjects viewed 60 halftone photographs, 10 exemplars of each of the 6 basic emotions (happiness, sadness, fear, surprise, anger and disgust). For each face, subjects were presented with the six emotion labels and were told to "pick the one that best fits what you think the person is feeling". Before beginning the experiment, subjects completed 6 practice examples, one for each emotion. These items were included merely to familiarise participants with the experimental set-up and task, therefore feedback was not given on these items, the stimuli were not used in the subsequent test and responses to them were not analysed. For the purposes of this paper, the dependent variable was simply the number of fearful faces correctly identified.

The study was approved by the Institute of Child Health and Great Ormond Street Hospital COREC ethics committee and was therefore performed in accordance with the 1964 Declaration of Helsinki.

Results

Self-reported Arousal in Response to the Words

Table 2 summarises how the groups rated the words in terms of emotional arousal. These data were analysed via analysis of variance (ANOVA). There was a highly significant effect of word type, $F(1,32) = 70.5$, $p < .001$, suggesting that the participants did indeed find the arousing words more emotionally salient than the neutral words. However, there was no effect of group, $F(1,32) = 1.6$, $p = .22$ and no significant interaction, $F(1,32) = .09$, $p = .76$, suggesting that the two groups were comparable in terms of how emotionally arousing they found the words.

Autonomic Measure of Arousal in Response to the Words

Table 3 shows the mean amplitude of the SCRs made to the words. These data were normalized via a square root transformation and then analysed via ANOVA. There were significant effects of word type, $F(1,32) = 49.8$, $p < .001$, and group, $F(1,32) = 4.5$, $p = .04$. Importantly, however,

Table 2 Summary of how the two groups rated the arousal quality of T2 words

Word type	Group	Mean	*SD*
Neutral words	Controls	2.0	.73
	AS	1.8	.63
Arousing words	Controls	3.6	.78
	AS	3.3	1.16

Subjects rated each word on a scale of 1–5, where 1 was 'not at all arousing' and 5 was 'very arousing'

Table 3 Mean SCR magnitudes to the words, split by group

Word type	Group	Mean	*SD*
Neutral words	Controls	.12	.107
	AS	.22	.082
Arousing words	Controls	.29	.148
	AS	.35	.135

Data are in $\sqrt{\mu}$ Siemens

there was no evidence of a group × word type interaction, $F(1,32) = 0.73$, $p = .4$. This suggests that, while the AS subjects had a higher autonomic reaction to the words in general, the groups were comparable in terms of how they differentiated between the neutral and arousing words.

Emotional Modulation of the Attentional Blink

Figure 2 summarises the results of the attentional blink experiment. These data were entered into an ANOVA with word type and lag as repeated measures variables and group as a between-measures variable.

As predicted, there were highly significant effects of lag, $F(3,96) = 30.9$, $p < .001$, and word type, $F(1,32) = 23.2$, $p < .001$. Post-hoc comparisons show that significantly more T2 words were identified for lags 3 and 4 compared to both lags 1 and 2 (all $p < .001$, Bonferroni corrected). Subjects correctly identified more arousing than neutral T2s at every lag (all $t > 3.1$, all $p < .012$, Bonferroni corrected). However, there was a significant word type x lag interaction—$F(1,32) = 23.2$, $p < .001$, reflecting the fact that the effect of word type was stronger at earlier lags (mean enhancing effect of arousing words for each lag: 120 ms = 16%, 360 ms = 9%, 600 ms = 5% and 840 ms = 6%). In summary, arousing T2s were more easily identified and this effect was stronger at shorter T1–T2 lags when attention was most stretched.

Contrary to predictions, there was no word type x group interaction, $F(1,32) = 1.7$, $p = .2$,—i.e., there was no evidence that AS subjects showed less enhancement of perception for the arousing words *in general*. However, there was a significant word type × lag × group interaction—$F(3,96) = 5.2$, $p = .002$. To investigate this further, the difference in the percentage of correctly identified arousing, compared to neutral, T2s was calculated separately for each group at each lag. As Table 4 (and Fig. 2) shows, at the shortest lag (120 ms) controls demonstrated a greater perceptual benefit for arousing words than did AS subjects—$t(32) = 2.5$, $p = .018$ uncorrected, 0.072 Bonferroni corrected. Such differences were not evident at longer lags—all $t < 0.71$, all $p > .48$.

Correlations with ADOS scores and fear recognition ability: Our preliminary results indicated that a failure to enhance perception for emotionally or socially salient events occurred in AS. We hypothesised that if this cognitive mechanism contributes to the social-perceptual impairment that is characteristic of AS, within-group variation in the magnitude of affective modulation of the attentional blink should predict the severity of autistic symptoms and/or performance on a social perception task. To test these hypotheses, we performed correlational analyses between the magnitude of perceptual enhancement measured in the attentional blink task (as indicated by Fig. 2) and both ADOS and fear recognition scores. There was a weak negative correlation, in the predicted direction, between ADOS score and the size of affective modulation, but this was not significant—$r = -.36$, $p = .16$. However, as predicted, AS subjects did show a significant positive

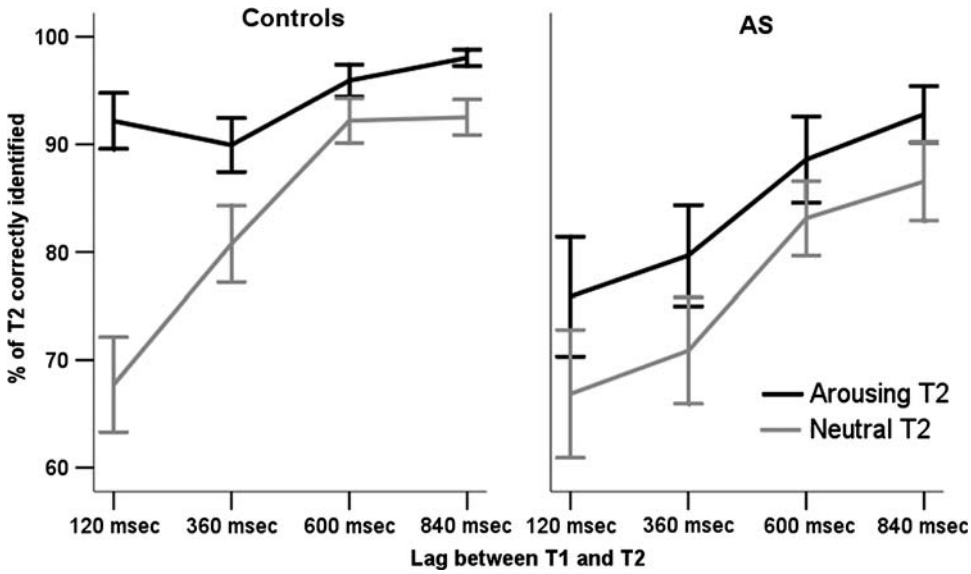

Fig. 2 Results of the emotional modulation of the attentional blink experiment

Table 4 The enhancing effect of arousal on T2 identification

T1–T2 lag		Mean	*SD*
120 msec	Controls	22.3	14.19
	AS	7.4	19.88
360 ms	Controls	8.0	8.77
	AS	7.1	13.42
600 ms	Controls	2.7	7.97
	AS	3.8	12.60
840 ms	Controls	4.9	5.66
	AS	3.2	7.75

Data are the mean difference between the percentage of arousing and neutral T2s correctly identified, broken down by lag and group

correlation between the accuracy of their facial fear recognition score and the size of their emotional modulation to the attentional blink: $r = .50$, $p = .04$. This correlation was completely absent in controls—$r = -.01$, $p = .98$.

Experiment 2

In experiment 1, AS subjects appeared to show reduced emotional modulation of the attentional blink in association with the shortest T1–T2 interval (when attention was most stretched). We next tested the competing hypothesis that, rather than reflecting an impairment specifically in the affective modulation of perception, their impairment could have been due to a more global processing anomaly, which encompassed any type of modulation of perceptual encoding at short inter-stimulus lags. To investigate this possibility, we conducted a second experiment where the salience of the T2 stimuli was altered in terms of their visual-perceptual, rather than their emotional, quality, at the same inter-stimulus intervals.

Methods

Participants

The same subjects who took part in experiment 1 took part in experiment 2.

Design and Procedure

The design and procedure was largely the same as experiment 1. However, on this occasion all T2 words were neutral in content and their salience was adjusted by altering their brightness. Higher salience T2s were jet black (R,G,B channels all 0%), which meant that they stood out clearly against the grey background and multi-coloured distractors. Lower salience T2s were dark grey (R,G,B channels all 25%), with the result that they stood out less clearly. T1 stimuli were mid-way between the two types of T2 in terms of brightness (R,G,B channels all 12.5%). As before, the subjects' task was to monitor the stream of words and to report the identity of the two target words (T1 and T2) by writing them down at the end of the sequence.

Results

Figure 3 summarises the results of the control attentional blink experiment. Again, these data were entered into an ANOVA, with lag and T2 salience as repeated measures variables and group as a between-measures variable.

As before, there were main effects of lag, $F(3,96) = 59.8$, $p < .001$, and T2 salience, $F(1,32) = 33.5$, $p < .001$, as well as a lag x salience interaction, $F(3,96) = 2.7$, $p = .04$. This interaction again seems to be due to T2 salience having a greater effect at shorter lags, when attention is most stretched (see Table 5), but the magnitude of this effect is rather less than was found for emotionally salient T2 stimuli.

There was no main effect of group, $F(1,32) = 2.4$, $p = .13$ and, importantly, no lag $\times$ group, $F(1,32) = .22$, $p = .64$, or lag $\times$ group $\times$ salience interaction, $F(3,96) = .38$, $p = .77$. This suggests that the effect of manipulating T2 salience was the same for both groups at each lag.

Discussion

There was no evidence that AS subjects showed a *general* reduction in perceptual benefit for emotionally arousing stimuli, compared to controls—i.e., in experiment 1 there was no group $\times$ word type interaction. However, when attentional resources were stretched AS subjects showed a much reduced perceptual enhancement for the arousing words, compared to controls. In this regard the AS subjects were reminiscent of a patient with bilateral amygdala damage, whose reduced perceptual benefit for arousing words was most evident at the shortest T1–T2 lag (Anderson et al. 2001).

Control experiments showed that these differences could not be explained by differences in the perceived arousal level of the T2 stimuli, measured by both self-report and skin conductance responses. In addition, a control attentional blink experiment in which T2 salience was modulated by altering its visuo-perceputal properties rather than its emotional arousal, found no significant differences between the two groups at any inter-stimulus interval. In the control experiment, neither group showed the large benefit for the high salience T2s during the shortest T1–T2

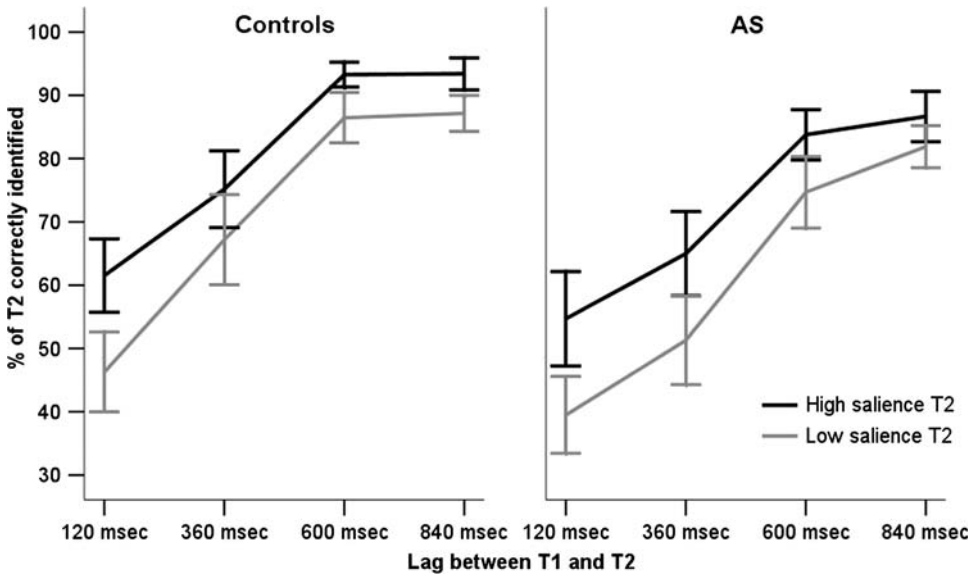

Fig. 3 Results of the attentional blink control experiment

Table 5 The perceptual benefit for high-salience T2s at different T1–T2 lags, compared for each group

T1–T2 lag		Mean	*SD*
120 ms	Controls	15.3	8.06
	AS	15.2	20.70
360 ms	Controls	8.0	16.20
	AS	13.7	18.59
600 ms	Controls	6.8	13.47
	AS	9.1	23.38
840 ms	Controls	6.3	10.37
	AS	4.8	12.88

Data is the percentage difference between the number of high and low salience T2s correctly identified

A significantly greater percentage of high salience T2s were correctly identified at each T1–T2 lag (all $t > 2.8$, all $p < .02$, Bonferroni-Holm corrected), but as the above table shows, this effect was greater at shorter lags

lag which was evident for the control group in experiment 1, perhaps highlighting the particular significance of emotional arousal for cognitive processing in the control, but not the AS, subjects. In addition, our experiments were carefully controlled to be independent of potentially confounding factors that could have influenced the results, especially the influence of limited verbal or visuospatial intelligence.

The data therefore offer partial support for our prediction that the efficacy of the cognitive system which serves to enhance perception for emotionally salient events would be reduced in AS. This builds on several studies from the gaze-processing literature, which show that individuals with an ASD often fail to demonstrate preferential attention towards socially meaningful stimuli (Ristic et al. 2005; Klin et al. 2002; Dalton et al. 2005, Corden et al. 2007).

Our results are consistent with theories that cite a breakdown in the perceptual enhancement for salient stimuli, mediated by the amygdala, as a root cause of social perceptual and social cognitive impairment in ASD (e.g., Grelotti et al. 2002; Schultz 2005; Schultz et al. 2000). In these 'hypo-active amygdala' theories, the basic premise is that, during development, either through impairment with associative learning or via a failure of brain inter-connectivity, the amygdala is unable to flag social stimuli as meaningful, manifesting behaviourally as reduced 'social interest'. Through cognitive development, the net result is hypothesised to be a hindering of social-perceptual learning and consequent underdevelopment of social-perceptual skills (Schultz 2005). In our study, the finding that reduced emotional modulation of the attentional blink predicts poorer ability to recognise fearful faces adds support to this position.

However, there are a number of caveats to our provisional conclusion that amygdala-cortical functional integrity is compromised in people with AS. Firstly, although reduced emotional modulation of the attentional blink may be consistent with a failure of the amygdala to modulate cortical processing, it does not constitute direct evidence of this process, which would require a functional neuroimaging experiment. Furthermore, even if such a neuro-modulation deficit could be demonstrated, it would be unclear where in the system neuropathology is occurring—are the amygdala output nuclei failing to transmit the required signal or is the problem located in the white matter tracts transporting the signal to cortex? An experiment measuring neural activity, i.e., via fMRI, whilst subjects perform the attentional blink task would help resolve these issues and is the logical next step for this project.

Another caveat is that, in the AS subjects of our study, a reduced perceptual benefit for the arousing stimuli did not significantly predict the severity of their autistic symptoms. Within ASD samples, linking performance on laboratory-based tests of social perception and cognition with individual differences in real-life social functioning has often proved difficult (Volkmar et al. 2004). The reasons for this lack of predictability are unclear, but the existence of compensatory strategies and limitations in the methods used for measuring autistic symptomology may be possibilities. The properties of the ADOS, which was used to rate the degree of autistic symptomatology, do not allow for a wide range of dimensionality to be scored, hence the limited range of inter-individual variation provides little scope for parametric correlation. In future, it may be more fruitful to use a measure of autistic trait severity that has sensitivity over a wide range of functioning, for example the AQ (Baron-Cohen et al. 2001b) or the SRS (Constantino et al. 2003).

It should also be noted that our 'arousing' words were only rated as moderately arousing by the participants (3.5/5). It may be interesting to repeat the study using words that the groups rate as more arousing. However, it seems more likely that this would enhance our current findings rather than alter them substantially.

Despite these limitations, we have provided evidence that individuals with AS can show impairment in the cognitive system which modulates attention in response to an emotionally or socially arousing event. Future experiments, using this paradigm in conjunction with neuroimaging, could prove invaluable in testing specific hypotheses from 'hypo-active' amygdala theories of ASD, which place a failure of the amygdala to flag social stimuli as meaningful at the heart of autistic pathophysiology.

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