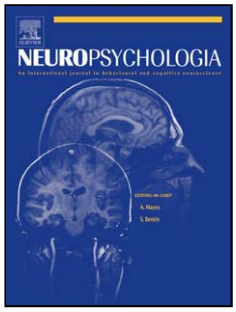

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Avoidance of emotionally arousing stimuli predicts social-perceptual impairment in Asperger's syndrome

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Abstract

We combined eye tracking technology with a test of facial affect recognition and a measure of self-reported social anxiety in order to explore the aetiology of social perceptual deficits in Asperger's syndrome (AS). Compared to controls matched for age, IQ and visual-perceptual ability, we found a group of AS adults was impaired in their recognition of fearful and sad expressions and spent significantly less time fixating the eye region of all faces. For AS subjects, but not controls, the extent of the failure to fixate the eyes predicted the degree of impairment at recognising fearful expressions. In addition, poor fear recognition and reduced fixation of the eyes were independently associated with greater levels of social anxiety in AS individuals. These findings support the hypothesis that avoidance of emotionally arousing stimuli, such as eyes, contributes to social-perceptual impairment in AS. Furthermore, our findings are consistent with theories implicating amygdala mediated over-arousal and anxiety in the development of these social-perceptual deficits.

Key words: social cognition, amygdala, emotion, anxiety, eyetracking, autism

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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 can be unaware of social norms and may 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, especially those involving face processing. Examples include problems recognising facial identity (e.g. Klin et al., 1999; Hobson, Ouston, & Lee, 1988; Boucher & Lewis, 1992) or emotion (Baron-Cohen, Wheelwright, Hill, Raste, & Plumb, 2001; Baron-Cohen, Wheelwright, & Jolliffe, 1997), as well as more abstract reasoning about others' thoughts, beliefs and desires (e.g. Happe, 1994; Castelli, Frith, Happe, & Frith, 2002). Several of these neuropsychological impairments are reminiscent of those seen following iatrogenic or degenerative amygdala lesions, supporting the theory that amygdala anomalies could be involved in the aetiology of a range of symptoms characteristic of autistic spectrum disorders (Baron-Cohen et al., 2000).

A primary consequence of amygdala lesion in humans is impairment in recognising fear, and to a lesser extent sadness, anger and disgust, in facial expressions of emotion (Adolphs et al., 1999). The question of whether individuals with an ASD also show a fear recognition deficit has been a controversial one, with both positive (Howard et al., 2000; Pelphrey et al., 2002) and negative findings (Adolphs, Sears, & Piven, 2001; Castelli, 2005; Grossman, Klin, Carter, & Volkmar, 2000). However, many of these previous studies have suffered from a small sample size (for example, 5 ASD

subjects in Pelphrey et al., 2002, 7 in Adolphs et al. 2001 and 10 in Howard et al. 2000). In addition, previous investigations have not attempted to match or control within the design for group differences in non-verbal IQ or basic visual perceptive ability, factors which could feasibly affect performance on this visually-based face processing task. Therefore, an initial objective of our experiment was to address these problems in a new study of facial emotion recognition in ASD, using a sample of high functioning adults with AS who were well-matched to a control group in terms of age, verbal and non-verbal IQ and basic visual perceptive ability. This experiment comprised the first stage of our investigation.

A number of recent studies have shown that individuals with an ASD attend to faces abnormally. In particular, they spend less time fixating the eyes than control groups (Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Pelphrey et al., 2002; Dalton et al., 2005; Nacewicz et al., 2006, but see van der Geest, Kemmer, Verbaten & van Engeland, 2002 and Bar-Haim, Shulman, Lamy and Reuveni, 2006). Using an eye tracking paradigm with SM, a patient with bilateral amygdala lesion, Adolphs et al., (2005) has shown that her failure to fixate the eyes is likely to be the mechanism of her pervasive fear recognition impairment. This is consistent with other work showing that the eyes play a critical role in normal recognition of fear in facial expressions (Smith, Cottrell, Gosselin, & Schyns, 2005; Kohler et al., 2004). Considering these findings together, we predicted there would be a link between eye fixation and emotion recognition in ASD, with those individuals who spend the least time fixating the eyes having the lowest fear recognition scores. This investigation comprised the second part of our study.

A link between low eye fixation and poor fear recognition ability in AS would be consistent with theoretical accounts suggesting that reduced attendance of socially meaningful aspects of the environment, such as eyes, could impair the ability of AS individuals to accrue social knowledge, thereby leading to social-perceptual and social-cognitive deficits (Grelotti, Gauthier, & Schultz, 2002; Schultz, 2005; Schultz, Romanski, & Tsatsanis, 2000). Amygdala dysfunction has been cited as a potential cause of this reduced attendance of social stimuli – with at least two rival theories of how this could occur. In 'hypo-active amygdala' models, the amygdala fails to flag social stimuli as meaningful with the result that they do not receive preferential attention (Grelotti et al., 2002; Schultz, 2005; Schultz et al., 2000). In 'hyper-active amygdala' models, social stimuli are thought to cause an aversive over-arousal, with the result that they are actively avoided (Dalton et al., 2005; Nacewicz et al., 2006). There is some recent evidence which supports the hyper-active model, for example eye fixation is associated with greater amygdala activity in high-functioning autistics but not controls (Dalton et al., 2005), implying a heightened emotional response to gaze fixation in autism. However, no study has yet attempted to link a behavioural measure of the anxiety or distress experienced by ASD individuals in social situations to their level of attention to social stimuli or to their social-perceptual performance. In the final part of our study we aimed to measure participants' self-reported social anxiety levels and to examine whether higher anxiety can predict a failure to fixate the eyes and poorer recognition of fearful faces.

In summary, we combined eye-tracking of visual scan path with a test of facial affect recognition and a measure of self-reported social anxiety in order to test the following three hypothese: i) when compared with well-matched groups, adults with AS, like

patients with an amygdala lesion, are likely to be impaired at recognising fearful faces. ii) Within-group differences in the ability of AS adults to do this task will be correlated with the degree to which they fixate the eye region of faces. iii) Both of these factors, failure to recognise fearful faces and non-fixation of eye regions, will be associated with heightened social anxiety, as predicted by the 'hyper-active amygdala' model of ASD.

Methods

Participants

21 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, Rutter, & Lecouteur, 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. Autistic symptomology was further characterised via the autistic-spectrum quotient questionnaire, a brief, self-administered instrument for measuring the degree to which an adult with normal intelligence has the traits associated with the autistic spectrum (Baron-Cohen et al., 2001). 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. 21 gender

matched, healthy controls were also recruited. Age, verbal IQ, non-verbal IQ and visual-perceptive ability were matched as closely as possible (see table 1). All subjects gave written informed consent prior to their inclusion in the study.

Table 1. Group characteristics.

	Age (years)	Gender	Verbal IQ	Non-verbal IQ	Full IQ	DTVP Score	AQ score	ADOS Total
AS Mean (*SD*)	33.8 (13.60)	16 M 5 F	115.8 (9.68)	116.2 (13.73)	117.9 (11.67)	106.7 (6.66)	37.1 (6.21)	11.1 (3.40)
Controls Mean (*SD*)	32.1 (11.58)	16 M 5 F	115.1 (8.19)	115.5 (8.75)	117.2 (8.00)	106.3 (6.16)	14.9 (8.58)	NA

DTVP = Developmental Test of Visual Perception. ADOS = Autism Diagnostic Observation Schedule. AQ = Autistic-spectrum quotient.

Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, (2001) found the mean AQ score for a sample of 58 adults with AS or high-functioning autism to be 35.8 (*SD* = 6.5); the mean score for a sample of 174 randomly selected controls was 16.4 (*SD* = 6.3). Fuller descriptions of these neuropsychological variables can be found in the *Design and Procedures* section.

There were no significant differences between any of these variables (all $p > .72$), apart from AQ ($p < .001$).

Design and procedure

Subjects first completed a test of facial affect recognition. Between one and two months later, the same groups repeated this test while their visual scan paths were recorded using an eye-tracker. Scan paths were also recorded during a passive viewing task of the same faces. Finally, subjects completed a self-report measure of social anxiety.

Neuropsychological measures

Verbal, non-verbal (performance) and full-scale IQs were measured via the Wechsler Abbreviated Scale of Intelligence (WASI, Psychological Corporation, 1999).

Although the non-verbal IQ subsets of the WASI are thought to measure aspects of visuo-spatial ability (Psychological Corporation, 1999), we took the view that a more direct test of visual-perceptual skills should be applied, in order to rule out the possibility that emotion recognition ability was related to visuo-perceptual skills in general. To this end, we aqdministered the Developmental Test of Visual Perception (DTVP, Reynolds, Pearson, & Voress, 2002). This test battery is thought to provide a purer test of visual perceptual skill than the non-verbal IQ subtests of the WASI because, unlike the latter test, competence does not rely on reasoning ability (Reynolds et al., 2002).

Ekman-Friesen test of facial affect recognition

In the first part of our investigation, a computerised version of the Ekman-Friesen test of facial affect recognition (Ekman & Friesen, 1976) was administered. Subjects viewed 60 halftone images of emotionally expressive faces, presented in the same order as that used in the original Ekman-Friesen test (Ekman and Friesen, 1976). There were 10 exemplars of each of the 6 basic emotions (happiness, sadness, fear, surprise, anger and disgust). The 6 emotion labels were presented adjacent to each image and participants were required to indicate which emotion they thought was being portrayed via a mouse click. Subjects had an unlimited time to answer. Before beginning the experiment, subjects completed 6 practice examples, one for each emotion. Feedback was not given on these items, the stimuli were not used in the subsequent test and responses to them were not analysed. The facial affect recognition task was repeated as part of the eye-tracking experiment, as described below.

Eye-tracking task

In the second part of the investigation, completed between one and two months after the first, the participants took part in an eye tracking study while viewing the Ekman-Friesen pictures of facial affect (Ekman et al., 1976). Of the original 21 AS subjects, one was unable to take part due a lack of wheelchair access to the testing room, one became anxious during the testing procedure and asked to stop and one completed the task but his recording was not of sufficient quality to be used – therefore data is presented on 18 subjects. The AS group were matched in terms of age and gender with 18 control subjects, who had also participated in the first part of the study. Unfortunately, eyetracking data was corrupted for one control subject, leaving 17 control participants. Details of this truncated group are given in table 2. The AS subjects' impairment in recognising fearful and sad facial expressions persisted with these truncated groups – fear, $U = 95$, $p = .025$; sad, $U = 73.5$, $p = .003$). There were no significant differences between the groups in terms of age, IQ or visual perceptive ability (all $p > .38$).

Table 2 – Group characteristics for those subjects for whom eyetracking data was analysed

	Age (years)	Gender	Verbal IQ	Non-verbal IQ	Full IQ	DTVP Score	AQ score	ADOS Total
AS Mean (*SD*)	32.9 (13.35)	13 M 5 F	116.3 (9.14)	117.1 (14.56)	119.9 (11.10)	106.6 (6.78)	36.9 (6.35)	9.8 (3.75)
Controls Mean (*SD*)	31.9 (11.30)	13 M 4 F	115.1 (8.37)	115.9 (8.87)	117.4 (8.26)	106.7 (6.14)	14.6 (9. 12)	NA

DTVP = Developmental Test of Visual Perception. ADOS = Autism Diagnostic Observation Schedule. AQ = Autistic-spectrum quotient.

Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, (2001) found the mean AQ score for a sample of 58 adults with AS or high-functioning autism to be 35.8 (*SD* = 6.5); the mean score for a sample of 174 randomly selected controls was 16.4 (*SD* = 6.3). Fuller descriptions of these neuropsychological variables can be found in the *Design and Procedures* section.

There were no significant differences between any of these variables (all $p > .49$), apart from AQ ($p < .001$).

Data acquisition, experimental design and procedure

Line of gaze data was acquired at 120 Hz using an ASL (Applied Science Laboratories) Model 504 remote infrared pupil-corneal reflection eye tracker. The assessment took part in a darkened and sound-proof room. Participants were seated in a comfortable chair in front of the computer monitor and were asked to place their chin on a padded chin rest, which minimised head movements and ensured a stable distance of 80 cm between the subject and the centre of the stimulus display screen. The task was divided into two phases, with a rest period in between. At the beginning of each phase, a brief calibration procedure took place, during which subjects were asked to look at nine points of known position on the screen. This corrected for individual differences in head and seating position.

The experiment was realised using Cogent 2000 (http://www.vislab.ucl.ac.uk/Cogent/), which is a MatLab toolbox, designed for creating psychological experiments. The stimuli were the same 60 faces used in the Ekman-Friesen test of facial affect recognition, described above. Briefly, they consisted of 10 exemplars each of the 6 'basic' emotions: happiness, sadness, fear, surprise, anger and disgust.

The experiment consisted of two phases. The first was a 'free-viewing' phase in which there was no experimental task – subjects simply viewed the faces while their eye-movements were recorded. In the second phase, subjects were required to make a judgment about what emotion each face was expressing. The 'free-viewing' phase was included to assess whether any differences between the two groups were task dependent.

In the first phase of the eye-tracking experiment, participants were shown the images in a randomised order, with the constraint that no emotion was shown more than twice in a row. Images were presented for 2500 ms, preceded by a central fixation cross for 1500ms and followed by a blank grey screen for 500ms (total ISI = 2000ms). Participants were instructed to fixate the cross and then to look at the face, "however they wanted to". As stated above, there was no experimental task during this initial phase. In the second phase, the participants were again shown the 60 faces but this time the images were in the standardised order used in the Ekman-Friesen test of facial affect recognition (Ekman et al., 1976). Again, each face was presented for 2500 ms, preceded by a central fixation cross for 1500ms and followed by a blank grey screen. This time, however, following the presentation of each face the participants were asked to decide which emotion had been expressed. The emotion labels (happiness, sadness, fear, surprise, anger and disgust) appeared on the screen following each facial image and were numbered 1-6. Subjects responded via a key press and had an unlimited time to answer.

Data analysis

Any trials showing loss of tracking integrity were excluded from subsequent data analysis, as were off-screen gazes. The regions of interest, the eye and mouth areas, were predefined for each face. The number of fixations made in these regions and on the face as a whole during the 2500ms presentation was calculated. A fixation was defined as a set of consecutive gaze coordinates, confined within a diameter of 1 degree of visual angle for a duration of 100 milliseconds or more (Norton & Stark, 1971). The fixations on the eyes and mouth were calculated as a percentage of the

total fixations made to the face for each stimulus. The average proportion of fixations made to the eye and mouth regions were calculated separately for each emotion, for each subject.

This is the method that has been used in virtually all of the previous studies of visual tracking of faces with autism and amygdala damage (e.g. Pelphrey et al., 2002; Klin et al., 2002; Adolphs et al., 2005; Dalton et al., 2005; Nacewicz et al., 2006). However, it does raise a number of issues, which will briefly be discussed. Firstly, the number of fixations to the eyes will vary (inversely) as a function of the duration of fixations. Hence, there is the potential for a participant who makes lots of short fixations to bias the results by erroneously appearing to be paying much more attention to the eyes than a participant who makes a few, long fixations. Because of this problem, a simultaneous analysis was conducted that used the total *duration* of fixations rather than the total *number* of fixations. The pattern of results was the same, both qualitatively and quantitatively. Therefore, for clarity, only the total number of fixations data is presented here.

Secondly, proportionalising the data may also obscure some important group differences: it is unclear whether or not the individuals with AS looked less at the face overall than controls. Therefore, the total number of fixations made to the face as a whole was also assessed and compared between the AS group and controls.

Social anxiety

To assess the level of anxiety suffered by participants in social situations, the Social Phobia and Anxiety Inventory (SPAI, Turner, Beidel, & Dancu, 1999) was

administered. This is a 45 item, empirically derived questionnaire, designed to assess the somatic, cognitive and behavioural aspects of social anxiety across a wide range of social situations and settings.

The validity of self-reported anxiety measures can be diminished by social desirability, i.e. the tendency to portray oneself in a positive light. (Egloff & Schmukle, 2003). To control for this potential response bias, we administered the Marlowe-Crowne Social Desirability Scale (SDS, Crowne & Marlowe, 1960). The SDS consists of 33 questions, drawn from a population of items defined by behaviours which are culturally sanctioned and approved but which occur only infrequently in everyday life.. Participants decide 'true' or 'false' depending on whether or not they believe themselves to engage in the behaviour. The resulting score is an index of the tendency to portray oneself in a good light, which can be entered as a covariate in statistical analyses.

Results

Part 1 – Facial affect recognition

Figure 1 summarises the Ekman-Friesen test results as a box-plot. The data were not normally distributed and could not be normalised by transformation; therefore Mann-Whitney tests are used to compare group differences. Correction for multiple comparisons was done via the Bonferroni-Holm method (Hochberg & Tamhane, 1987). AS subjects performed significantly worse than controls at recognising fearful ($U = 124$, $p < .05$, Bonferroni-Holm corrected) and sad ($U = 104.5$, $p < .05$, corrected) facial expressions.

Reaction times[1] were slightly slower for the AS group, although this was not significant (mean ± *SD*, Asperger's, 1061 ± 542 msec; controls, 909 ± 363 msec; $t(40) = 1.05$, $p = .3$), suggesting that the differences between the two groups were not due to a speed-accuracy trade-off. In addition, there were no significant correlations between fear or sadness recognition score and reaction time, age, verbal IQ, performance IQ or basic visual perceptive ability (as measured by the DTVP) for either group (all $r < .28$, all $p > .21$), suggesting that differences in cognitive or perceptual ability could not explain the variability in fear and sadness recognition score.

In the AS group, neither fear or sadness recognition significantly correlated with the severity of autistic symptomology, measured either as part of the ADOS or self-reported by the AQ (all $r < .25$, all $p > .31$).

FIGURE 1 ABOUT HERE

Part 2 - Eye-tracking study

Emotion recognition re-test

Table 2 shows the results the Ekman-Friesen emotion recognition test, which subjects repeated as part of the second phase of the eyetracking experiment. Both groups showed an improvement compared with the first time they took the test, but the rank

[1] These are the mean reaction times, averaged over all emotions. Unfortunately, due to loss of the raw data it was not possible to present reaction times for the individual emotions. However, the Ekman-Friesen test was repeated as part of the eye-tracking part of the study and for these data the reaction times are given in full.

order of the groups did not change, as shown by non-significant Wicoxon signed ranks tests for each emotion (all $p > .35$).

AS subjects were again significantly impaired at recognising fearful faces ($U = 85$, $p < .05$, corrected), but there were no significant effects for the other emotions after correcting for multiple comparisons (all $p > .2$ corrected).

		Mean	*SD*
Happy	AS	9.9	.24
	Controls	10.0	.00
Sad	AS	7.7	1.19
	Controls	8.2	1.25
Fearful	AS	7.5	1.41
	Controls	8.8	1.33
Angry	AS	8.5	1.34
	Controls	8.7	1.10
Surprised	AS	8.5	2.20
	Controls	8.8	1.29
Disgusted	AS	6.6	2.57
	Controls	8.1	2.26

Table 2. Re-test of the Ekman-Friesen test with the AS group and controls.

The reaction times for the emotion recognition task are shown in figure 2, split by emotion and group. These data were entered into an ANOVA. There was a highly significant effect of emotion, $F(5,160) = 19.3$, $p < .001$. Post-hoc comparisons revealed that this was driven by the faster mean response time for happy faces, which was significantly lower compared to all other emotions (all $p < .001$). No other pairwise comparisons were significant. Although the mean response time of the AS group was longer for each emotion, there was no significant effect of group, $F(1,32) =$

1.5, p = .23, or a significant group x emotion interaction, $F(5,160)$ = .3, p = .93. In addition, there was no significant correlation between mean emotion recognition score (averaged across all emotions) and mean reaction time for either group (AS, r = .004, p = .99; controls, r = .25, p = .34). Together, these results confirm the suggestion made in part 1 that emotion recognition deficits in the AS group can not be attributed to a speed-accuracy trade-off.

FIGURE 2 ABOUT HERE

Fixations to the eye region

The fixation data were entered into an ANOVA with group as a between-measures variable with experimental phase (two levels) and emotion (six levels) as repeated-measured variables. The dependent variable was the percentage of fixations made to the eyes. There was no significant effect of phase or a significant interaction between phase and the other variables (all F < 1.5, all p > .2), indicating that both groups scanned the faces in a similar manner during both the free viewing and emotion recognition tasks. Therefore, for display purposes, data from both phases are combined (figure 3). There was a main effect of group, i.e., as predicted, AS subjects spent significantly less time fixating the eye region compared to controls, $F(1,32)$ = 5.6, p = .02 – see figure 3. The main effect of emotion was also significant, $F(5,160)$ = 17.7, p < .001, as was the group x emotion interaction, $F(5,160)$ = 2.8, p =.02.

Post-hoc comparisons were used to investigate the main effect of emotion. These revealed that subjects (both groups combined) spent significantly less time fixating the eyes when viewing disgusted and angry faces, compared to all other emotions (all

$p < .05$, Bonferroni corrected). In addition, subjects spent significantly more time fixating on the eyes when viewing surprised compared to happy faces. No other pairwise comparisons were significant following correction for multiple comparisons.

FIGURE 3 ABOUT HERE

To investigate the significant group x emotion interaction, separate ANOVAs for each group were first conducted. These revealed that the effect of emotion on eye-fixations was significant for both groups (AS group: $F(5,85) = 4.4$, $p = .001$; control group: $F(5,80) = 19.8$, $p < .001$). Therefore, to further investigate the group x emotion interaction t-tests were conducted to compare the groups on each emotion separately. The effects for surprised, $t(1,32) = -2.9$, $p = .02$, sad, $t(1,32) = -2.7$, $p = .03$, and happy, $t(1,32) = -2.6$, $p = .03$, faces were significant, following Bonferroni-Holm correction for multiple comparisons. There was a trend towards significance for all the other emotions ($.06 < p < .09$, corrected).

The data entered into the ANOVA reported above were proportionalised: i.e. total fixations to the eyes as a percentage of total fixations to the face. This may mask the possibility that AS subjects may be fixating on the *whole* face less than controls. To assess this, we compared the total fixations made to the face as a whole between the two groups: there was no significant difference (AS group, mean number of fixations to whole face = 8.85, $SD = 1.04$; controls, mean number of fixations to whole face = 8.94, $SD = .89$; $t(33) = .25$, $p = .81$).

Fixations to the mouth

Data on the amount of time spent fixating the mouth was also entered into an ANOVA with group, phase and emotion as independent variables. Again, the main effect of phase and its interactions with the other variables were not significant (all $p > .1$); therefore data for the different phases are combined for display (figure 4). There was a trend towards the AS group spending more time fixating the mouth than controls but this did not reach significance, $F(1,32) = 3.5$, $p = .07$. However, there was again a main effect of emotion, $F(5,160) = 7.74$, $p < .001$. Post-hoc comparisons suggest that this was driven by fewer mouth fixations being made to sad expressions: this was significant in comparison with every other emotion (all $p < .004$, Bonferroni corrected). No other pair-wise comparisons were significant.

FIGURE 4 ABOUT HERE

In the AS group, less time spent fixating the eyes was not associated with greater severity of autistic symptomology, as measured by either the ADOS or the AQ ($r < .18$, all $p > .40$). The percentage of fixations made to the mouth showed a trend towards a correlation with self-rated autistic behaviour as measured by the AQ ($r = .47$, $p = .07$) but not with the reciprocal social interaction, communication or total ADOS score (all $r < .26$, all $p > .33$)

Correlating time spent fixating the eyes with fear recognition score

Next we tested the prediction that the AS subjects with the worst fear recognition ability[2] would be those that fixated the eyes the least by examining the correlations between these variables separately for each group. Figure 5 shows the relevant scatter

[2] All the emotion recognition scores used in this section are mean scores, calculated from both repetitions of the test.

plots. Poor fear score was correlated with low eye fixation within the AS group (r = .56, p = .01) but not the control group (r = .06, p = .41). However, these correlation co-efficients were not significantly different, z = 1.29, p = .20. The correlation for the AS group remained significant even after removal of the outlier seen in the bottom left hand side of the scatterplot (r = .45, p = .03).

FIGURE 5 ABOUT HERE

As discussed in the introduction, previous work with typically-developed individuals has shown that the eyes play a crucial role in normal recognition of fearful, but not other, facial expressions (Smith et al. 2005; Kohler et al., 2004). Furthermore SM, who has bilateral amygdala lesions, shows reduced fixation of the eyes for all faces regardless of emotional expression, yet displays *selective* fear recognition impairment (Adolphs et al., 2005). Consistent with these findings, the amount of time the AS group spent fixating the eyes significantly predicted *only* the ability to recognise fear – not the other emotions (table 3).

		Recognition score for…					
		Happy faces	Sad faces	Fearful faces	Angry faces	Surprised faces	Disgusted faces
% fixations made to eyes (mean of all faces)	*r*	-.01	.35	.56*	-.02	-.14	.23

Table 3. For people with AS, the amount of time spent fixating the eye region of faces predicts recognition score only for fearful expressions, consistent with the critical role of the eyes for recognising this, but not other, emotions (Smith et al. 2005; Kohler et al., 2004).

*p < .06, Bonferroni-Holm corrected.

Furthermore, consistent with the fact that the AS subjects showed reduced eye fixations to all the faces (see figure 2), and again consistent with SM (Adolphs et al.,

2005), their fear recognition score was predicted by the amount of time spent fixating the eyes of *all* facial expressions (except happy), not just fearful faces (table 4).

		Percentage of fixations made to the eyes for…					
		Happy faces	Sad faces	Fearful faces	Angry faces	Surprised faces	Disgusted faces
Mean fear recognition score	*r*	.34	.55*	.56*	.58*	.51*	.48*

Table 4. For people with AS, fear recognition ability is predicted by the amount of time spent fixating eyes of all facial expressions (except happy). $^{*}p < .05$, Bonferroni-Holm corrected

Greater social anxiety predicts poorer fear recognition and less time spent fixating the eyes in AS

Next, we examined the level of social anxiety, measured as a self-rated personality trait, experienced by the participants. We tested the prediction that, within the AS group, poorer fear recognition and less time spent fixating the eyes would both be associated with greater levels of social anxiety.

The AS group rated themselves as significantly more socially anxious than the controls: AS group, mean SPAI rating = 110. 1 (*SD* = 17.90); control group, mean SPAI rating = 65.5 (*SD* = 46.78); $t(16.13) = 3.4$, $p = .004$, equal variances not assumed. However, for the AS group, social anxiety did not correlate significantly with the severity of autistic symptoms, measured via the ADOS or AQ (all $r < .22$, all $p > .42$).

For the AS group, higher social anxiety predicted a lower fear recognition score, even after controlling for differences in the Marlowe-Crowe social desirability scale (see methods), $r = -.51$, $p = .04$. This correlation is absent in controls, $r = .27$, $p = .38$.

Furthermore, higher social anxiety was associated with less time spent fixating the eyes in AS subjects (r = -.50, p = .04), but not controls (r = .07, p = .8), again after correcting for differences in social desirability.

Discussion

As predicted, individuals with AS were impaired at recognising fearful faces compared to controls. Like patients with amygdala damage, this deficit extended to other negative emotions – there was a significant effect for sad faces and a trend towards an effect for angry faces. These data replicate studies by Howard et al. (2000) and Pelphrey et al. (2002), who both found a fear recognition impairment in adults with high functioning autism or AS, as well as trends toward an impairment for recognising sad and angry expressions. However, the present findings do not concur with studies by Adolphs et al. (2001), Grossman et al. (2000) or Castelli (2005), who all failed to find an impaired ability to recognise fear or any of the other 'basic' emotions in ASD. It is interesting to note that the latter two of these studies were conducted with children (mean chronological age 11.8 and 12.3 respectively, verbal age 9.3 years for the latter) rather than adults, which may go some way to explaining the negative findings. For example, control children may not yet have reached a sufficient stage of development to enable them to complete the task successfully (Wade, A.M., Lawrence, K., Mandy, W. & Skuse, D., 2006). This would appear to be the case in the Grossman et al. (2000) study where control children correctly identified only 50% of the fearful faces (Asperger's children identified 48% correctly). The other study which failed to find a fear recognition deficit in ASD was conducted with adults (Adolphs et al., 2001). However, this was with a small sample

size (7 ASD subjects) and examination of the data shows that all but one ASD subject scored below the control mean (see figure 2 of Adolphs et al., 2001), suggesting the possibility that there may have been insufficient power to find a fear recognition deficit.

Our two groups were well matched in terms of age, verbal and non-verbal IQ and basic visual perceptual abilities (as measured by the DTVP). This is in contrast to earlier studies, none of which controlled for non-verbal IQ or basic visual perceptive ability. In addition, reaction times did not differ significantly between our groups and neither this nor age, IQ or DTVP correlated with fear or sadness recognition ability within the AS group. Therefore, neither differences in cognitive/perceptual abilities nor a speed-accuracy trade-off can be invoked to explain the observed fear and sadness deficits. Rather, they would appear to reflect a specific impairment in social-perceptual ability within the AS group. Considering our data and that of previous studies together, there would appear to be a growing consensus of a fear recognition deficit amongst adults with high-function autism or AS.

As predicted, the AS group made significantly fewer fixations to the eyes than did control subjects. This replicates a number of earlier studies, which found the same result with adults diagnosed with autism (Pelphrey et al., 2002; Klin et al., 2002; Dalton et al., 2005; Nacewicz et al., 2006), and is consistent with clinical and anecdotal reports of poor eye fixation generally in autistic spectrum disorders (for example, see, American Psychiatry Association, 1994; Hutt & Ounsted, 1969).

In the eye-tracking task, there was no significant effect of experimental phase or a significant phase by group interaction, suggesting that both groups fixate faces in the same way regardless of whether or not they are overtly engaged in judging facial expression. There was, however, a significant group by emotion interaction, which post-hoc t-tests suggested was due to a greater difference between the groups when fixating sad, surprised and happy faces compared to the other emotions (see figure 3). Although the post-hoc t-tests for fearful, angry and disgusted faces were not significant following correction for multiple comparisons, there were trends towards significance in each case ($.06 < p < .09$, corrected, see figure 3) and it seems likely that reduced fixation to the eyes for all six emotions contributed to the overall reduction in eye-fixations found for the AS group compared to controls.

Also as hypothesised, the degree of fear recognition impairment amongst the AS subjects was predicted by the extent of their failure to fixate the eyes when viewing faces. A number of previous studies have shown a fear recognition deficit in ASD (Howard et al., 2000; Pelphrey et al., 2002), others have shown that autistic individuals spend less time fixating eyes (Pelphrey et al., 2002; Klin et al., 2002; Dalton et al., 2005; Nacewicz et al., 2006) and we know that eyes are a critical feature for fear recognition in normal subjects (Smith et al., 2005; Adolphs et al., 2005; Kohler et al., 2004). However, to our knowledge, this is the first study to formally link eye fixation to fear recognition in an ASD.

In the AS group, greater fear recognition ability was predicted by the amount of time spent fixating the eyes in *any* face, regardless of facial expression. However, the amount of time spent fixating the eyes only significantly predicted recognition

performance for fearful faces – it did not predict how accurately AS subjects recognised the other emotions. These findings are consistent with the data from normal subjects, showing the crucial role of the eyes in recognising fearful faces but not other expressions (Smith et al., 2005; Kohler et al., 2004) and is reminiscent of SM, who showed reduced fixation to the eyes for *all* faces, regardless of emotion, but who has a *specific* fear recognition deficit (Adolphs et al., 2005).

Our findings are consistent with theories that suggest avoidance of emotionally arousing stimuli, such as eyes, can contribute to social-perceptual impairment in ASDs (Grelotti et al., 2002; Schultz, 2005; Schultz et al., 2000). Under these schemes, chronic inattention to socially meaningful stimuli interferes with the ability to accrue social knowledge and thereby impairs social-perceptual ability (Schultz, 2005). A number of theorists have argued that more complex social-cognitive abilities, such as theory of mind, build upon basic social perceptual knowledge (Tager-Flusberg & Sullivan, 2000; Baron-Cohen, 1994; Hobson, 1993) and so deficits in perceiving social stimuli may have a wide impact on the development of social skills as a whole.

Recently, an 'amygdala hyper-activity' model has emerged which is perhaps able to explain the suggested lack of attendance to social stimuli in ASDs (Dalton et al., 2005; Nacewicz et al., 2006; Amaral & Corbett, 2002). In this model, which has its roots in the work of Hutt and Ounsted (1969), amygdala dysfunction is associated with increased anxiety in individuals with an ASD (Amaral et al., 2002). Social stimuli, especially those with particular emotional relevance such as the eyes, are thought to result in an amygdala-mediated over-arousal and so are actively avoided (Dalton et al., 2005). This could lead to social-perceptual and social-cognitive

impairment via the pathway described in the previous paragraph. We found that the level of social anxiety experienced by AS subjects predicts the extent of both their gaze aversion and their fear recognition. This is consistent with the predictions of the 'hyper-active' model and provides the first evidence linking a behavioural measure of the anxiety experienced by ASD individuals in social situations to their level of attention to social stimuli and to their social-perceptual performance.

There are two important and related caveats that should be noted. If the hyper-activity model does apply in the way stated above, it clearly does not apply equally to all individuals with AS. The AS group varied from normal to abnormal both in terms of fear recognition and the amount of time spent fixating the eyes. On its own this would not necessarily be a problem – one could posit that those with the least abnormality on these measures would be the least severely autistic. However, this brings us to the second caveat: neither poor fear recognition ability nor the lack of time spent fixating the eyes predicted the severity of autistic symptoms, as measured by the ADOS and AQ. A lack of association between laboratory based measures of social perception and real world social functioning is not uncommon in ASD research (Volkmar, Lord, Bailey, Schultz, & Klin, 2004). However, in a similar eye-tracking study to our own, Nacewicz et al. (2006) found that autistic symptoms correlated negatively with time spent fixating the eyes. One possible reason for this inconsistency is that the participants in the Nacewicz et al. (2006) study were low functioning individuals with autism, in contrast to our high-functioning AS group. In addition, Nacewicz et al. (2006) measured autistic symptoms via the ADI-R, which produces more dimensional results than the rather categorical ADOS.

Another possibility is that inattention to socially relevant stimuli, and the social-perceptual deficits that ensue, may simply not be sufficient, on their own, to cause problems in day to day interaction and communication. There may exist a multitude of compensatory strategies that allow for appropriate interaction in spite of problems with social perception, particularly in cognitively able individuals, like our AS group, who may use their visual-perceptual or verbal abilities to scaffold their performance (Klin, Schultz, & Cohen, 2000). In addition to social cognitive deficits, as a group, individuals with an ASD are known to have difficulties with executive function (Pennington & Ozonoff, 1996) and with integrating details into meaningful wholes ('weak central coherence', Happe & Frith, 1996). These deficits may reduce the ability to compensate for social perceptual problems. In this framework, social-perceptual, executive and central coherence deficits interact such that 'multiple insults' are required to produce the profound difficulties in social interaction often seen in ASD. To predict the severity of autistic symptoms in ASD, measures of all three factors may be required. Clearly, more research into the mapping of laboratory demonstratable social-perceptual / social-cognitive impairment onto real world social ability, perhaps via interaction with other core autistic deficits such as executive function, would prove beneficial. Information of this kind could potentially have important consequences for treatment of social difficulties in this population.

Another important factor, which may help bridge the gap between laboratory deficits and 'real world' social impairments, is the ecological validity of the social stimuli used. Like the majority of studies into face processing in ASD, we used static, black and white photographs of emotional expressions. However, there is evidence that more valid, dynamic videos of facial affect may magnify the impairments

demonstrated on these tasks by individuals with AS (Klin et al., 2002; Klin, Jones, Schultz & Volkmar, 2003). In future studies, it will be important to use more ecologically valid stimuli, either using videos or perhaps even live interactions between the subject and an actor, utilising the new lightweight, head-mounted eye-tracking equipment now available. Related to the ecological validity of the stimuli is the fact that two subtly different explanations exist for why the AS subjects looked less at the eyes in the Ekman photographs. On the one hand, they may find the actual eyes in these pictures aversive and so avoid them or, alternatively, they may be demonstrating a learned response from a life-time of aversive experience with real eyes and faces. Whichever is the case our conclusions would remain largely unchanged but this does highlight some of the subtle difficulties of interpretation that can arise when trying to relate laboratory stimuli to real world behaviour.

A specific caveat to our argument concerns the ~4 outliers in the correlation between the percentage of fixations made to the eyes and fear recognition score (see figure 6).

FIGURE 6 ABOUT HERE

These individuals rarely fixated the eyes (making 7 – 14% of fixations to the eyes, compared to a mean of 29% for the AS group and 40% for controls) yet they correctly identified the majority of the fearful faces (mean fear recognition score between 7 and 8 out of 10). These individuals are potentially very interesting because they show that it is possible to achieve a normal fear recognition score without looking at the eyes. These individuals may be using a compensatory cognitive strategy, solving the task in

an unusual way, not involving the eyes[3]. This would be consistent with previous studies on other aspects of face processing in ASD, such as face recognition memory, that have shown that individuals with an ASD can perform a given task as well, and sometimes better, than controls, but may be doing so in an atypical fashion (e.g. Langdell, 1978). A method of assessing any alternative strategy would be to use the *Bubbles* task (Gosselin & Schyns, 2001; Smith et al., 2005) with these individuals, which would give precise details of the areas of the face that are critical for them to identify a fearful expression. Recent results using the *Bubbles* task (Spezio et al., 2007; Spezio et al., 2006) show that experiments such as these have the potential to provide useful insight into the mechanism of the social-perceptual impairment experienced by those with an ASD.

Another point to note is that ceiling effects compromise our data to a certain degree. For example there is little variation in the emotion recognition abilities of the control subjects, which may explain the finding of numerous null correlations in the control group (i.e. between fear recognition ability and the number of fixations made to the eyes). Ceiling effects may also be an issue for the AS data: the variability in the recognition of fear is greater than it is for the other emotions. This may partially explain why the number of fixations made to the eyes correlated with fear recognition ability but not the ability to recognise the other 5 emotions. However, there was still reasonable variability in the recognition of the other emotions in the AS group (see figure 1). Therefore, it still remains likely that the correlation reflects the importance of the eyes for recognising fear, as has been shown with other studies using different methods (Smith et al., 2005; Adolphs et al., 2005; Kohler et al., 2004).

[3] In fact, if their strategy did not involve the eyes it may *add* to their low eye fixation percentages by directing them away from the eyes towards other features.

Another caveat is that our social anxiety instrument measured a general personality trait rather than direct responses to the facial stimuli. The 'hyper-active amygdala' model suggests that AS subjects would avoid stimuli such as eyes because they find them aversive and anxiety-inducing. To investigate whether this is indeed the case, it will be necessary in the future to take more direct measurements of the subjects' emotional responses to looking at eyes. It is important to note that this would not be possible within a free-viewing paradigm such as the one used in the current study, which would erroneously show no difference between the arousal experienced by AS subjects and controls because the AS subjects would avoid the eyes and hence the arousal. Therefore, it will be necessary to *control* where on the face the subjects are looking, i.e. to instruct them to look either at the eyes or the mouth, while taking both self-report and physiological (e.g. skin conductance) responses to the stimuli on a trial-by-trial basis.

Finally, our current study collapsed the eyetracking data over the whole 2500 msec period. This is what has been done in previous eyetracking studies (e.g. Pelphrey et al., 2002; Klin et al., 2002; Adolphs et al., 2005; Dalton et al., 2005) and has yielded results. However, this method may be masking important group differences in the way that individuals with AS, compared to normal controls, scan faces. Therefore, in the future it will be important to conduct finer-grained temporal analyses of scan-path data.

Despite these issues, we have been able to demonstrate for the first time a link between eye fixation and social perceptual ability in an autistic spectrum disorder. In

addition, we have provided novel data linking a behavioural measure of social anxiety with both inattention to socially meaningful stimuli and with social-perceptual performance in AS. These findings have theoretical relevance in light of recent models of amygdala pathophysiology in autistic spectrum disorders (Schultz, 2005; Dalton et al., 2005; Nacewicz et al., 2006). In future, it will be important to integrate investigations of potential compensatory strategies that AS subjects may be using to complete social tasks, as well as measures of their impairment in other areas of the autistic phenotype, such as executive function, in order to explore more fully the aetiology of the social-cognitive deficits seen in this condition.

Figure captions

Figure 1. Results of the Ekman-Friesen test of facial affect recognition for AS subjects and age and gender matched controls. Boxes show the median (black line) and the inter-quartile range (IQR, edges of boxes). Whiskers are 1.5 x IQR. Circles represent mild outliers (between 1.5 and 3 x IQR) and stars represent extreme outliers (> 3 x IQR).

Figure 2. Mean reaction times for the emotion recognition test taken in phase 2 of the eyetracking experiment. Error bars indicate ± 1 standard error.

Figure 3. Subjects with AS spend less time fixating the eye region of faces than age and gender matched controls. Error bars indicate ± 1 standard error.

Figure 4. The amount of time spent fixating on the mouth in the eyetracking task, split by emotion and group. Error bars indicate ± 1 standard error.

Figure 5. Scatterplots showing the correlation between fixations to the eye region and the ability to recognise fearful faces, shown separately for each group. Fear recognition scores have been averaged across the two repetitions of the test.

Figure 6. Outliers (enclosed within the ellipse) in the correlation between the percentage of fixations made to the eyes and fear recognition score.

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