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Review

Vision in autism spectrum disorders

David R. Simmons *, Ashley E. Robertson, Lawrie S. McKay, Erin Toal, Phil McAleer, Frank E. Pollick

Department of Psychology, University of Glasgow, 58 Hillhead Street, Glasgow G12 8QB, Scotland, UK

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ABSTRACT

Autism spectrum disorders (ASDs) are developmental disorders which are thought primarily to affect social functioning. However, there is now a growing body of evidence that unusual sensory processing is at least a concomitant and possibly the cause of many of the behavioural signs and symptoms of ASD. A comprehensive and critical review of the phenomenological, empirical, neuroscientific and theoretical literature pertaining to visual processing in ASD is presented, along with a brief justification of a new theory which may help to explain some of the data, and link it with other current hypotheses about the genetic and neural aetiologies of this enigmatic condition.

1. Introduction

Autism is a developmental disorder characterized by difficulties with social interaction, social communication and an unusually restricted range of behaviours and interests (Frith, 2003). A diagnosis of autism also requires a clinically significant delay in language development before the age of 3 years. Asperger Syndrome has similar signs and symptoms to autism except without the language delay. Together (with PDD-NOS)[1] these two diagnostic groups have come to be known as Autism spectrum disorders (ASDs), although there is ongoing controversy about how distinct they really are (see Volkmar, State, & Klin, 2009).

A second controversy concerns whether or not the prevalence of ASDs (about 1% in western countries) is increasing (Charles, Carpenter, Jenner, & Nicholas, 2008). The prevalence controversy is complicated by two factors: the historical changes in diagnostic criteria (see below) and the continuing debate over the causes of ASDs. Concerning the latter, those who favour a genetic cause point to the strong evidence for a genetic component in the aetiology of ASD: chiefly the male/female differences in prevalence (about a factor of 4 higher in males) and the data indicating that ASD is more prevalent in monozygotic than dyzygotic twins (Bailey et al., 1995; Baron-Cohen, 2003; Bourgeron, 2008). If the cause is genetic (rather than epigenetic) then any increase in prevalence must result from improved diagnosis. An environmental explanation centres on a genuine increase in the numbers of those who have ASD arising from environmental stressors, such as pollution, diet or lifestyle (see Altevogt, Hanson, & Leshner, 2008; Rutter, 2009; Thornton, 2006). It is likely that both genetic and environmental factors contribute to the aetiology of ASDs, although a number of genetic disorders can result in similar symptoms (e.g. Fragile-X, Tuberous Sclerosis: see Gillberg & Coleman, 2000, for more).

2. The diagnosis of ASDs

The signs and symptoms of ASD amenable to diagnosis are almost entirely behavioural, so a large variety of diagnostic instruments are based on either the direct or indirect observation of individual behaviour. These include the Autism Diagnostic Interview (ADI; Lord, Rutter, & Le Couteur, 1994), the Autism Diagnostic Observation Schedule (ADOS; Lord et al., 2000), the Developmental, Dimensional and Diagnostic Interview (3Di; Skuse et al., 2004) and the Diagnostic Interview for Social and Communication Disorders (DISCO; Leekam, Libby, Wing, Gould, & Taylor, 2002). Variants of these tests are used depending on the age and/or language level of the individual concerned. Diagnostic instruments also vary in the way that they gather data. Most instruments include a history-taking procedure, particularly important for determining early language development (e.g. ADI). However, others rely largely on interviewing the individual concerned and/or observing them directly (e.g. ADOS). It is generally thought that, for research purposes, a directly observed diagnosis is more reliable because it does not utilize third-party information. However, the most reliable diagnoses probably involve a combination of approaches (e.g. ADI together with ADOS). Within a clinical context the validity of research is frequently judged on the method and source of diagnosis. Obtaining such reliable diagnoses requires at the very least specialist training, and probably collaboration with clinical partners. In many studies that we will consider the ASD

* Corresponding author. Fax: +44 (0)141 330 4606.
E-mail address: david@psy.gla.ac.uk (D.R. Simmons).

[1] Pervasive Developmental Disorder-Not Otherwise Specified (PDD-NOS) is the diagnostic category used if the severity of the signs and symptoms are just below the level required for full autism or Asperger syndrome.

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population is characterized vaguely as "diagnosed by clinical specialists" or may even rely solely on parental report of previous diagnosis.[2] Others will specify the use of standard diagnostic criteria, such as DSM-IV (American Psychiatric Association, 1994) or ICD-10 (World Health Organisation, 1993). Sometimes further checks will be made using screening instruments, either self-report questionnaires such as the Autism Spectrum Quotient (AQ; Baron-Cohen, Wheelwright, Skinner, Martin, & Clubley, 2001b), or third-party questionnaires, such as the Social Responsiveness Scale (SRS; Constantino et al., 2003). We shall try to assess the reliability of diagnoses used, whilst bearing in mind that important insights into visual functioning in ASD may still be obtained even when the "gold standard" of diagnosis is not (Dover & Le Couteur, 2007; McClure & Le Couteur, 2007). Note also that, for a study to be specific to ASD, the known genetic syndromes which have similar symptom profiles to ASD should be explicitly ruled out, as, ideally, should other forms of learning disability or neuropathology.

3. Other general issues with research into ASDs

The commonest design for an experiment in this area is to source a population of individuals with ASD and compare their performance with a control group, which may be "typical" of the general population, or be drawn from another clinical population, such as individuals with Attention Deficit Hyperactivity Disorder (ADHD) or Down's syndrome. Normally these populations are "matched", using IQ (verbal (VIQ), non-verbal (NVIQ) or full-scale (FSIQ)), chronological age (CA) or mental age (MA), with the choice depending upon the task concerned, such as whether or not it makes high demands on language and whether performance might be more influenced by physical maturation or cognitive ability. These issues are typical of those encountered in clinical research, but there are some added difficulties when dealing with ASD. First, there is the question of which part of the autism spectrum to target. Most studies use people with either "high-functioning" autism (HFA), or Asperger Syndrome (AS) who generally have average, or even above-average, IQ and do not require an additional learning-disabled control group for comparison. However, because the HFA/AS group will also have less profound symptoms, any performance differences between this group and controls will tend to be smaller. If a more severely affected autism group is chosen then a learning-disabled control group may be required, introducing problems of how to convey instructions, maintain participant attention and record experimental responses. Furthermore, even taking a population that is close in terms of IQ, age and position on the autism spectrum does not guarantee homogeneity within the population. It is often suggested (only half jokingly) that the heterogeneity of performance in a given task is the only reliable difference between data obtained from an ASD and a typical control population! Finally, given that social reticence and maintenance of routine is one of the symptoms of ASD it can be hard to recruit volunteers. Thus, obtaining reliable and statistically significant differences between ASD and control populations can be a major challenge. Added to this is the care with which task design must be undertaken. It is hard to determine whether poor performance on a task is due to an ASD-specific performance deficit, or whether there is a more generalised issue, such as attention control or a misunderstanding of the task instructions. Another potential factor is the use of medication by the ASD group, particularly in adults. The most convincing studies demonstrate differential performance on very similar tasks where these collateral demands are the same (see Bertone & Faubert, 2006; Burack, Iarocci, Flanagan, & Bowler, 2004; Jarrold & Brock, 2004).

A final important issue to consider is the fact that ASD is a developmental disorder, the signs and symptoms of which can change dramatically over time in a given individual (Fecteau, Mottron, Berthiaume, & Burack, 2003). Hence, what is true for, say, 8–12 year old children on the autism spectrum may not apply to adults. This is a particular issue in ASD given that it is characterized by what are sometimes called "asymmetric" development patterns.

4. Sensory symptoms in ASDs

Given that ASDs are defined primarily in terms of social symptoms it may seem odd that sensory processing in these conditions is of interest at all. However, there is a large body of literature which attests to the prevalence of sensory symptoms within ASDs.

Sensory symptoms were featured in the original descriptions of ASDs (Asperger, 1944; Kanner, 1943), and further explored by Wing (1969) and Hermelin and O'Connor (1970). There are also the eloquent accounts written by people on the autism spectrum (e.g. Grandin, 1992, 2009; Grandin & Scariano, 1986; Jackson, 2002; Williams, 1998) or by their parents/caregivers (e.g. Jackson, 2003). Many of these accounts describe both hyper- and hypo-sensitivity to sensory stimuli – often fluctuating between the two extremes (Jones, Quigney, & Huws, 2003).

Despite the obvious value of their insight, there are problems with the interpretation of first-hand descriptions. Firstly, the reports tend to come from high-functioning individuals with ASD, so we do not know how well they apply to the autism spectrum as a whole. Secondly, an account may have been written in conjunction with someone who does not have ASD (e.g. Grandin & Scariano, 1986), risking the intrusion of interpreter bias. Lastly, O'Neill and Jones (1997) suggest that, for individuals with ASD, there can be confusion over 'real' versus 'echoed' memories and a lack of insight into typical perceptual experiences. A helpful summary of this literature is given by Bogdashina (2003).

A number of studies have collated this information by evaluating parent/caregiver questionnaires (Baker, Lane, Angley, & Young, 2008; Baranek, David, Poe, Stone, & Watson, 2006; Ben-Sasson et al., 2007; Robertson & Simmons, 2008; Rogers, Hepburn, & Wehner, 2003; Watling, Deitz, & White, 2001), sometimes combined with information from the individuals themselves (Kern et al., 2006, 2007; Leekam, Nieto, Libby, Wing, & Gould, 2007). These tell the broadly similar story that (a) particular sensory symptoms appear to be more common in ASD than in other developmental disorders; (b) these symptoms lessen with age; (c) these symptoms are correlated with the severity of social symptoms of ASD, at least in children (Kern et al., 2007).

There are a number of problems with caregiver/parent report data. Methodological shortcomings (such as small sample sizes and variability across groups) have affected the degree to which these results can be generalized, reporters may attribute the child's reaction to the wrong sensory stimulus and are open to bias (e.g. unwittingly over- or under-estimating the child's sensory difficulties; see Nader, Oberlander, Chambers, & Craig, 2004). There are also issues of recall bias, especially when recounting stressful situations.

Although it has been suggested that the importance of sensory symptoms from the report literature has not been confirmed in lab-based studies (O'Neill & Jones, 1997; Rogers & Ozonoff, 2005), we have recently found a strong correlation between sensory symptoms reported by members of the general public and

[2] Given the significant changes in diagnostic criteria in the years since ASD was first described, very careful attention should be paid to the group characterization in any studies which pre-date publication of DSM-IV in 1994. See Hincha-Ownby (2008) for an overview of these changes. Note also that these criteria will themselves shortly be updated when DSM-V is published (see American Psychiatric Association web-site for the latest developments: http://www.psych.org).

their score on the AQ which addresses some of the issues to do with third-party report (Robertson & Simmons, 2009).

5. Visual symptoms in ASD

A non-exhaustive list of visual sensory symptoms is given by Bogdashina (2003):

Hyper:

(1) focusing on tiny pieces of dust/particles,
(2) dislike of the dark and bright lights,
(3) dislike of sharp flashes of light,
(4) looking down most of the time,
(5) covering/closing eyes at bright lights.

Hypo:

(1) attracted to light,
(2) looking intensely at objects or people,
(3) moving fingers or objects in front of the eyes,
(4) fascination with reflections and/or brightly coloured objects,
(5) running hands around the edges of objects.

Another list is given in Leekam et al. (2007) and corresponds to questions asked in the DISCO diagnostic interview (Leekam et al., 2002):

Quotes from individuals on the autism spectrum are also informative, e.g.:

> *"my bed was surrounded and totally encased by tiny spots which I called stars, like some kind of mystical glass coffin. I have since learned that they are actually air particles yet my vision was so hypersensitive that they often became a hypnotic foreground with the rest of 'the world' fading away."*

-Donna Williams, p. 15, "Nobody Nowhere" (Williams, 1998).

There are also some commonly observed social symptoms which may have a visual component such as unusual socially directed pointing, difficulties with interpretation of gestures, unusual eye contact, difficulty with the interpretation of facial expressions, difficulty with following the gaze of others and difficulties with joint attention, as well as non-social signs like repetitive and stereotyped behaviours. Assessment of these symptoms is included in the ADOS (Lord et al., 2000).

6. Studies of visual processing in ASD

In this section of the review we shall deal with the full range of possible visual differences between ASD and typical control populations.

6.1. Optometric issues

Although many studies report screening the vision of their participants, relatively few have focussed specifically on optometric comparisons of ASD and typical populations. Scharre and Creedon (1992) reviewed previous findings and also evaluated 34 children (2–11 years) with autism for ocular alignment, refractive error, visual acuity, oculomotility skills and stereopsis. Scharre and Creedon (1992) noted a higher than average incidence of refractive errors (consistent with results from other developmental disabilities such as cerebral palsy), and also found that 21% of their children had strabismus, comparing this with figures of 3.7% for typical children, 69% in cerebral palsy and 21.4% in developmental delay from other studies. They also reported that a "significant number" of the children had difficulty with voluntary pursuit eye movements and had atypical OKN responses. They could not report on the level of amblyopia in their sample because most of the children resisted ocular occlusion, although only 3 of the 34 failed the Lang stereotest. Given that Sharre and Creedon (1992) pre-dates the publication of DSM-IV and ICD-10 diagnostic criteria it is possible that their data only apply to more severe cases of autism, as DSM-III criteria were much more restrictive (Hincha-Ownby, 2008). A high prevalence of strabismus in ASD (50%) was also reported by Kaplan, Rimland, and Edelson (1999) from a sample of older children (7–19 years) along with a slightly lower prevalence (20%) taken from a questionnaire survey of 7640 parents. Neither of these studies is definitive in terms of establishing optometric anomalies in ASD populations, since both relied on a previous clinical diagnosis by unspecified techniques, the numbers of participants in the experimental studies were relatively small and there were no control groups. The Kaplan et al. (1999) questionnaire data also suffer from the known problems with parental reports. Nevertheless, this finding would appear to be worthy of further investigation. It does seem surprising that the large number of researchers who have performed routine optometric screening of their participants have not noticed the high prevalence of strabismus in their ASD population (even if the conservative estimate of 20% applies). Kaplan (2006) has recently embedded these optometric data into his theory of visual dysfunction in autism.

6.2. Spatial vision

6.2.1. Visual acuity

The visual acuity of children and adults with ASD has been routinely measured as part of the screening procedure in a number of studies with nothing untoward being reported, but one very recent study focused exclusively on visual acuity (Ashwin, Ashwin, Rhydderch, Howells, & Baron-Cohen, 2009). They measured visual acuity in 15 individuals with ASD and 15 controls using the Freiburg Visual Acuity and Contrast Test (Bach, 1996) and reported a very high mean level of visual acuity in their ASD sample (20/7) compared to the 20/13 of the typical control group. Unfortunately it appears that the spatial resolution of the display screen was not high enough to support acuity measurement at the viewing distance used, and some of the settings on the computerized test were inappropriate (Bach & Dakin, in press). Consequently this apparently exciting result must be regarded as unsound.

6.2.2. Static contrast sensitivity

de Jonge, Kemner, de Haan, Coppens, and van den Berg (2007) used the Vistech® contrast sensitivity charts to compare performance at a range of spatial frequencies (1.5–18 c/deg) between a group of 29 people with ASD and age-matched controls. The clinical sample (7–33 years) was carefully diagnosed using multiple instruments including the ADI-R and ADOS. De Jonge et al. (2007) found no significant differences between the two groups. Obviously the clinical sample was relatively small, and the test was not as sensitive to subtle differences as a computer-based test would be, but it does suggest that there are no large differences in static contrast sensitivity between individuals with HFA and matched controls. Behrmann et al. (2006a) and Milne, Scope, Pascalis, Buckley, and Makeig (2009) have also reported no differences in static contrast sensitivity for gratings of different spatial frequencies between ASD and matched control populations.

Bertone, Mottron, Jelenic, and Faubert (2005) compared contrast thresholds for orientation identification between a group of 13 HFA participants (11–31 years) and matched typical controls. Diagnosis was using ADI and ADOS. The stimuli were either luminance- (i.e. first-order) or contrast-modulated (i.e. second-order) grayscale noise. The sinusoidal modulations had a spatial

frequency of 0.75 c/deg in both cases and were presented for 750 ms. The ASD participants were carefully guided through the task, with the experimenter remaining in the room, reminding the participants to fixate and initiating successive trials. This care will have served to reduce any noise in the data due to attentional lapses. The result found was surprising in that the HFA group obtained significantly *lower* thresholds with the first-order stimuli than the matched controls, but significantly higher thresholds with the second-order stimuli (see Fig. 1).

Sanchez-Marin and Padilla-Medina (2008) measured the detectability of a static bright bar embedded in Gaussian noise in six participants with autism (7–17 years) and six controls. The children with autism were characterized using the Childhood Autism Rating Scale (CARS; Schopler, Reichler, DeVellis, & Daly, 1980), although there was no attempt to match IQ with controls, which is unfortunate as the children with autism had relatively severe symptoms. Sanchez-Marin and Padilla-Medina (2008) found that children with autism performed significantly worse than controls at a range of signal/noise ratios. They argued that these data could reflect the influence of increased levels of internal noise in visual processing pathways in autism. Whilst this conclusion is very interesting (see Section 13 below), the experiment should have used a more carefully controlled group of participants.

6.2.3. Dynamic contrast sensitivity

Bertone et al. (2005) also measured contrast thresholds for a flickering grating stimulus using a temporal 2AFC paradigm. They used a conventional 0.5 c/deg grating counterphasing at 6 Hz (ostensibly to stimulate magnocellular pathways) and a 6 c/deg grating counterphasing at 1 Hz (ditto for parvocellular pathways). There were no significant differences in contrast sensitivity for either of these stimuli between the HFA group and controls. A similar result was found by Pellicano, Gibson, Maybery, Durkin, and Badcock (2005) using similar procedures and carefully diagnosed

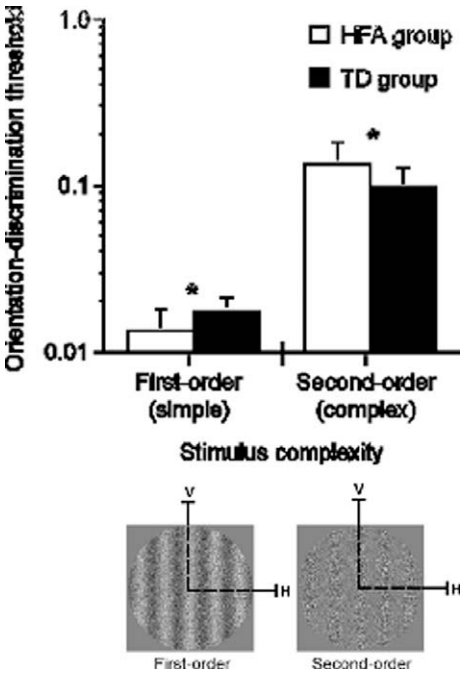

Fig. 1. Figure from Bertone et al. (2005) showing contrast thresholds for orientation identification (horizontal/vertical) in noise for first-order and second-order grating stimuli collected from children with high functioning autism (HFA) and typically developing controls (TD). The stimuli used are shown beneath the graph. Note that the units on the *y*-axis are contrast, not orientation. Reproduced with the permission of Oxford University Press.

and matched participants, except that the stimulus was a Gaussian blob (3.15 deg sigma) flickering sinusoidally at 10 Hz.

Bertone, Mottron, Jelenic, and Faubert (2003) reported contrast sensitivities for drifting grating stimuli defined by either luminance- (i.e. first-order) or contrast- (i.e. second-order) modulated grayscale noise. The gratings were either conventional vertical sinusoids, radially symmetric sinusoids or angled sinusoids in order to test translational, radial and rotational motion, respectively. For the translating and radial patterns the spatial frequency was 1 c/deg with a temporal frequency of 2 Hz, giving a drift rate of 2 deg/s. For the rotating grating the angular velocity was $\pi/2$ rad/s. The participants were a well diagnosed HFA sample (mean age 13 years) and a typical control group (mean age 12 years). Bertone et al. (2003) found no difference between contrast thresholds for first-order motion detection with these two groups, but did find significantly higher thresholds for second-order motion detection with the ASD group. There was no effect of motion type (see Fig. 2). Whilst this result has yet to be replicated, an interesting recent study by McCleery, Allman, Carver, and Dobkins (2007) does lend qualified support. They measured contrast thresholds for the detection of 0.27 c/deg gratings drifting upwards or downwards at 15.6 deg/s. Their sample was 13 6-month-old infants who each had older siblings that had been diagnosed with ASD. The chance of being diagnosed with ASD is 10–20 times more likely in this "high-risk" group than in the general population (Dawson et al., 2002b; Plomin & McGuffin, 2003) and the study was aimed at defining the characteristics of this population before ASD diagnosis was possible. Being infants, a preferential-looking technique was used to gather detection data. Curiously, they found that the contrast thresholds of this "high-risk" group for the drifting gratings were significantly lower than those of the control population. This was true even when those infants who were subsequently diagnosed with ASD (two) were removed from the analysis. McCleery et al. (2007) found no performance difference with similarly drifting isoluminant chromatic red-green gratings and interpreted their results in terms of differential sensitivities of M and P pathways in the high-risk and control groups.

Sanchez-Marin and Padilla-Medina (2008) also measured thresholds for detection of their bright bar stimulus when it was moving across the display. As with the static version of their task, they found that the children with autism were significantly worse at detecting the stimulus at a range of signal-to-noise ratios. However, the reservations about the methodology of this study presented above apply here too.

6.2.4. Contrast sensitivity summary

To summarise, no study with well-matched controls has demonstrated poorer contrast sensitivity in an ASD group where the stimuli are defined by luminance contrast. One study (Bertone et al., 2005) has demonstrated significantly lower thresholds in an ASD group for a static contrast sensitivity task, and this was also true for a so-called "high-risk" infant population when the stimulus was dynamic (McCleery et al., 2007). In both studies where the stimulus was "second-order" (i.e. defined by contrast modulations), the modulation thresholds of the ASD group were significantly higher than those of controls (Bertone et al., 2003, 2005).

6.2.5. Spatial grouping/contour integration

Several studies have compared performance of ASD and typical populations in contour integration tasks similar to those first employed by Field, Hayes, and Hess (1993). These have been targeted at evaluating the "weak central coherence" (WCC) theory, first put forward by Frith (1989) and most recently elaborated by Happé and Frith (2006). WCC theory suggests that individuals with ASD have difficulty integrating information, including visual information, from different spatial and or temporal sources. However,

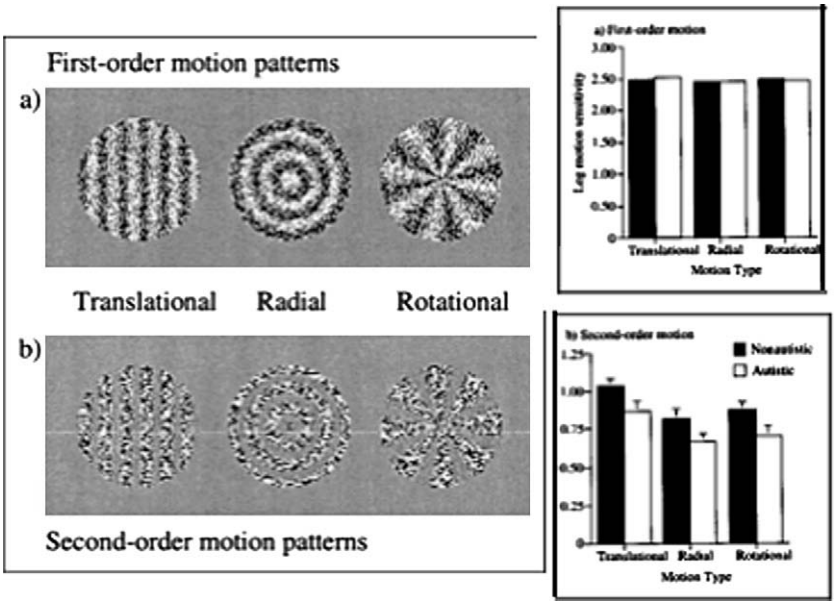

Fig. 2. Stimuli and data from Bertone et al. (2003). Reproduced with permission of MIT Press.

Dakin and Frith (2005) criticized a number of these contour integration studies for using oriented line elements, arguing that the target contour would be detectable simply by low-pass filtering the stimulus, and therefore that they were not true tests of contour integration, but merely contrast detection. This criticism applies to the studies by Spencer et al. (2000), Blake, Turner, Smoski, Pozdol, and Stone (2003) and Milne et al. (2006) (none of which demonstrated a significant difference between ASD and control groups). More recently, however, two studies using Gabor elements – which are not prone to the same criticism – have both shown no significant differences between ASD and control populations in this task (Del Viva, Igliozzi, Tancredi, & Brizzolara, 2006; Kemner, Lamme, Kovacs, & van Engelund, 2007). Thus there is no evidence for a performance deficit in contour integration tasks amongst ASD populations. Two caveats should be added, though. The first is that only high-functioning ASD populations and controls have been compared and the second is that all of these studies used closed, rather than open contours.

Two recent related studies have reported a deficit in visual form processing in ASD. Spencer and O'Brien (2006) used oriented "Glass" patterns composed of correlated dot triplets. Structured elements were intermixed with randomly oriented elements (see Fig. 3) to obtain a threshold coherence measure for locating the circular patch either side of the display centre. The display was designed to match as closely as possible a similarly constructed motion coherence display (see below). Unusually there were three participant groups: an autism group of 15 (mean age 13.5 years), an Asperger group of 10 (mean age 12 years) and a typical group of 15 (mean age 12 years). The groups were matched for CA and VMA using the British Picture Vocabulary Scale (BPVS; Dunn, Dunn, & Whetton, 1982). Note that both autism and Asperger groups had IQ greater than or equal to 70 and therefore were both "high-functioning". Spencer and O'Brien (2006) reported a significant threshold difference between their autism group and both the typical controls (35.5% higher) and the Asperger group (26% higher) in the form coherence task. This result is surprising, given negative results in this sort of task from other groups and that it differentiates performance between AS and HFA. If replicable, it is important in suggesting that task performance might depend on position on the autism spectrum (assuming, of course, that HFA and AS correspond to differing severities of ASD). However,

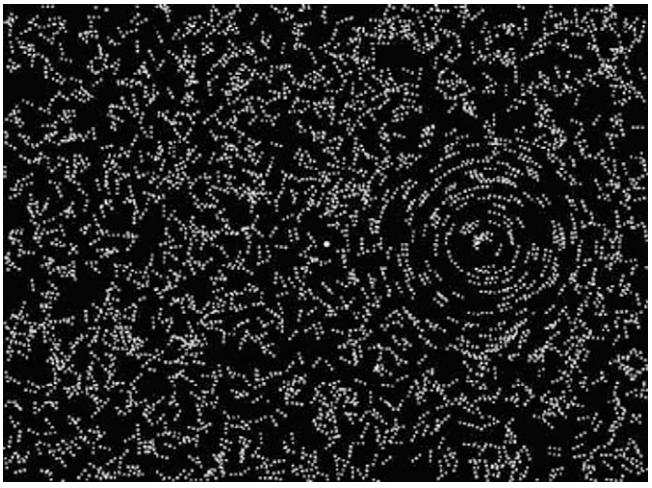

Fig. 3. The Glass pattern stimulus used by Spencer and O'Brien (2006) in their study of motion and form coherence in autism. Reproduced with the permission of Pion Limited, London.

there are some problems with this study. First, the method of diagnosis: it is simply stated that they "met the relevant diagnostic criteria in DSM-IV". If task performance were related to ASD severity a quantitative assessment of this would have been desirable (see, e.g., Blake et al., 2003). A second concern is about the stimuli. Although these were Glass patterns rather than line elements, the same problems of low spatial-frequency luminance artifacts raised by Dakin and Frith (2005) apply. However, this would not explain the pattern of the results unless somehow the HFA group was insensitive to these luminance cues for some reason, but the AS and control group were. Finally, the presentation time was exceptionally brief for experiments of this kind (250 ms), and too brief for participants to make stimulus-elicited eye movements. It is plausible that the deficit in the HFA group was not one of form coherence *per se*, but the problem of dividing attention between two halves of a stimulus display, the centres of which were separated by 15.4 deg of visual angle (cf. Plaisted, Swettenham, & Rees, 1999, but also Rutherford, Richards, Moldes, & Sekuler, 2007b).

This argument is reinforced by the fact that a similar size of deficit was found in the matched motion coherence task (see below). Tsermentseli, O'Brien, and Spencer (2008) replicated the pattern of Spencer and O'Brien's (2006) data in a sample of adults, lending force to the argument for a form processing deficit specific to autism diagnoses, but not Asperger Syndrome. Some of the same criticisms apply to this study. Tsermentseli et al. (2008) suggest that the presence of superior language skills in the Asperger group might be related to the differential performance which, as with Spencer and O'Brien (2006), seems to carry over to the motion coherence task as well.

Further support for the view put forward by Spencer and O'Brien (2006) and Tsermentseli et al. (2008) is provided by Brosnan, Scott, Fox, and Pye (2004). They looked at a variety of tasks in a group of 25 children with autism and 25 age- and VMA-matched controls. The mean CAs were about 10 years and the mean VMAs about 5 years, meaning that the control group had moderate learning difficulties. The tasks utilized the gestalt grouping rules of proximity, similarity and closure in simple line and dot figures. Brosnan et al. (2004) found that their autism group were effectively at chance in these tasks, unlike the controls, and also impaired on identifying impossible figures, although this deficit was not so pronounced on having to draw reproductions of them (cf. Mottron & Belleville, 1993; Mottron, Belleville, & Menard, 1999a). A similar result has been found by Bölte, Holtmann, Poustka, Scheurich, and Schmidt (2007) in a group of adults with HFA.

6.2.6. Static shape perception

De Jonge et al. (2007) found normal shape discrimination in their ASD population (squares vs. rectangles matched in area). However, Ropar and Mitchell (2002) found a curious failure of shape constancy in their group of children with autism. The task involved setting the shape of an ellipse on a computer screen to match the shape of a circle viewed in a special chamber, but tilted away from the viewer. Control participants typically set the ellipse to be more circular than it looks due to the knowledge that it is really a circle, and this effect is strongest when the circle is presented by itself and without any perspective cues. However, the autism group did not show this effect and set the shape of the ellipse more accurately, suggesting that they were not so influenced by the knowledge that the shape was a circle. Ropar and Mitchell (2002) suggested that this result shows that individuals with autism are less influenced by prior knowledge in visual judgements and consequently visual processing may be less 'top-down' dominated in autism.

6.2.7. Susceptibility to visual illusions

Happé (1996) was the first to suggest that ASD populations were less susceptible to these illusions (specifically the Ponzo, Poggendorff, Ebbinghaus/Titchener, Hering and Kanisza triangle, but not the Müller-Lyer). These claims have been supported by Bölte et al. (2007). Happé's (2006) original findings were, however, disputed by Ropar and Mitchell (1999, 2001). Commentators (e.g. Dakin & Frith, 2005; Happé & Frith, 2006) have suggested that this apparent discrepancy could be due to "methodological differences" between the studies. In particular, in Happé (1996) participants were asked to judge whether the elements in an illusion display (normally perceived as different in size) were "the same" or "different". Many of the ASD participants reported "the same", suggesting that they were not susceptible to the illusion. Ropar and Mitchell (1999, 2001) used a computer-based method for adjusting the relative sizes of the illusory-sized elements and found no significant differences between their ASD population and controls. No significant difference between ASD and control groups in illusion susceptibility has also been reported by Hoy, Hatton, and Hare (2004) and Milne and Scope (2008).

An interesting recent study by Walter, Dassonville, and Bochsler (2009) may have clarified this discrepancy. Walter et al. (2009) used a large sample of 146 undergraduate students who performed a battery of tasks including psychophysical tests of a number of standard illusions and the Autism Spectrum, Empathising and Systemising Quotient questionnaires (AQ: Baron-Cohen et al., 2001b; EQ: Baron-Cohen & Wheelwright, 2004; SQ: Baron-Cohen, Richler, Bisarya, Gurunuthan, & Wheelwright, 2003, respectively), which measure traits associated with autism in the general population. Walter et al.'s (2009) key result was that susceptibility to the Zöllner, Rod-and-frame, Roelofs, Ponzo and Poggendorff illusions related to score on the SQ, such that immunity to these illusions was associated with a high Systemizing Quotient. This suggests that previous results with populations on the autism spectrum (only one of the students scored high enough on the AQ to be considered a possible candidate for diagnosis) may have been affected by this trait, which is associated with ASD, rather than the diagnostic category in itself. Walter et al. (2009) also make a number of pertinent criticisms about methodology in previous studies of illusory susceptibility.

Brown, Gruber, Boucher, Rippon, and Brock (2004) looked at a different illusion: the Kanisza triangle. They found no difference in behavioural performance between their six adolescents with autism and matched learning-disabled controls but there were anomalies in the simultaneously recorded EEG responses. In particular the induced activity in the gamma band (25–70 Hz) over parietal regions was different from that of the control group. A recent technical paper has cast doubt on the integrity of some induced gamma band activity recorded with EEG, suggesting that if it has the profile illustrated by Brown et al. (2004) it is most likely an artifact due to small-amplitude saccadic eye movements (Yuval-Greenberg, Tomer, Keren, Nelken, & Deouell, 2008). Any study which does not control for these eye movements can no longer claim to demonstrate neural gamma oscillations when measured in this way, so Brown et al.'s (2004) results may well be reflecting differential fixational eye movement activity between the diagnostic groups, rather than anything significant about neural responses. Some ideas about how to control and interpret these artifacts have recently been suggested by Melloni, Schwiedrzik, Rodriguez, and Singer (2009).

6.2.8. Visual completion

A different approach to exploring context sensitivity effects within ASD was taken by De Wit, Schlooz, Hulstijn, and van Lier (2007), who used a shape completion task. In shape completion tasks a shape (e.g. a circle) is initially presented partially occluded behind another shape (e.g. a rectangle). After a short break the participant is then presented with two alternative shapes and has to choose which one they think they saw (Sekuler & Palmer, 1992). De Wit et al.'s (2007) variation involved using a shape completion priming task. The occluded shape was presented as a prime. Participants were then presented with two different (or identical) alternative shapes and asked to speedily report whether they were the same or different. Depending on the type of prime stimulus this judgement was performed either as fast or more quickly when the prime was in place than when it was not. The argument is that priming effects show evidence of different types of shape sensitivity. De Wit et al.'s (2007) clinical sample was slightly unusual in that they chose to focus on PDD-NOS, defined by the presence of only a subset of the usual symptoms of autism. In total there were 19 participants in their clinical group (mean age 12 years): 16 PDD-NOS and 3 AS. The control group was matched for CA and IQ. Their pattern of results was quite complicated. The clinical group had largely similar reaction times to controls, but differed

in the pattern of priming stimuli that were effective. De Wit et al. (2007) argued that their results demonstrate that their PDD group was able to integrate context so as to complete partially occluded stimuli effectively, but they had somewhat more difficulty with unusual/unfamiliar or complex shapes. This may indicate a greater difficulty in learning novel shapes (or a greater sensitivity to differences between them).

6.2.9. Reading

The published data on reading ability in ASD show a large amount of variability (Whitehouse & Harris, 1984). Recent studies have suggested that the problems that people with ASD have with reading are dissociable from their symptoms (Ludlow, Wilkins, & Heaton, 2006; White et al., 2006).

6.3. Colour vision

There are a large number of anecdotal reports of unusual responses to colour among people on the autism spectrum (e.g. Williams, 1999; see also Franklin, Sowden, Burley, Notman, & Alder, 2008; Ludlow et al., 2006). Individuals on the autism spectrum can display strong affinities to, or aversions from, objects of particular colours (Ludlow & Wilkins, 2009; Moore, 2004). A few studies have reported incidental effects of colour: Brian, Tipper, Weaver, and Bryson (2003) found an unexpected facilitation effect of colour in their study of inhibitory mechanisms in ASD and Greenaway and Plaisted (2005) found a similar effect in a cueing task, where invalid colour cues resulted in greater costs for individuals with ASD than for controls.

The first dedicated study of colour vision in ASD was Ludlow et al. (2006), who tested the efficacy of coloured overlays on the reading performance of a group of children on the autism spectrum and a gender-, age- and VIQ-matched control group. The ASD group showed a modest but significant average improvement in reading speed of 13% with the overlays in place. Note that part of Ludlow et al's (2006) screening process used the City and Ishihara colour vision tests, and nothing unusual was found, but this was too small a sample to assert that colour vision is clinically normal in ASD. Ludlow et al. (2006) couch their explanation of the reading improvements observed in the ASD group in terms of Wilkins' (2003) theory that appropriately chosen coloured filters can limit the spread of activation in hyper-excitable areas of visual cortex (see also Ludlow & Wilkins, 2009).

Three very recent studies deal directly with the visual processing of chromatic information in ASD: Heaton, Ludlow, & Roberson, 2008; Franklin et al., 2008, in press. Heaton et al. (2008) used three groups of 13 children and adolescents (mean age ~11 years). One group was from a school specializing in autism and thus was assumed to meet appropriate diagnostic criteria, one from a school for children with moderate learning difficulties (MLD) and one group of typically developing (TD) children. Groups were all individually matched for chronological age and the MLD and autism groups were matched for non-verbal MA (assessed using Raven's matrices). However, VMA (measured using the BPVS) was significantly different between all three groups, with the autism group having the lowest (mean ~5.5 years). After checking that all groups could name and distinguish the 11 basic colours correctly, they performed a discrimination task: three coloured patches, two the same and one differing by a small step in Munsell hue space were presented on a computer screen and participants chose the odd one out. Both autism and MLD groups were impaired on this task relative to the TD controls, although not with respect to each other. Performance in this task correlated with VMA. The second experiment tested colour memory. The same children were taught to associate pictures of familiar animals with "focal" colours (i.e. red, green, blue and yellow) then subsequently tested by presenting the animal together with the four colours, with the children being asked to choose which one matched the animal. In this phase of the experiment the autism and MLD groups were just above chance, but the TD group performed quite well. In the second phase of the experiment, after a memory refreshment, the participants were presented with three alternative colours for each animal picture, but this time each colour was drawn from the same colour category (e.g. three shades of red). In this phase of the task only the autism group scored above chance. In fact, when calculated as a *z*-score, the performance of the autism group was about the same in both phases of the task. Subsequent analysis showed that phase 1 performance correlated significantly with both VMA and discrimination performance from the first experiment, but in phase 2 there was a significant *negative* correlation with VMA, suggesting that the children with lowest VMA performed best in the task. Heaton et al. (2008) suggest that there is a profound link between verbal ability and perceptual discrimination and that typically developing children apply verbal labels to the colours, and therefore are confused in the memory task when presented with colours which have the same verbal label. Children with language difficulties, on the other hand, are forced to remember the colour perceptually, and thus are less confused so long as the colours are discriminably different. This differential performance is reminiscent of the performance of ASD groups in the Embedded Figures Task (Jolliffe & Baron-Cohen, 1997; Shah & Frith, 1983) where a learned grouping of information disrupts performance in a TD group, but not in a group with ASD.

Franklin et al. (2008) worked with 19 children with HFA (7–13 years) attending specialist schools and 14 CA- and NVIQ- (Raven's Matrices) matched controls. Importantly, none of the children were diagnosed with ADHD, as it has been shown that these children tend to have blue-yellow colour deficiencies (Banaschewski et al., 2006). Franklin et al. (2008) specified their stimuli in CIELAB colour space which were either coloured patches or abstract form stimuli, constructed from a standard set (Pick, 1965). The first experiment consisted of a visual search task and a visual memory task. In the search task, participants were asked to spot the odd coloured patch or the odd form in an array of distracters. The memory task was a delayed match-to-sample, with two choices, one the same as the target and one foil, differing slightly in colour or in form (curvature). Having checked that there was no interaction, data from the two tasks were combined, and it was found that the ASD group was impaired on the colour task, but not the form task. The second experiment was performed with two groups of 14 slightly older children (11–13 years). The experiment tested categorical perception of colour across the blue–green boundary (Franklin, Pilling, & Davies, 2005). The 2AFC task was to locate the presence (i.e. left or right of centre) of a coloured target on a coloured background. The target was either drawn from the same or different colour categories. Accuracy on the task did not differ between within- and between-category judgements, but the ASD group performed significantly worse than the controls. Reaction times (RTs) showed a category boundary effect, with the task being performed more quickly by both groups when the colours were sampled from different categories. However, there were no significant differences in RT between the groups.

Franklin et al. (in press) tested similarly characterized groups on the Farnsworth–Munsell 100-hue test (Farnsworth, 1943), and a conventional chromatic discrimination task which involved detecting the orientation of the boundary between two isoluminant colours. In both experiments there were control tasks which used stimuli differing only in luminance. As with Franklin et al. (2008) the ASD group performed significantly worse on the colour experiments with higher error scores on the FM-100 hue test and higher thresholds on the chromatic discrimination task. However, no significant differences were found in the luminance tasks. Note that the chromatic discrimination difficulties of the ASD group

were not confined to a particular axis of colour space, such as the red–green or blue–yellow opponent axes.

The conclusion from this series of studies is that children with ASD are challenged in a range of chromatic discrimination tasks compared to typical controls matched on non-verbal intelligence and these difficulties do not transfer to similar luminance discrimination tasks. Franklin et al. (in press) estimate that performance of the ASD group in the FM-100 task is comparable to that of children 3 years younger. It is unfortunate, however, that Franklin et al. (2008, in press) did not report VMA in their studies, given the potential importance of this factor (Heaton et al., 2008). Verbal ability is known to be closely linked to colour naming performance in young children (Pitchford & Mullen, 2002) and a very recent ERP study in mono- and bi-lingual adults has suggested that colour language can affect the very earliest stages of perception (Thierry, Athanasopoulos, Wiggett, Dering, & Kuipers, 2009). Also, all three studies (Franklin et al., 2008, in press; Heaton et al., 2008) failed to characterize fully the diagnostic status of their participants. Nevertheless, these results are exciting and, if they are found to be specific to ASD, have profound implications for our understanding of early visual processing in this condition.

6.4. Depth perception and stereopsis

There are three lines of evidence which suggest that the perception of depth in ASD merits further attention. First, there is a range of clinical and anecdotal reports about depth perception being unusual in ASD. Kaplan (2006), for example, notes his observations that people with ASD often mis-judge inter-personal distance during social interaction and have difficulties with tasks such as ball-catching. Second, there is evidence for a higher incidence of strabismus in ASD populations (Kaplan et al., 1999; Scharre & Creedon, 1992), which would suggest that binocular vision and stereopsis might also be affected. Third, because stereopsis requires a precise developmental registration of information from each eye, it is developmentally fragile (Atkinson, 2000). Depth perception and stereopsis in ASD would seem to be an area where at the very least a screening study is warranted.

6.5. Motion perception

Motion perception is one of the most-studied and most controversial areas in the field of vision in ASD. Reviews by Dakin and Frith (2005) and Milne, Swettenham, and Campbell (2005; plus associated peer commentaries) cover the literature up to 2005 in considerable detail, so we will concentrate on more recent studies.

6.5.1. Local motion

Surprisingly, only one published study has examined low-level local motion processing in an ASD population: the Bertone et al. (2003) study described in detail above (see Fig. 2). Bertone et al. (2003) found no significant difference in contrast thresholds for first-order motion direction identification between their ASD and control groups, but there was a relative deficit in second-order motion processing, with the ASD group exhibiting significantly higher modulation thresholds for the direction identification task. Published criticisms of Bertone et al. (2003) have tended to focus on the explanation of the result in terms of the "complexity" of motion processing, rather than questioning the result itself (see Dakin & Frith, 2005; Jarrold & Scott-Samuel, 2005; Mitchell, Ledgeway, & Landry, 2005). Interestingly, Kogan et al. (2004) measured local motion processing in Fragile-X Syndrome (FXS) – a genetic disorder which shares some of its symptomatology with ASD – using similar techniques and stimuli to Bertone et al. (2003). Kogan et al. (2004) found that modulation thresholds for local motion direction identification were impaired for *both* first- and second-order stimuli in this population, suggesting that the pattern of results found by Bertone et al. (2003) is particularly specific to ASD (see also Bertone & Faubert, 2006).

Vandenbroucke, Scholte, van Engelund, Lamme, and Kemner (2008) recently examined two-grating plaid motion processing in a group of adults with HFA and typical controls, matched for CA and IQ. They found no significant differences between the participant groups in the relative amount of time that the plaid was seen moving as a coherent whole, rather than as two transparent components. There were also no significant differences between groups in the rivalry rate. They concluded that there was no evidence for a difficulty with combining motion information in their ASD population, invoking an explanation in terms of the spatial frequency content of their displays, which was predominantly low. They argued that individuals with ASD may have less difficulty processing motion of low than high spatial frequency gratings, comparing their data with that of Bertone et al. (2003) and the motion coherence studies considered below. Given this explanation it is unfortunate that Vandenbroucke et al. (2008) did not use sinusoidal gratings as their plaid components, as this would simplify the interpretation of their results, but they certainly suggest that the perception of high-contrast two-component motion is unaffected in adults with HFA. A further useful piece of information, given the results of Spencer and O'Brien (2006) and Tsermentseli et al. (2008) discussed below, would be how many of their HFA group could be characterized as having Asperger Syndrome.

6.5.2. Motion coherence

A typical motion coherence stimulus consists of a large number of randomly moving dots of which a small proportion move coherently in a given direction and give a fleeting perception of motion (Newsome & Paré, 1988). Threshold for the task is defined as the percentage of dots required to be moving coherently before the observer can reliably report their direction of motion. Usually this is run as a 2AFC task, with the motions being up vs. down or left vs. right. Newsome and Paré (1988) originally used this stimulus as a probe for investigating the efficacy of microscopic lesions in Area V5/MT of macaque monkeys on the monkeys' motion discrimination abilities.

Wattam-Bell (1994) developed a slightly different version of this stimulus for use in preferential looking tasks with infants, which Atkinson and Braddick (2005) describe as a "road in the snowstorm" stimulus. In this stimulus the signal dots oscillate backwards and forwards in a horizontal direction and the noise dots appear transiently for the same duration (120 ms) in random locations. In one half of the display the signal dots all move in the same direction. In the other half of the display a central strip contains dots moving in the exact opposite direction. This results in a segmented percept rather like looking at a road in a snowstorm. This was precisely the class of stimulus used by Spencer et al. (2000) to look at motion coherence thresholds in an ASD population. Their sample of 23 children with "autistic disorder" (diagnostic technique not specified) had significantly higher motion coherence thresholds than the CA-matched controls (see Table 1). There was a smaller and non-significant difference between ASD and control performance in a similar form coherence task. Interestingly, motion coherence thresholds in the ASD group did decrease with age (7–11 years), as did those of the controls, although the ratio of ASD/control performance remained about the same in all age groups. Also, whereas control performance reached adult levels by the age of 11, this was not true of the ASD group, although there were no data on teenagers with ASD to test whether motion coherence thresholds were consistently higher through to adulthood or just developmentally delayed.

Milne et al. (2002) criticized Spencer et al. (2000) for not matching their control population for IQ. Milne et al.'s (2002) sample

Table 1
Stimulus parameters from studies on motion coherence. Note that ** denotes a significant difference and * a marginal difference between coherence thresholds.

	Dot diameter (deg)	Coherent motion speed (deg/s)	Total dots per frame	Dot density (dots/deg^2)	Display duration (ms)	Dot lifetime (ms)	Results (%) A = ASD C = CONTROL
Spencer et al. (2000)	??	5.8	2000	4	330[a]	17	A: 25.5** C: 17.5
Milne et al. (2002)	1 pixel (=0.1?)	8.8	150	0.3	1010	224	A: 25.0** C: 15.3
Pellicano et al. (2005)	0.1	6.3	100	0.4	600	30	A: 22.4** C: 11.1
Milne et al. (2006)	0.1	7	300	2.1	85[b]	85	A: 17.2* C: 10.3
Spencer and O'Brien (2006)	??	5.8	5655	4	166	50	Aut: 46.2** Asp: 28.7 C: 24.6
Del Viva et al. (2006)	0.4	10	100[c]	0.4	160	66	A: 4.9 Cl: 6.9 C2: 5.5

[a] Before direction reversal. The total duration was self-limited.
[b] Also before direction reversal. Total duration 2300 ms.
[c] 50% black; 50% white.

consisted of 25 children with ASD (9.5–15.5 years) and 22 typical controls matched for CA and NVIQ (which was in the normal range for both groups). The diagnostic method was "according to DSM-IV criteria". The children were also all attending specialist schools. Milne et al.'s (2002) stimulus was closer to the Newsome and Paré (1988) style with a single display region and the task being to identify the direction of coherent motion. The display dynamics were also slightly different. A central fixation cross was present throughout stimulus presentation which the children were instructed to fixate. Mean thresholds for the ASD and control groups gave a similar performance ratio to that of Spencer et al. (2000). As with Spencer et al. (2000), the range of performance was greater in the ASD group (6–64% rather than 6–29%), but the inter-group difference was still significant when two of the ASD group with very high thresholds were removed from the calculations.

The studies of Spencer et al. (2000) and Milne et al. (2002) seemed to provide a coherent story: motion coherence thresholds were significantly higher in juvenile ASD populations, consistent with either Magnocellular pathway or "Dorsal stream vulnerability" arguments about the neural symptoms of ASD (Braddick, Atkinson, & Wattam-Bell, 2003; Milne et al., 2005). However, results from more recent studies have complicated this interpretation.

The most extreme position is occupied by Del Viva et al. (2006). In their version of the paradigm a display of 100 black and white dots was used on a gray background. Their ASD population was carefully diagnosed using ADI-R and ADOS-G and they excluded participants with genetic syndromes. They also used two control groups: one matched on CA and the other matched on VMA. Their motion stimuli were based on optic flow stimuli previously used by Morrone, Burr, and Vaina (1995) including rotational, translational and radial motion. They found no overall difference in coherence thresholds between their ASD group and either of the control populations.

The other extreme amongst recent papers is represented by Pellicano et al. (2005). They found a highly significant difference between global dot motion thresholds of their ASD and control groups, without any overlap in the 95% confidence intervals based on the data although, as usual, the variance on the ASD population's thresholds was considerably larger than that of the control group. The other recent studies which have looked at motion coherence obtained results between these two extremes. Spencer and O'Brien (2006) divided their participants into those with HFA and those with AS and found that motion coherence thresholds differed significantly from controls for the HFA group, but not the AS group. This pattern of results was confirmed in an adult population by Tsermentseli et al. (2008). Milne et al. (2006) also found that only a sub-group of their ASD population (about 20%) had motion coherence thresholds outside the typical range.

Recent data using the motion coherence paradigm to compare performance of children with and without ASD thus gives completely conflicting results ranging from no difference, to partial difference to complete difference, making it very difficult to draw firm conclusions on what the data mean. Clearly there are methodological differences between the studies that may explain the conflicting data. As this is such a well-studied and important area of vision in ASD it is worth considering these in some detail.

Both Del Viva et al. (2006) and Pellicano et al. (2005) took considerable care in the diagnosis of their ASD populations. As mentioned above Del Viva et al. (2006) used both ADI and ADOS and excluded genetic syndromes from their population. Pellicano et al. (2005) did not use the ADOS, but they did confirm diagnoses using ADI. They also screened their control population using the Social Communication Questionnaire (SCQ; Rutter, Bailey, & Lord, 2003). Del Viva et al. (2006) used two control populations, one matched for CA and the other matched for VMA, but they found no difference between them. Pellicano et al. (2005) had twice the number of ASD participants (20 vs. 10) and matched on NVIQ, as measured by Raven's Standard Progressive Matrices. Pellicano et al. (2005) point out that the VIQ of their comparison populations was different (means of 119 and 137, respectively, for ASD and controls) but that the receptive language of the ASD group was adequate for understanding task instructions. Here we have the first potential difference between the two studies, although Del Viva et al.'s (2006) second control group (C2), matched on CA, would probably have been the most similar to the controls of Pellicano et al. (2005).

The stimuli used by these two studies were, however, substantially different. Detailed parameters are presented in Table 1. Key differences are that although both studies used the same number of dots in their displays, those of Del Viva et al. (2006) were black *and* white on a gray background, were four times larger in diameter, and moving about 1.6 times faster than those of Pellicano et al. (2005). The display dynamics were also different. In Del Viva et al. (2006), total duration was brief, at 160 ms, with an individual dot lifetime of 66 ms, corresponding to four frame refreshes of the display. In Pellicano et al. (2005) the overall stimulus duration was much longer (600 ms) although the dot lifetime itself was shorter

(30 ms). In fact, Pellicano et al.'s (2005) paradigm was specifically designed to prevent participants tracking individual dots through consecutive frames because the dots carrying the coherent signal were randomly switched on each frame refresh (they criticized Milne et al. (2002) for having too long a dot lifetime). On the face of it, one would expect the briefer presentation time of Del Viva et al. (2006) to cause more problems for the ASD participants, but one factor that may be at work is the differential correspondence demands in the two sets of stimuli. As Del Viva et al. (2006) used two dot colours, larger dots, a slightly longer dot lifetime and a brief overall presentation (too short to initiate an eye movement) perhaps participants were less likely to mis-combine signal dots with noise dots. Barlow and Tripathy (1997) discuss and model correspondence noise in these classes of stimuli in considerable detail. The interesting implication of this observation is that children with ASD may be more susceptible to correspondence noise than their typical counterparts. Some ideas of why this might be are dealt with in the theory section below.

There are other differences in methodology between Del Viva et al. (2006) and Pellicano et al. (2005) that may be important. Dakin and Frith (2005) pointed out that the use of staircase routines is widespread in threshold assessments in ASD populations. Staircase routines have the obvious advantage of speed over constant-stimulus designs, but they are also susceptible to influence by finger errors by the participant which may mis-direct the threshold search in an unusual direction. Pellicano et al. (2005) used a PEST method to find their motion coherence thresholds which converged on the 75% correct point. They averaged all points following the fourth reversal to determine threshold. Pellicano et al. (2005) also employed auditory feedback. Del Viva et al. (2006) used a QUEST routine to alter stimulus levels, but then fitted the resultant proportion correct data with Weibull functions, also using 75% correct as the threshold criterion. They did not give any trial-by-trial feedback to participants and their data collection sessions were slightly longer. Del Viva et al.'s (2006) method was therefore the more robust of the two.

This leaves us really with two ways of dealing with the current contradictions in the data on motion coherence in ASD. One is simply to indicate that Del Viva et al. (2006) used the most carefully diagnosed population and the most robust psychophysical techniques and therefore that their result indicating no significant differences between ASD and control populations should be regarded as the most reliable. The other is to consider that the other studies did produce useful and informative results that may be amalgamated by examining the different methodological conditions. Del Viva et al. (2006) report their motion coherence data as sensitivities. Converting to coherence *thresholds* (see Table 1) they are considerably lower than those in other studies (for both ASD and control populations). As discussed, the larger level of correspondence noise in Pellicano et al. (2005) may have contributed to this difference. Milne et al. (2006) used quite a long duration and also required participants to detect which side of the display the oscillating target was presented. These would have presented a greater challenge to the ASD group, but they also used relatively long dot lifetimes, which may have allowed for a bit more tracking. It is curious that the threshold range in the ASD group in Milne et al. (2006) is actually very similar to that in Milne et al. (2002) when a significant group difference was reported. It seems as though there was a more heterogeneous ASD population in the later study and there was a more homogeneous control population. In any case, the correspondence noise argument would suggest a result for Milne et al. (2006) between the extremes set by Del Viva et al. (2006) and Pellicano et al. (2005). Spencer and O'Brien (2006) split their ASD population into Asperger and autism groups (a potential confound in other studies). Overall their coherence thresholds were much higher than the other studies (see Table 1), probably due to the higher dot density and larger number of dots in the display.

In sum, it seems as though motion correspondence, the "purity" of the ASD population and the psychophysical methodology, can go a long way towards explaining the discrepancies in the published data on motion coherence in ASD.

6.6. Optic flow

Gepner, Mestre, Masson, and de Schonen (1996b) were the first to suggest that there might be difficulties with motion perception in ASD populations. When children with ASD were asked to stand on a force platform and were presented with a large optic flow field, they were less posturally reactive to the motion than typically developing controls. A follow-up study showed additional differences between a small number of children with autism and a developmental delay, who were still less posturally reactive, and another small group with Asperger Syndrome (and typical IQ) who were *more* posturally reactive than typical controls (Gepner & Mestre, 2002). Clearly this result may involve both unusual responses to optic flow information and/or abnormal postural control (Milne et al., 2005). However, the methodology of both Gepner et al. (1996b) and Gepner and Mestre (2002) has been heavily criticized by Jarrold and Scott-Samuel (2005). Furthermore, Del Viva et al. (2006), who also measured responses to optic flow stimuli in the study discussed in detail above, found no differences between ASD and control groups.

6.7. Biological motion

"Biological motion" refers to the representation of human or animal actions using point-light displays (PLDs), generated by placing lights or reflective patches onto key anatomical points of a moving actor and then filming the result (Johannsson, 1973; see Blake & Shiffrar, 2007, for a review).

Moore, Hobson, and Lee (1997) presented 5- and 10-point PLDs depicting a walking person and various moving household objects (e.g. scissors opening and closing) to a group of 17 children/adolescents with autism (age 10–19 years). The control group was matched in CA and performance on the BPVS. As all of the ASD group had impaired language processing, the control group were learning disabled although not diagnosed with ASD. Stimulus presentation accumulated gradually from 40 ms to 5000 ms. Participants were asked to "name what the dots are stuck to". Psychometric functions showed the number of participants in the sample that were able to name the object correctly as a function of time, so they were in some sense comparable to average reaction times, although the cumulative presentation method meant that the total exposure time to each stimulus was also cumulative. Moore et al. (1997) reported that there were no significant differences in performance between ASD group and controls for this task, either with point-light walkers or with the moving objects, but they found that the ASD group did have difficulty with spontaneously describing and recognizing portrayals of emotion in PLDs, despite being able to accurately describe the mechanics of the motion itself. Moore et al. (1997) therefore suggested that the visual processing of biological motion is intact in ASD, but that the problems come with the interpretation of the internal states of others.

Blake et al. (2003) questioned the results of Moore et al. (1997), arguing that the verbal report responses and cumulative presentations were susceptible to bias. Blake et al. (2003) used a more robust and conventional psychophysical procedure: a 12-point-light actor performed a familiar activity (e.g. running or jumping), but each motion sequence also had a "phase scrambled" version in which dots underwent the same motion trajectory but offset by a random temporal phase difference. Previous work (Bertenthal &

Pinto, 1994) had shown that "scrambling", while retaining local motion, diminishes the ability of observers to organize a point-light display into a human activity. The advantage of comparing performance with these two stimulus variants is that the low-level visual structure of the stimuli is the same, the only difference being the relationships between the dots. Blake et al. (2003) calculated a d' score for the biological motion task and found that their typically developing group performed significantly better in this task than their ASD group with mean d' of close to 2.5 and close to 1, respectively. This is despite the two groups showing equivalent performance on a control "pathfinder" task (discussed above). In addition, Blake et al. (2003) plotted the individual d' scores for the ASD group as a function of their "level" of autism as measured by the ADOS and CARS tests and found a significant negative correlation, although there was also a correlation, only within the ASD group, with MA. Note that some 25% (4/16) of Blake et al.'s (2003) original ASD sample could not complete the biological motion task and 3 of these could not complete the form task either. These were all children with lower scores on a test of expressive language.

What are the potential causes for the discrepant results between Blake et al. (2003) and Moore et al. (1997)? The Blake et al. (2003) ASD sample was younger than that of Moore et al. (1997), being between 8 and 10 years old, rather than averaging 14 years, and were diagnosed more robustly. The control group was also younger, and matched on CA to the ASD sample's MA. The main difference, however, was the nature of the stimuli. Blake et al.'s (2003) participants were given a 1-s presentation of a randomized stimulus, rather than a cumulative presentation of the same stimulus repeated until person identification was successful. Based on these results, we might expect children with ASD to take longer to recognize human forms defined by PLDs. In agreement with this, Annaz et al. (submitted for publication), also using 1-s presentation times, have shown that, at a CA of 12 years, TD children are better than ASD children in discriminating intact from scrambled displays. Moreover, there is a flat developmental trajectory from 5 to 12 years for the ASD group and near identical performance of the TD and ASD groups at 5 years of age.

Hubert et al. (2007) looked at the age question by presenting the stimuli used by Moore et al. (1997) to a group of adults with HFA/AS and IQ-matched controls. There were four sets of PLD sequences, each defining a different motion category: 10 were "actions" (e.g. hopping, running), 5 were "subjective states" (e.g. bored, itchy), 5 were "emotional states" (e.g. surprised, sad) and 5 were object motions (e.g. ball rotating, ironing board opening and closing). Significant performance differences between groups were only found for the emotional states, which Hubert et al. (2007) argued was consistent with Moore et al.'s (1997) results. However, it should also be noted that whilst none of the other conditions reached significance there were larger variances on the performance of the ASD group in all conditions, and mean performance was only *identical* for the object motion condition. This suggests that the ASD group may have struggled with the task whenever it involved a human actor. Indeed, the authors reported a main effect of group following on from their ANOVA, but this is contaminated by a group-by-condition interaction. Another important point about the stimuli of Hubert et al. (2007), and therefore, by extension, Moore et al. (1997) is that there was no formal attempt to match the speed or complexity of the local motion content of the stimuli. In other words, the ASD group may have found the object motion easiest and the human motion harder simply because the motion signal was more complex, with more non-rigid relationships between point-light stimulus components. A final note of concern is the subjectivity of the response assessment, given that participants were asked to describe the motion, which was then judged by the experimenter as being an appropriate or inappropriate description. Whilst the anecdotal evidence of the rather mechanical descriptions of emotional actions by the ASD group are persuasive, it is less clear how well the descriptions of actions in the other conditions matched. Similar conclusions were reached in a follow-up paper by the same group (Parron et al., 2008).

Recent fMRI studies by Herrington et al. (2007) and Freitag et al. (2008) provide converging evidence on the neural processing of biological motion in ASD populations. The participants in Herrington et al. (2007) were a group of 10 adult males diagnosed with AS and 10 age-, sex- and IQ-matched controls. The participants in Freitag et al. (2008) were a carefully diagnosed group of 15 adolescents with HFA and controls matched for age, sex and IQ. The task used by Herrington et al. (2007) was to identify a 1s display as either intact or scrambled, and the synthetic Cutting algorithm (Cutting, 1978) was used to generate the 13-point intact displays with scrambling done by vertical displacement of a point's original location by a random distance. The task used by Freitag et al. (2008) was to identify a 1.5 s display as either intact or scrambled, and motion capture data of 80 walkers were used to generate the 15-point intact displays with scrambling done by permuting the location of the points, and changing the speed of each point to be equal to the average speed of that point. Herrington et al. (2007) reported that activity for the intact walker versus baseline was greater in the control than the ASD population in several regions including the right middle temporal gyrus. A similar trend in the middle temporal gyrus was reported by Freitag et al. (2008) in the contrast of all motions versus baseline. However, they also reported regions with greater activation by the ASD group, including the postcentral gyri, left hippocampus and middle frontal gyrus.

The study by Freitag et al. (2008) went on to show that the contrast of brain activity of intact versus scrambled revealed substantially different patterns of activation for the two populations. For the control group, when intact biological and scrambled motion were compared, activations were found bilaterally in parietal, temporal and frontal lobes as well as basal ganglia and insula. The activation network included the right Superior Temporal Sulcus (STS), which is known to be a central structure in biological motion processing (see Puce & Perrett, 2003). In contrast, the ASD group showed less activated clusters overall. What activations there were were primarily in the left hemisphere in parieto-temporal (limbic) and frontal areas as well as basal ganglia. In the right hemisphere, activations specific for biological motion were found only in the limbic system and Thalamus. Freitag et al. (2008) argue strongly that the processing of biological motion stimuli by people with ASD is very different from that of typical controls. Whilst the ASD group was capable of discriminating the biological motion from scrambled motion they seemed to be doing it using a different network of brain regions.

To summarise, the data on biological motion are consistent with a low-level difficulty with motion processing feeding through and complicating the interpretation of biological motion stimuli, especially when they present complex motions like human point-like walkers. However, the ease with which typical observers can attribute emotions and feelings to these curiously sparse stimuli is not present in ASD populations, and it even seems as though the brain circuits used for processing these stimuli are different, beyond low-level motion areas.

Among many areas of potential further development in biological motion perception in ASD one concerns the threshold for recognition. A possible interpretation of the results of Herrington et al. (2007) and Freitag et al. (2008) is that the stimuli were less salient for the ASD group: in other words the position on the recognition psychometric function was different for ASD and control groups. If, rather than simply presenting the same stimulus for the same duration to both groups the stimulus was somehow equated for

performance, would the neural activations still be different or would they now be much more comparable? The second issue is the extent to which the hierarchy of difficulty in structure-from-motion tasks demonstrated by the results of Moore et al. (1997), Hubert et al. (2007) and Parron et al. (2008) are due to the increased complexity of point-light human stimuli which depict such abstract notions as internal states and emotions or whether there is some sort of top-down enhancement mechanism for these types of stimuli which is lacking or deficient in ASD populations.

6.8. Animacy

A seminal study by Heider and Simmel (1944) demonstrated that typical individuals readily endow simple animated shapes with thoughts, feelings, and intentions. The perception of animacy in ASD has received a considerable amount of attention. Whilst this is arguably outside the domain of vision science *per se*, it has raised some relevant questions about the interpretation of visual motion information which will be covered briefly. First of all, as with biological motion, there is a consensus that all age groups with ASD appear to have difficulty interpreting animations to which typical observers attribute social content. However, this difficulty is a subtle one and seems to be confined to paradigms in which an extended verbal response is required. The precise deficit seems to be in the *appropriateness* of the language used to describe these animations. In particular, although the descriptions can be semantically elaborate, they tend not to include the same socially based vocabulary as typical descriptions (Abell, Happé, & Frith, 2000; Bowler & Thommen, 2000; Campbell et al., 2006; Castelli, Frith, Happé, & Frith, 2002; Klin, 2000; Salter, Seigal, Claxton, Lawrence, & Skuse, 2008). At the same time, participants with ASD do seem able to distinguish propelled from self-propelled motion and animacy from non-animacy, although possibly with developmental delay or a training requirement (Bowler & Thommen, 2000; Johnson & Rakison, 2006; Rutherford, Pennington, & Rogers, 2006). Furthermore, relatively elaborate animations which have explanations in physical, rather than social, terms, seem to be interpreted with relative ease (Klin & Jones, 2006). Castelli et al. (2002) have postulated that problems associated with interpreting these animated displays, and with social understanding in general, in ASD, are a result of the neural activity evoked in extra-striate visual cortex failing to transmit to the "social brain" regions of the STS, Temporal lobe and pre-frontal cortex.

However, a confound appears to run throughout these experiments. The key question is what makes an animation "social"? We know that the distinction is partly based on whether or not the agents appear to be self-propelled (Bowler & Thommen, 2000), but there are also aspects of the motion trajectory and the relationships between the trajectories of the agents that are crucial to the interpretation. None of the studies so far has formally matched the motion content of their animacy displays when comparing "social" versus "non-social" animation. Castelli et al. (2002) report no differences in MT/V5 activation in their PET study, but this is not really adequate. Klin and Jones' (2006) space animation appears to be complex, but the physics of this motion sequence would probably be familiar to boys who are experienced with contemporary video games. Rutherford et al.'s (2006) study points to the importance of training and motivation as well. Could it be that the problem with so-called "social" animations is that the complexity of the trajectories and inter-relationships between moving agents is higher and therefore more difficult to interpret? So the difficulty is not with the "socialness" *per se* but with the subtlety of the information structure in the displays which, somehow, typical observers pick up automatically during development but which observers with ASD need focused training with.

6.9. Visuomotor control

Gowen, Stanley, and Miall (2008) compared imitation behaviour in adults with ASD to CA- and IQ-matched controls on a task that involved making simple movements back and forth in the vertical or horizontal directions while viewing congruent (same movement direction) or incongruent (orthogonal movement direction) displays. These displays took the visual form of either an actual other person making the movements, or the motion of a single dot that was animated by a real movement recording, or a single dot that moved at constant velocity that instantaneously changed directions at the extremes. Results showed that the performance of both groups showed a significant interference effect for all conditions, however the ASD group did show the effect strongest when viewing the movements of an actual person. These results demonstrate that, for both groups, observing others' movements will interfere with one's own movement production but suggest that overall, the visuomotor integration processes are not identical between groups.

7. Perception of faces and objects

One of the most-studied, and heavily reviewed, areas of visual processing in ASD is face processing (for recent reviews see Behrmann, Thomas, & Humphreys, 2006b; Dawson, Webb, & McPartland, 2005a; Golarai, Grill-Spector, & Reiss, 2006; Jemel, Mottron, & Dawson, 2006; Sasson, 2006). This is unsurprising, given the usual characterization of ASD as a social deficit, and faces being arguably *the* most "social" of visual stimuli. Here we deal with object processing as well, because an object-based task is often used as a control in studies of face processing.

7.1. Early studies

In the first detailed study of face processing in ASD, participants were tested on their ability to recognize isolated features of known peers from grayscale photographs of their faces (Langdell, 1978). Two ASD groups were used: a "younger" group, aged about 10 years and an "older" group aged about 14 years. Each ASD group was matched with three different control groups: two typical control groups matched on either MA (and therefore much younger, being about 6 and 8 years old, respectively) or CA; a third control group was learning disabled and was matched on both CA and IQ. Note that the mean IQ of these ASD groups was quite low, at 60 and 63, respectively, making them both below the cut-off for learning disability themselves. It was found that both ASD groups were better able to use the lower part of the face for identification purposes than controls. The older ASD group was also less affected by inversion than the other experimental groups. Langdell's (1978) discussion of the results focused on two issues. First, did his data suggest that children with ASD tend to regard the face simply as a complex object, without the social relevance invested in it by typical children (in Langdell's words as a "pure pattern" rather than a "social pattern")? Second, was there evidence for what he termed "asocial looking" (i.e. fixating on the "wrong" part of the face), and did this relate to the well-known phenomenon of gaze avoidance in ASD (Hutt & Ounstead, 1966)? These ideas continue to reverberate through the literature on face processing in ASD to the present day.

The next major development in the field was a landmark series of studies by Peter Hobson and his co-workers at the Institute of Psychiatry in London (Hobson, 1986a, 1986b, 1987; Hobson, Ouston, & Lee, 1988a, 1988b, 1989; Weeks & Hobson, 1987) on the theme of emotion recognition. Weeks and Hobson (1987) asked children with ASD and VMA-matched learning-disabled controls

to sort photographs either according to facial expression (happy or not) or type of hat (floppy or woollen). Most children with ASD sorted by type of hat in preference to facial expression (see also Jennings, 1973). Hobson et al. (1988a) found that children with ASD had difficulty in matching basic facial expressions (happy, unhappy, angry and scared) across different individuals when presented with photographs of faces, or parts of faces, taken from the classic set of Ekman and Friesen (1975). The childrens' performance in identity and emotion sorting was also less affected than controls by changes in face orientation, consistent with Langdell (1978). Hence, by the late 1980s, the consensus was that children with ASD tend to regard faces as less "special" objects than either typical, or learning-disabled children (with equivalent verbal abilities), and that there were particular problems with the recognition of emotional facial expressions. There were no similar difficulties with non-social stimuli like visual scenes or objects (Hobson et al., 1989). How has this consensus been altered by more recent studies?

7.2. Is Face recognition impaired in ASD?

Face recognition is one of the most basic of face-processing skills. Whilst there is a considerable body of evidence that individuals with ASD demonstrate impairments in facial identity-based tasks (Boucher & Lewis, 1992; Boucher, Lewis, & Collis, 1998; Critchley et al., 2000; Curby, Schyns, Gosselin, & Gauthier, 2003; Davies, Bishop, Manstead, & Tantam, 1994; Dawson et al., 2002a; De Gelder, Vroomen, & van der Heide, 1991; Gepner, de Gelder, & de Schonen, 1996a; Hauck, Fein, Maltby, Waterhouse, & Feinstein, 1998; Jambaque, Mottron, Ponsot, & Chiron, 1998; Klin et al., 1999; Langdell, 1978; Teunisse & de Gelder, 1994; Ashwin, Wheelwright, & Baron-Cohen, 2005; Dalton et al., 2005; Riby, Doherty-Sneddon, & Bruce, 2009), a significant number of studies have disputed this finding, especially when the faces are familiar (Adolphs, Sears, & Piven, 2001; Barton et al., 2004; Braverman, Fein, Lucci, & Waterhouse, 1989; Celani, Battachi, & Arcidiacono, 1999; Chawarska & Volkmar, 2007; Davies et al., 1994; Deruelle, Rondan, Gepner, & Tardif, 2004; Gepner et al., 1996a; Ozonoff, Pennington, & Rogers, 1990; Volkmar, Sparrow, Rende, & Cohen, 1989; Wilson, Pascalis, & Blades, 2007). The evidence is, therefore, mixed.

7.3. Are there particular problems with emotional facial expressions in ASD?

Difficulties in the interpretation of facial expressions are often associated with ASD, but the scientific evidence for this difficulty is again remarkably mixed (for a review, see Jemel et al., 2006). Studies which report difficulties with various aspects of facial expression processing by individuals with ASD are Hobson (1986a, 1986b, 1987), Weeks and Hobson (1987), Gioia and Brosgole (1988), Hobson et al. (1988a, 1989), Braverman et al. (1989), McDonald et al. (1989), Tantam, Monagham, Nicholson, and Stirling (1989), Ozonoff et al. (1990), de Gelder et al. (1991), Capps, Yirmiya, and Sigman (1992), Sigman, Kasari, Kwon, and Yirmiya (1992), Baron-Cohen, Spitz, and Cross (1993), Davies et al. (1994), Gepner et al. (1996a), Loveland et al. (1997), Celani et al. (1999), Critchley et al. (2000), Grossman, Klin, Carter, and Volkmar (2000), Pelphrey et al. (2002), Ogai et al. (2003), Deruelle et al. (2004), Dalton et al. (2005), Hefter, Manoach, and Barton (2005), Ashwin, Chapman, Colle, and Baron-Cohen (2006a), Ashwin, Wheelwright, and Baron-Cohen (2006b), Boraston, Blakemore, Chilvers, and Skuse (2007), Humphreys, Minshew, Leonard, and Behrmann (2007), Mazefsky and Oswald (2007), Gross (2008), Wright et al. (2008). Some studies suggest specific deficits with particular emotional expressions such as fear (De Jong, van Engelund, & Kemner, 2008; Pelphrey et al., 2002), sadness (Boraston et al., 2007), or "negative" expressions (Ashwin et al., 2006a; Humphreys et al., 2007). Adolphs et al. (2001) showed that their small sample of adults with ASD did not differ significantly from controls in their ability to discriminate facial expressions, and were only mildly impaired in the recognition of basic emotions from faces. However, they did tend to mislabel the approachability and trustworthiness of faces, suggesting a parallel with amygdalar damage (Adolphs et al., 2001; Ashwin et al., 2006a).

A number of other studies have failed to find any dysfunction for high-functioning groups when the emotions are "basic", such as happiness, sadness, anger and disgust (Baron-Cohen, Wheelwright, & Jolliffe, 1997; Baron-Cohen et al., 1993; Davies et al., 1994; Volkmar et al., 1989b; Adolphs et al., 2001; Gepner, Deruelle, & Grynfeltt, 2001; Grossman et al., 2000; Hubl et al., 2003; Loveland et al., 1997; Piggot et al., 2004; Ashwin et al., 2005; Ashwin et al., 2006b; Castelli, 2005). Kätsyri, Saalasti, Tiipana, von Wendt, and Sams (2008) found equivalent performance in a basic facial expression rating task with their adults with AS and controls except for when they used an extreme low-pass filter on the stimulus (cut-off less than 1.8 cycles per face width).

There is more consistency in the literature that the performance of ASD populations on "complex" facial expression recognition is relatively poor (Adolphs et al., 2001; Baron-Cohen et al., 2001a, 1997; Boraston, Corden, Miles, Skuse, & Blakemore, 2008; Kleinman, Marciano, & Ault, 2001). The "Reading the mind in the Eyes" task illustrated in Fig. 4 is a case in point. Even individuals with HFA and AS struggle with this task, despite being able to define the emotional terms involved (Baron-Cohen et al., 2001a, 1997b). A potential concern with this task is Grossman et al.'s (2000) observation that people with ASD find it harder to choose an emotional match from a verbal description than from a pictorial description. However, the "eyes" test has been validated in a number of countries and adapted to a number of different age groups (see http://www.autismresearchcentre.com/arc), so it does provide a useful tool with which to check the facial expression processing capabilities of people at the high functioning end of the autism spectrum. Those with lower VIQs will obviously have difficulty with the semantic complexity of the verbal descriptions, although there are tests adapted specifically for use with children where the language demands are less severe. Boraston et al. (2008) found that their ASD participants were confused by fake and genuine smiles (where the eye region was giving either conflicting or consistent information) and were fixating less on the eye region of the target faces. They also found that difficulty with this task was correlated

Fig. 4. Slide from the "Reading the Mind in the Eyes" test (Baron-Cohen et al., 2001a, 2001b). Reproduced from the actual test with permission of the Autism Research Centre, University of Cambridge.

with the social interaction score from the individual's ADOS assessment (Boraston et al., 2008).

One recent study has questioned that individuals with ASD have problems interpreting facial expressions. Back, Ropar, and Mitchell (2007) used video clips of complex facial expressions and presented them to a group of eighteen 10–14-year-old children with ASD, and found that they performed above chance at naming the mental state from a choice of four descriptions, but were not as proficient as controls. However, when the eye region of the faces was digitally "frozen", the ASD group were affected, so they were clearly using information from the eye region. When the eyes were isolated and presented alone, the ASD group were as successful as controls and also when they were presented in the context of the whole face. Whilst Back et al. (2007) clearly demonstrate that individuals with ASD are capable of interpreting complex facial expressions under some circumstances one issue of comparison is that the task was less challenging because the same female actor was used throughout, whereas with Baron-Cohen et al.'s (2001a) "eyes" task there are a number of different faces.

7.4. Discrimination of other facial attributes

A number of studies suggest poorer performance of ASD groups in gender discrimination tasks based on faces (Baron-Cohen et al., 1999; Behrmann et al., 2006a; Deruelle et al., 2004; Hobson, 1987; Hobson et al., 1988a). Notably, gender discrimination is also thought to depend largely on information located in the eye region (Gosselin & Schyns, 2001). Hobson (1987) and Gross (2002, 2005, 2008) looked at age discrimination, the latter in human and non-human faces, and found a deficit in their population of children with ASD relative to controls. The data on lip-reading are more equivocal, with two in favour of a relative deficit (Deruelle et al., 2004; Gepner et al., 1996a), and two against (de Gelder et al., 1991; Gepner et al., 2001).

7.5. Is there a face processing impairment in ASD?

Jemel et al. (2006) argue, along with Behrmann et al. (2006b) and Mottron, Dawson, Soulières, Hubert, and Burack (2006), for an amodal and non-domain-specific difference in perceptual processing characterized by "locally oriented" perception of faces without a specific deficit in perception of global features, face identity or emotion. Certainly the behavioural data reviewed above does not provide unquivocal back-up for there being a specific face-processing deficit in all individuals diagnosed with ASD, although it could be argued that this ability is less robust than in typical populations. Could this fragility have an underlying perceptual basis?

7.6. Is object identification and recognition spared in ASD?

A general problem with object identification and recognition would potentially contribute to poorer performance in face processing tasks, but the object processing abilities of individuals with ASD are generally reported to be equivalent to those of control populations (Boucher & Lewis, 1992; Braverman et al., 1989; Davies et al., 1994; Hobson, 1986a, 1986; de Gelder et al., 1991; Tantam et al., 1989 (low IQ group), Celani et al., 1999; Dawson et al., 2002a; Gepner et al., 1996a, 1996b).

There are only a couple of studies which suggest that ASD populations may have problems with some types of object judgement (Berhmann et al., 2006a; Davies et al., 1994). Behrmann et al. (2006a) presented their group of adults with HFA with two object processing tasks. In the first they were required to determine whether two pictures of naturally occurring objects were the same or different. In the second, the "Greeble" class of alien-like objects was used (Gauthier & Tarr, 1997). In both cases the ASD group demonstrated more difficulty with fine object discriminations at the exemplar/individual level, than controls. Behrmann et al. (2006a) suggested that visual discrimination difficulties are not confined to social objects like faces, but may reflect more general anomalies in visual processing.

7.7. Are faces "special" for individuals with ASD? The cases of inversion and Thatcherisation

It is generally thought that faces are a "special" class of objects for the typical visual system, with their own dedicated neural processing system (see below). One of the perceptual indicators of this specialness is that faces are harder to recognize upon inversion than other objects. Individuals with ASD appear able to distinguish faces from non-faces (Ashwin, Baron-Cohen, Wheelwright, O'Riordan, & Bullmore, 2007; Volkmar et al., 1989), but Langdell's (1978) original suggestion that children with ASD are less affected by facial inversion has received only mixed support. Tantam et al. (1989) and Van der Geest, Kemner, Verbaten, and van Engelund (2002) argued for unusual performance of ASD populations with inverted faces. Joseph and Tanaka (2003) presented evidence that children with ASD were more sensitive to image manipulations in the mouth region than in the eye region of faces, unlike controls, and that only this sensitivity showed an inversion effect: a partial replication of Langdell (1978). But most recent studies have shown significant face inversion effects in ASD populations (Barton, Hefter, Cherkasova, & Manoach, 2007; Gross, 2008; Lahaie et al., 2006; Teunisse & de Gelder, 2003) shifting the balance in favour of an intact face inversion effect in ASD, at least in high-functioning groups. There is a possibility that this effect (or, effect absence!) might be more pronounced in ASD populations with lower IQ (Teunisse & De Gelder, 2003) or, at least, those with poorer face discrimination abilities (Barton, Hefter, Cherkasova, & Manoach, 2007), but there seem to be confounding factors of overall task performance, some of which may not be related to visual processing.

Another related stimulus manipulation is the selective inversion of facial features known as the "Thatcher Illusion" (Thompson, 1980). Rouse, Donnelly, Hadwin, and Brown (2004) and Riby et al. (2009) have shown that this illusion is intact in children with ASD. However, Riby et al. (2009) also manipulated eye and mouth regions separately and found that their group of 10–18-year-old children with autism were equally sensitive to "Thatcherisations" of the eyes and mouth and did not favour the eyes as the controls did.

7.8. Are faces processed as a collection of individual features in ASD, rather than as a coherent whole?

"Configural processing" has been defined as the perception of relations between features of a stimulus, such as a face, as contrasted with "componential" or "featural" processing (Maurer, Le Grand, & Mondloch, 2002). Behrmann et al. (2006a) found that their group of adults with ASD found it harder to discriminate masked down (i.e. "internal features only") images of faces by identity and gender than matched controls. Barton et al. (2007) explicitly compared featural and configural processing of faces by manipulating the eye separation or mouth position (configural) or eye and mouth colour (featural) properties of face images and asking their population of SDD[3] adults which of three faces simultaneously present on screen was the odd one out. Those who were able

[3] Barton et al. (2007), in common with other studies from the same laboratory (Barton et al., 2004; Hefter et al., 2005) use the clinical specification "Social Developmental Disorder", which is a superset of those with Autism Spectrum Disorder together with those with slightly different symptomology similar to PDD-NOS.

to do famous face recognition ("SDD-1") were only mildly impaired relative to controls in this task, but those unable to do famous face recognition ("SDD-2") were severely impaired. Notably, however, they were equally impaired on featural as configural changes, suggesting that there was no specific deficit in sensitivity to configural changes to faces. Note that, while this experiment was ingenious there is a small concern that the featural changes were not matched for discriminability with the featural changes. Teunisse and de Gelder (2003) looked at the facial composite effect, where the photograph of a face is split in two along a horizontal axis and combined with another. When typical participants are asked to spot whether the top half of this face is the same as that of a simultaneously presented target face they are poorer when the composites are aligned rather than misaligned (Young, Hellawell, & Hay, 1987). The ASD group tested by Teunisse and de Gelder (2003) did not show this effect, although the controls did. However, despite being statistically significant, the differences found were quite small. Joseph and Tanaka (2003) reported a variation on the "whole-face advantage" normally found when trying to spot which features of a previously seen face have been altered. Whereas typical children were more sensitive to eye substitutions, the ASD group were more sensitive to mouth substitutions, echoing the results of Langdell (1978). However, performance was generally quite poor in this task for both ASD children and controls, only reaching a maximum of 76% correct (controls on eye substitutions). Riby et al. (2009) have also reported that their ASD group were less sensitive to eye than mouth manipulations in a similar experiment, but Lopez, Donnelly, Hadwin, and Leekam (2004) found that they could generate a whole-face advantage in their adolescents with ASD by adding a cue to the appropriate feature.

All in all, there seems to be a large amount of variability in the data on configural processing of faces in ASD. Barton et al.'s (2007) data suggest that this variability may be due to a bimodal split in the ASD population between those that are quite capable in face identity processing tasks and those that are not.

7.9. Are face processing difficulties in ASD confined to the eye region?

It is commonly asserted that people with ASD have difficulty attending to the eye-region of faces, unlike typical controls (Boraston et al., 2008; Dalton et al., 2005; Gross, 2004; Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Pelphrey et al., 2002; Rutherford, Clements, & Sekuler, 2007a) and/or attend selectively to the mouth region (Joseph & Tanaka, 2003; Langdell, 1978). Rutherford et al. (2007a) tested this prediction explicitly by measuring the discriminability of small spatial displacements in the eyes and mouths of digitally altered images of unfamiliar faces in 16 young adults with ASD and 19 matched controls. The test images only included the internal facial features (i.e. no hair or contour features). There were significant differences between ASD and control groups for eye-region displacements but not for mouth-region displacements. Rutherford et al. (2007a) were able to divide their ASD sample into two sub-groups: those who performed in a typical fashion and those who were severely impaired. The key difference between the sub-groups was in VIQ which was average (100.5) in the typically-performing sub-group, but below average (85.3) in the group that experienced more difficulty with the eye-region task. Control experiments provided no evidence for selective attention to the mouth in the ASD group (cf. Joseph & Tanaka, 2003; Riby et al., 2009).

A potential criticism of Rutherford et al. (2007a) is that there was no explicit measurement of eye fixations. However, Pelphrey et al. (2002) presented five adult males with autism with Ekman faces of the six basic emotions (happiness, sadness, anger, fear, surprise, and disgust) and monitored their eye movements with a high-precision eye tracker. Participants were asked to view the faces normally and also to identify the emotion. Stimulus duration was 2 s and subtended a relatively large visual angle (10.7 deg horizontally and 14.2 deg vertically), presumably to allow plenty of resolution in the eye movement measurements. They found clear qualitative differences between the scan paths of their ASD and control groups (see Fig. 5). In particular, the ASD group did not fixate as much on the eye region as the controls. They were also significantly worse at identifying the emotions, particularly the fearful faces.

This result is particularly significant. Smith, Cottrell, Gosselin, and Schyns (2005) have shown that the eye region is important for the detection of fear in emotional faces, and the observation provides a clear reason for the wide range of results found in face processing and ASD when eye movements are unmeasured on uncontrolled. As mentioned above, the idea that individuals with ASD simply look in the wrong place when presented with face stimuli (and no further instructions) was originally suggested in Langdell's (1978) study on face processing in ASD. If this is true in everyday life then the experience necessary for becoming "expert" face processors will not develop, especially if no explicit instructions are given or if the aversion to viewing the eyes of others is particularly strong (Hutt & Ounsted, 1966). Clearly this experiential effect is likely to vary between individuals and may give rise to the sorts of ability differences in ASD populations reported by Barton et al. (2004).

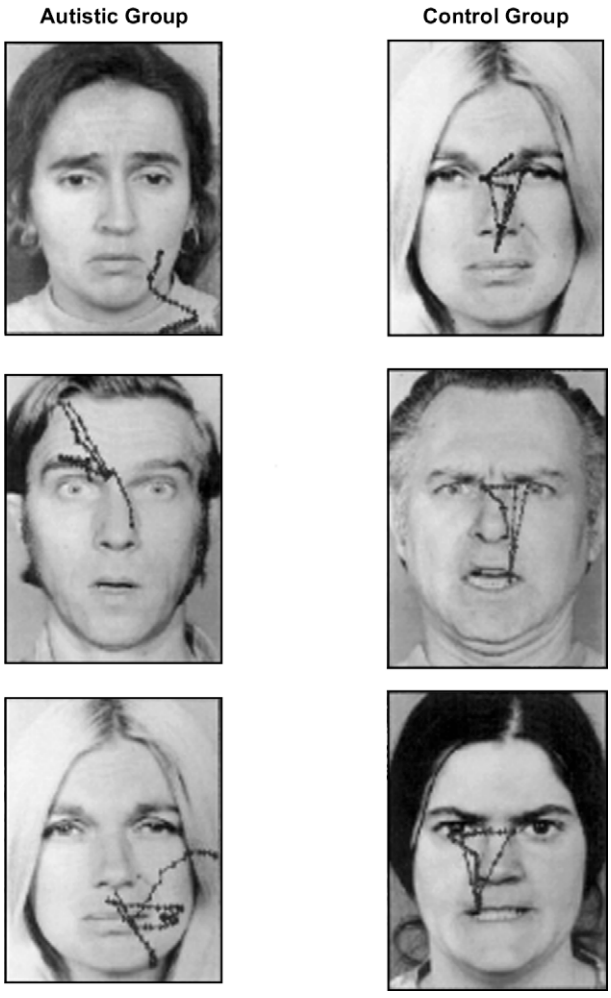

Fig. 5. Eye tracking data from Pelphrey et al. (2002). Reproduced with permission of Springer Science and Business Media.

Spezio, Adolphs, Hurley, and Piven (2007) looked explicitly at eye gaze to fearful and happy faces, but used the "Bubbles" method (Gosselin & Schyns, 2001) to reveal different amounts of information from trial to trial, and thereby test the information being used to perform the task whilst also tracking eye movements. The Bubbles technique works by using adaptive control to maintain behavioural performance at a constant level, giving the added advantage that all participants were performing the task equally well. Spezio et al. (2007) found that their eight adults with HFA showed less fixation specificity to the eyes and mouth, a greater tendency to saccade away from the eyes (even when useful information was present in the eye region) and abnormal directionality of saccades. Spezio et al.'s (2007) data reinforces the idea that face scanning is atypical in adults with ASD, which may have a profound influence on social functioning.

However, van der Geest et al. (2002) found no significant differences in scan paths or initial fixations to emotional faces with his sample of 17 children with ASD and Bar-Haim, Shulman, Lamy, and Reuveni (2006), using a dot-probe paradigm, found no differences between the relative attentional allocations made to the eyes and mouth by their group of 10-year-old boys with HFA and controls. A potential factor in the data of both van der Geest et al. (2002) and Bar-Haim et al. (2006) is the lack of fearful and, in the latter case, any emotional expressions among the face stimuli.

7.10. Gaze detection

If there are problems with using information from the eye region of faces in ASD, then it would be expected that eye-gaze detection might be particularly affected. Leekam, Baron-Cohen, Perrett, Milders, and Brown (1997) found that the ability of children with ASD to discriminate degrees of change in the orientation of the gaze of an adult from carefully prepared and calibrated photographs was well matched to their developmental age level, corresponding to a displacement of the iris as small as 3.5 arcmin, although their ability to spontaneously follow the gaze of an experimenter was well below that of controls. Some subsequent studies have confirmed this result (Kylläinen & Hietanen, 2004; Leekam, Hunnisett, & Moore, 1998; Warreyn, Roeyers, Oelbandt, & De Groote, 2005).

Campbell et al. (2006), however, did find a specific deficit in eye gaze sensitivity in their sample of 11-year-old children with ASD and carefully matched controls. Their task was likely more challenging than that of Leekam et al. (1997) in that the gaze displacements were smaller. There were also subtle differences in the instructions ("Is she looking at you?" rather than "Where is she looking?") which may have had an impact. Even so, Campbell et al.'s (2006) results do suggest that eye gaze acuity may be slightly worse in children with ASD. Webster and Potter (2008) have also suggested that careful measurements of eye gaze acuity (their smallest deflection was 5 deg from the midline) may find subtle differences in eye gaze processing, especially with younger children.

Swettenham, Condie, Campbell, Milne, and Coleman (2003) demonstrated that reflexive orienting is intact in older children with ASD (mean age 10 years). Both their control and their ASD groups were unable to resist following the cue given by the eyes of a face to a subsequently presented target, even though the cue was invalid 50% of the time. The effects were also apparent when the face was presented upside down. This suggests that not only can children with ASD perceive larger movements of the eyes, but they can also reflexively move their attention to the location cued by them. Note that in this study, the eyes were actually seen to move, rather than being presented as a static stimulus with eyes averted. Reflexive orienting to eye movements in ASD have also been investigated by Okada, Sato, Murai, Kubota, and Toichi (2003), Senju, Tojo, Dairoku, and Hasegawa (2004), Kylliäinen and Hietanen (2004), Vlamings, Stauder, van Son, and Mottron (2005), Kemner, Schuller, and van Engelund, 2006, Goldberg et al. (2008) and Rutherford and Krysko (2008). Although Swettenham et al. (2003) suggested that the success of their ASD group in using the eye cue was a developmental effect, Chawarska, Klin, and Volkmar (2003) found a similar effect in very young children (2 years) with autism diagnoses.

Senju, Yaguchi, Tojo, and Hasegawa (2003) looked at the ability of children with ASD to detect a face with direct eye gaze using the oddball paradigm. They found that the children with ASD failed to show a detection advantage when the gaze of the target face was direct, unlike the typical children, although the performance in oddball detection for averted gaze was the same. Senju et al. (2003) argued that children with ASD lack special mechanisms for detecting direct gaze and thus should not obtain the "stare-in-the-crowd" effect (von Grünau & Anston, 1995).

Senju, Hasegawa, and Tojo (2005) extended their previous result to a visual search paradigm, demonstrating that children with ASD show no search-time advantage for a face looking directly at the viewer, rather than a face with averted gaze, unlike control participants. However, in a clever additional experiment, Senju et al. (2005) used schematic pictures of pairs of eyes which maintained the symmetry cues normally present if a viewer is facing directly towards you. In this case, a direct gaze effect was observed in the ASD children, despite there being generally longer reaction times in the clinical group for all tasks.

Kylliäinen and Hietanen (2006) measured skin conductance responses (SCR) – an indicator of stress levels – in children with ASD and controls while viewing an adult face with either straight-ahead or averted gaze. The SCR responses from the children with ASD were significantly stronger for straight-ahead gaze stimuli than for averted gaze stimuli, unlike the controls. This result has obvious implications for the notion that eye contact can be particularly aversive for people with ASD. Wallace, Coleman, Pascalis, and Bailey (2006) also showed that eye-gaze detection is impaired in adults with ASD, particularly in the context of the whole face.

Kylliäinen, Brauetigem, Hietanen, Swithenby, and Bailey (2006) examined MEG responses to faces with different eye-gaze states in 7–12-year-old children. Only subtle differences were found between these responses and those of normative controls. However, Grice et al. (2005) found that their sample of young children (mean age 61 months) and age- and sex-matched TD controls showed significantly different High Density ERP responses to faces with direct and averted gaze. Grice et al. (2005) suggested that their results reflected a developmental delay in the processing of eye gaze in ASD.

Elsabbagh et al. (2009) studied EEG responses in infant siblings of children diagnosed with ASD. Whilst these infant siblings are too young themselves to receive a diagnosis of ASD, they are at high risk of developing it, although this will only be known in the future. Elsabbagh et al. (2009) found that the early ERP components did not differ between the sibling group and controls, but the later P400 potential (thought to be a pre-cursor of the adult N170) showed a prolonged latency in response to direct gaze. Measurements of neural oscillations in the gamma band also showed differences between the sib-ASD and control groups in response to direct gaze. This gamma activity was later and less persistent over the right temporal region in the sib-ASD group. Note that this result is unlikely to have been affected by the eye-movement artifacts which possibly contaminate measurements of gamma band activity in adults (Yuval-Greenberg et al., 2008).

To summarise this section on eye gaze processing, it is clear that children and adults with ASD have some sensitivity to eye gaze differences in that reflexive visual orienting to eye gaze cues seems to be largely intact, and acuity for modulation in eye gaze is indistinguishable from typicals down to about 10 deg away from midline.

Two studies have suggested that younger children with ASD may have problems with detecting very fine modulations of eye gaze (Campbell et al., 2006; Webster & Potter, 2008) and this would seem to be an interesting area for further investigation with rigorous psychophysical techniques. Another difference noted between ASD and control groups, which may be related, is that direct gaze (independent of the symmetry cues provided by differences between straight-ahead direct and averted gaze) is more problematic for those on the autism spectrum to process. This is particularly apparent in the data of Senju et al. (2003, 2005) and the electrophysiological and magnetoencephalographic studies of eye gaze processing in ASD and related populations (De Jong et al., 2008; Elsabbagh et al., 2009).

7.11. Neural processing of faces in ASD

7.11.1. Electrophysiological studies

Studies of face and object processing in ASD using EEG have been reviewed by Dawson et al. (2005). The advantage of this technique for studying ASD is that it can be used on children who are too young to take part in psychophysical experiments. Dawson et al. (2002a) looked at children aged 3–4 years of age with and without ASD, presenting them with faces of their mothers, matched unfamiliar faces, photographs of their favourite toy and photographs of a similar toy whilst monitoring their neural event-related potentials (ERPs) with high-density EEG. The typical matched controls showed ERP amplitude differences in potentials P400 and Nc to both familiar vs. unfamiliar toys and faces, but the ASD group only showed ERP amplitude modulation in these components to the toys. These data are clearly consistent with a face recognition impairment in ASD being present early in life. In a follow-up study, Dawson, Webb, Carver, Panagiotides, and McPartland (2004) found weaker differential responses to fear and neutral faces in ASD children compared to controls. In adolescents and adults with ASD, McPartland, Dawson, Webb, Panagiotides, and Carver (2004) found longer latency N170 potential responses to faces versus non-faces, but equivalent latencies for objects. O'Connor, Hamm, and Kirk (2005) found delayed P1 and N170 latencies and smaller N170 amplitudes in adults with AS in comparison to controls to a range of facial expressions. Curiously, no such differences were observed between these potentials in a group of children with AS and their controls. O'Connor, Hamm, and Kirk (2007) also found N170 delays in AS adults to faces and face parts, but not to objects. Webb, Dawson, Bernier, and Panagiotides (2006) found that 3–4-year-old children with ASD showed slower ERPs to faces and larger ERP amplitudes to objects than control children. All of these studies have therefore suggested that the neural basis of face processing is anomalous in ASD.

Boeschoten, Kenemans, van Engelund, and Kemner (2007) were interested in the cause of these face processing anomalies. They measured ERP responses to faces, objects and stimuli for which children with Pervasive Developmental Disorder[4] (PDD) were "experts" (e.g. cars, cartoon characters). These images were either low- or high-pass filtered and displayed upright or inverted in order to get differential ERP responses. Dipole source localization techniques were also used to give information about the location of the brain activity. Conventional ERP analysis revealed no differences between the responses of these 10–11-year-old children and CA- and IQ-matched controls. However, the source analysis revealed that the controls appeared to be using specialist face processing mechanisms when the faces were dominated by low spatial-frequency (LSF) content, but the children with PDD were not. The difference came in the relative amount of frontal and occipital activation to low-pass filtered faces. In the controls frontal sources were activated the most by LSF faces and occipital sources by HSF faces, but in the PDD group occipital sources were always the most active. Boeschoten et al. (2007) argued that their data echoed data from the fMRI literature showing differential frontal activations to faces in ASD (Dalton et al., 2005; Pierce, Haist, Sedaghat, & Courchesne, 2004) and were evidence against the "reduced expertise" explanation for face-processing deficits (Schultz, 2005) and in favour of a lower-level perceptual cause. Two potential problems with Boeschoten et al. (2007) however are that they do not report how well their low- and high-spatial-frequency stimuli were matched for contrast energy and there was no "expertise" comparison for the control group, so the specificity of the measured difference for faces is uncertain.

Grice et al. (2001) presented adults with ASD, Williams Syndrome and typical controls with upright or inverted colour faces of adult females whilst monitoring their neural responses with EEG. In the ASD group they found that the oscillatory potentials in the so-called gamma band (20–50 Hz), thought to be characteristic of visual coherence, were reduced in amplitude in the ASD group (see Tallon-Baudry, Bertrand, Delpuech, & Pernier, 1996). Recent EEG studies have however, suggested that this result may be unsound and demonstrated that gamma oscillations from EEG with the broad-band time–frequency signature indicated in Fig. 1 of Grice et al. (2001) are likely to be an artifact caused by spiking potentials from the eyes (see Yuval-Greenberg et al. (2008) and discussion above).

To date there is one published MEG study of neural responses in ASD populations to faces (Bailey, Braeutigam, Jousmäki, & Swithenby, 2005). This study implicates a processing network with subtly different timing and localization when adults with ASD view pictures of faces, possibly indicating unusual activity in the Fusiform Gyrus, consistent with many fMRI studies of face processing in ASD (see below).

7.11.2. Neuroimaging with magnetic resonance

As far as face processing in ASD is concerned, the two main foci of attention in neuroimaging studies have been the fusiform gyrus and the amygdala. It is well known that the fusiform gyrus contains a region that appears to be critical to face processing that has become known as the Fusiform Face Area (FFA) (Kanwisher, McDermott, & Chun, 1997). It is equally well known that this has been the focus of a lively debate about whether it really is an area specialized for face processing or simply for any class of objects requiring fine discrimination (Gauthier, Tarr, Anderson, Skudlarski, & Gore, 1999). Since the first studies in 2000, there have been a large number of replications of reduced or anomalous activation of Fusiform Gyrus in both adults and children with ASD during face processing tasks which might be consistent with a non-existent, inactive or at least substantially altered FFA (Critchley et al., 2000; Hall, Szechtman, & Nahmias, 2003; Pierce, Muller, Ambrose, Allen, & Courchesne, 2001; Schultz et al., 2000 (a PET study), Dapretto, Hariri, Sigman, & Bookheimer, 2004; Hubl et al., 2003; Piggot et al., 2004; Wang, Bailey et al., 2005 (MEG), Dalton et al., 2005; Grelotti et al., 2005). By 2005 there had been demonstrations of reduced FFA activations in ASD in 157 individuals (Schultz, 2005). The most popular explanation for this observation has been that, for some reason, the FFA does not develop face specialization in ASD, but is instead recruited by other objects with which the child with ASD is interested. The most striking support for this argument was the child with ASD who was obsessed with the Japanese cartoon series Digimon™ and whose FFA was apparently active to these distinctive cartoon characters rather than human faces (Grelotti et al., 2005).

However, three studies which used different presentation and stimulus monitoring techniques suggest that FFA hypo-activation in ASD might be an epiphenomenon. Pierce et al. (2004) used an

[4] This Dutch group consistently uses PDD groups rather than ASD.

event-related, rather than a block design, and utilized familiar faces to engage the attention of ASD participants more in the task. Pierce et al. (2004) found significant activations in what they argued was the FFA in their small sample of eight adults with ASD. Hadjikhani et al. (2004b) argued strongly that controlling attention, and therefore fixation, of the ASD participants was crucial to finding FFA activation. They showed that when a prominent fixation spot was placed in the centre of the display screen, the face stimuli were large (20 deg in diameter), participants were not asked to make a discrimination but passively monitor the display, and high-resolution (3T) scanning was used, significant bilateral activations of the Fusiform Gyrus were obtained which did not overlap with object processing regions. Dalton et al. (2005), who explicitly monitored eye movements during the scanning of their adolescents with ASD, found that activation of the fusiform gyrus in their face processing task correlated with eye movements to the appropriate parts of the face in the ASD participants, but did not observe the same correlations in the control participants. Thus, if hypo-activation of FFA is to be demonstrated convincingly in ASD, it must be found when fixation or eye movements are controlled.

The other brain region of particular interest for face processing in ASD has been the amygdala (e.g. Baron-Cohen et al., 2000; Schultz, 2005). As with the FFA, early neuroimaging evidence was in agreement that the amygdala was hypo-active during the viewing of (normally) emotional facial expressions (Baron-Cohen et al., 1999; Critchley et al., 2000; Pierce, Muller, Ambrose, Allen, & Courchesne, 2001). More-recent data has, however, been more circumspect. Wang, Dapretto, Hariri, Sigman, and Bookheimer (2004) found hypo-activation, but it was a small effect. Grelotti et al. (2005) found hypo-activation for emotional faces in their case study of ASD, but found significant activation for the Digimon™ characters that their participant was obsessed with. Welchew et al. (2005) reported reduced functional connectivity between the amygdala and other brain areas, but admitted that findings in this area can be highly task-dependent. Ashwin et al. (2007) reported reduced activation of amygdala together with higher activations in Superior Temporal Sulcus, and other brain regions, relative to controls. However, three studies, those of Ogai et al. (2003), Pierce et al. (2004) and Piggot et al. (2004), failed to find any significant differences in amygdala activation for the ASD samples compared to controls. Note that Piggot et al.'s (2004) participants were relatively young (age range 9–14 years) compared with other studies, which mainly focus on adults. One study, that of Dalton et al. (2005), actually found higher activations of the amygdala in their ASD sample. They argued that faces, when properly fixated by individuals with ASD (note that their ASD sample was also quite young, with a mean age of 15), actually cause *hyper*arousal of the amygdala, and that this effect may be a factor in the well known symptom of gaze aversion.

Whilst the weight of evidence, in terms of number of replications, currently favours the hypo-arousal camp, it seems that it is really too early to start building theories of the development of ASD which rely on this result. Dalton et al.'s (2005) study, together with Ogai et al. (2003), Pierce et al. (2004) and Piggot et al. (2004) suggest that it is possible to obtain functional activations of the amygdala in individuals with ASD, given appropriate control of the participant (with regard to fixation) and appropriate levels of stimulation (familiar or highly evocative faces). It could simply be that previous demonstrations of hypo-arousal have not really stimulated this region hard enough to get a response. Having said that, a threshold difference of this sort, if it is apparent under the conditions of everyday life, is bound to have functional consequences, depending on what the precise function of the amygdala is in typical populations (Diekhof, Falkai, & Gruber, 2008; Pessoa, 2008; Schaefer & Gray, 2007).

7.12. Is adaptation to faces anomalous in ASD?

Pellicano, Jeffery, Burr, and Rhodes (2007) investigated another face-specific phenomenon in ASD, the face-identity aftereffect, in which adaptation to a particular face biases perception towards the "opposite" identity (determined using face-averaging and morphing techniques, see Leopold, O'Toole, Vetter, and Blanz (2001) and Fig. 6). After a training session with the two unfamiliar target faces, participants were asked to group morphs of different strengths into "teams" corresponding to each target. This measured baseline performance. Presentation time was 400 ms. The adaptation trial was then introduced in the context of a scenario involving robbers of a jewellery store. The adaptation period was 5 s per trial. The adaptation face was the "anti-face" of one of the two target faces. Pellicano et al. (2007) found that their ASD group (children aged 8–13 years) could discriminate between the two target faces as well as matched controls in the baseline condition, but showed considerably less adaptation to the anti-faces with the amount of adaptation correlating with the child's symptomatology, as measured by the Social Communication Questionnaire (SCQ;

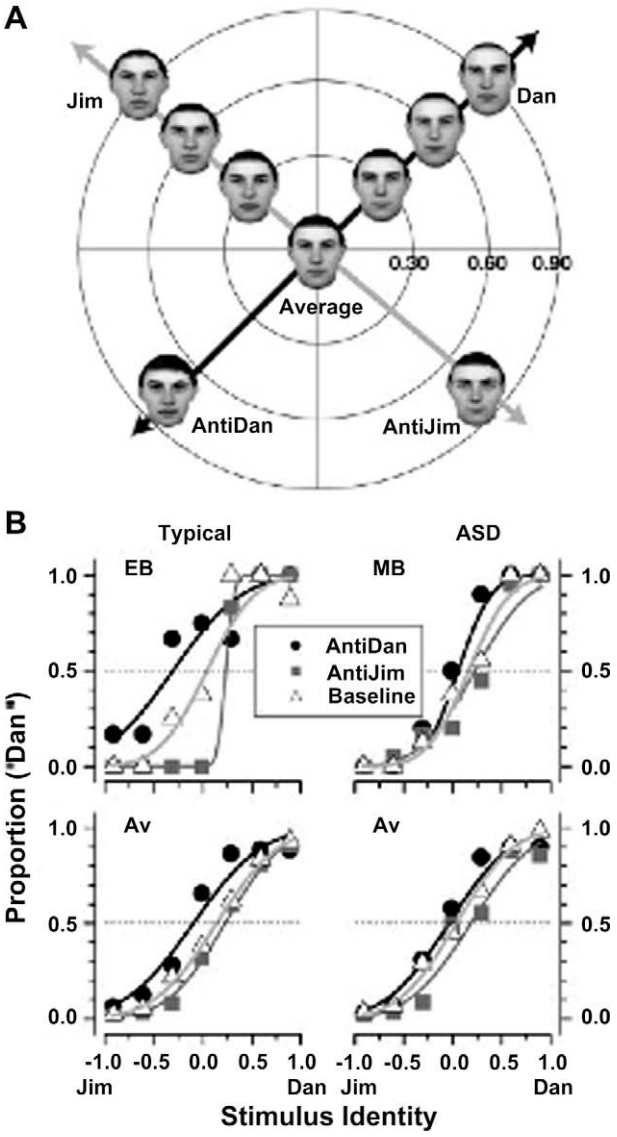

Fig. 6. Figure from Pellicano et al. (2007) showing the layout of the "face space" and representative data. Reproduced with permission of Elsevier Ltd.

Rutter et al., 2003). Note that there was no loss of precision in the face-identification judgements after adaptation, which argues against a more general worsening of performance due to the adaptation process.

Pellicano et al. (2007) used reliably diagnosed ASD groups and forced-choice psychophysics, although the observed effects were small and eye movements were not monitored directly during task performance. An obvious potential criticism of the Pellicano et al. (2007) result is that the ASD group simply did not fixate appropriately on the adaptation stimulus, but the experimenters used a relatively short adaptation period of 5 s, and did their best to encourage the children to fixate during this time.

Pellicano et al.'s (2007) findings suggest that visual adaptation mechanisms may be affected in ASD, and possibly not in a face-specific way. They argue that this difficulty may reflect a problem with generalization across stimuli, which has been highlighted in some theoretical approaches to ASD (Frith, 1989; Klinger & Dawson, 2001; Plaisted, 2001).

7.13. Face and object processing in ASD – summary

The literature on face processing in ASD presents quite a confusing picture, with very few clear results to hang potential theories on. There is evidence that individuals with ASD tend to process the eye region of faces less effectively than typical individuals (e.g. Spezio et al., 2007), and that this difficulty may arise from a tendency not to look at the eye region of faces unless specifically instructed to (Dalton et al., 2005; Pehplrey et al., 2002). This tendency could, in turn, arise from the well known aversion to direct gaze in ASD.

Rutherford et al. (2007a) highlight the importance of the VIQ scores of their ASD group in their ability to perform face eye-region spatial discriminations. This finding echoes previous work on facial expression tasks, where ASD groups were found not to be impaired relative to VMA-matched controls (e.g. Braverman et al., 1989; Ozonoff et al., 2000; discussed further in Jemel et al., 2006). If verbal comprehension ability is closely linked with facial interpretation judgements, then it is possible that previous failures or successes in finding face-processing deficits in ASD groups have been due to using different mixtures of verbal ability within the sample. If it proves to be true that verbal comprehension ability is a good predictor of face-processing deficits in ASD then it will be interesting to determine whether this may be a cause or a consequence. Barton et al.'s (2004, 2007) differential findings with groups differing in their famous face recognition abilities also suggests that there are a broad range of face processing abilities in ASD populations which must be divided further in order to provide meaningful results. Hidden correlations, possibly with the amount of exposure to magazines or television, may underlie these differential abilities. There is also the possibility that some more general visual processing deficit with object discrimination (Behrmann et al., 2006a), eye gaze acuity (Campbell et al., 2006; Webster & Potter, 2008) or adaptation processes (Pellicano et al., 2007) might be involved.

A final potential factor is suggested by Deruelle et al.'s (2004) result that participants with ASD made more errors in a face matching task when presented with low-pass-, rather than high-pass-filtered face stimuli. This tendency to use the higher, rather than the lower, spatial frequency information might cause particular problems when face stimuli are far away, although it would depend on whether or not the issue is with spatial frequency *per se*, or the relative spatial frequency content of the face. Could the stimuli in many demonstrations of poor face processing in ASD simply have been too small? There has been no explicit study of the role of viewing distance on face processing in ASD to find this out, and ideally this would need to be combined with spatial frequency filtering to address the normalization problem.

8. Visual search and attention

8.1. Embedded figures

It has been commented on for some time that both children and adults with ASD appear to be sensitive to minute changes in their environment which are often invisible to, or at least unnoticed by, their typical peers (Wing, 1976). Shah and Frith (1983) measured this ability directly using the "Embedded Figures Test" (EFT; Witkin, Oltman, Raskin, & Karp, 1971). This test, which forms a part of many IQ test batteries, requires the individual under test to find a simple figure, such as a triangle, in a complicated pattern that makes up a real image, such as a pram ("stroller" for US readers; see Fig. 7). Shah and Frith (1983) compared the performance in this task of 20 children with autism (8–18 years, mean ~ 13 years), to 20 children with learning disabilities, but matched in MA, and 20 typical children with a lower CA (about 9) to match the MAs of the two clinical groups. They found that not only was the performance of the children with autism significantly higher than that of the controls, but as good as, if not slightly better than, that expected of children matching their CA. They also mentioned that the location of the embedded figure by the children with autism was often immediate, without any obvious need for visual search (hence the title of their paper: "an islet of ability in autistic children" (Shah & Frith, 1983)). More recent studies by Ropar and Mitchell (2001), Pellicano et al. (2005), Jarrold, Gilchrist, and Bender (2005), De Jonge, Kemner, and van Engelund (2006) and Falter, Plaisted, and Davis (2008) have confirmed and extended the Shah and Frith (1983) result. Edgin and Pennington (2005) measured performance in the Children's version of the EFT in a group of children with ASD with ages ranging from 7 to 17. It was found that the performance advantage over controls (measured in terms of reaction time) for the ASD group was largest with the younger children, but gradually decreased with increasing age until the two groups matched at the top of the age band.

Jolliffe and Baron-Cohen (1997) performed a careful study on adults with ASD and matched controls, using the adult version of the EFT. They characterized the performance of their participants largely in terms of "completion" time, asking them to draw around the embedded object and including this drawing time in their performance measure. They also supported Shah and Frith (1983) in that their ASD group (both those with classic HFA and those with AS) was almost twice as fast, on average, as typical controls matched on IQ, although there were no significant differences in accuracy, all participants being quite close to ceiling in the task. Superior or equivalent performance in the EFT in adults with ASD has been confirmed by Bölte et al. (2007).

Jolliffe and Baron-Cohen (1997) followed up this behavioural paper with a fMRI study of the same cohort of participants (Ring et al., 1999). It was found that activations in control participants were more extensive and included pre-frontal cortical areas that were not recruited in the ASD group. The ASD group, however, demonstrated greater activation in occipito-temporal regions. Ring et al. (1999) suggested that the frontal activations may indicate the involvement of working memory systems in solving the task but that the ASD group only used visual areas. Manjaly et al. (2007) and Lee et al. (2007) have also reported unusual brain activations in ASD groups whilst they performed the EFT, or an analogue thereof.

The notion of superior performance of individuals with ASD in the EFT or its analogues has been challenged in a number of studies (Brian & Bryson, 1996; Kaland, Mortensen, & Smith, 2007; Ozonoff,

Pennington, & Rogers, 1991; Schlooz et al., 2006), but none has yet provided a convincing disconfirmation of either the Shah and Frith (1983) or the Jolliffe and Baron-Cohen (1997) results. Schlooz et al. (2006) do, however, provide a good summary of the conditions under which this superior performance is found. Jolliffe and Baron-Cohen (1997) emphasise that a key element of the procedure in the adult version of the task is to require participants to view and describe the overall design before attempting to locate the embedded figure. This obviously encourages a more "global" approach to the overall design and may impair detailed search in those with a tendency to process more globally anyway. This could be described as a form of priming at the global (i.e. overall pattern) level which individuals with ASD are less prone to.

The balance of evidence, therefore, seems to favour superior performance in the Embedded Figures Task by individuals on the autism spectrum, relative to IQ- or MA-matched controls. Furthermore, the neuroimaging evidence suggests that individuals with ASD employ different neural circuits to solve this task as compared to typical individuals.

8.2. Block design

The Block design sub-test (BDT) forms a part of many standard IQ test batteries (e.g. Wechsler, 1974, 1981). There have been numerous previous demonstrations of superior performance by ASD populations in the BDT relative to other IQ sub-tests (e.g. Lockyer & Rutter, 1970; Venter, Lord, & Schopler, 1992). The task involves constructing a particular bichromatic pattern from a fixed number of (identical) blocks with bichromatic sub-patterns on each face as quickly as possible (see Fig. 7). In Shah and Frith's (1993) study, there were 40 patterns in total and both accuracy and time to construct were measured. Shah and Frith (1993) used five different groups of participants: High (85+) and low IQ (below 85) participants with autism, both mean age ~18 years; a typical group of children with typical IQ, mean age ~16 years; a younger group of typical children, mean age ~11 years and a group of older children with learning disabilities, aged ~18 years, but matched to the low-IQ autism group on performance IQ, and the high-IQ autism group on VIQ. Shah and Frith (1993) confirmed that, for both autism groups, the BDT gave the peak performance of all the IQ sub-tests in the appropriate section of the Wechsler subscales, they then looked at several different block designs, dividing the patterns up into those with horizontal and vertical lines only and those with oblique lines only. These were presented both in the normal orientation and at oblique orientations and "whole" (undivided) or "segmented", where the design is split into its constituent components, thereby partially solving the task for the participant. The key result was that the autism group performed better than controls only when the block design was presented in its whole, unsegmented form, suggesting that their superiority in the task was dependent on a superior ability to break the original pattern down into sub-patterns.

The Shah and Frith (1993) study has been supported in more recent work by Happé (1994), Ruhl, Werner, and Poustka (1995), Siegel, Minshew, and Goldstein (1996), Ehlers et al. (1997), Dennis et al. (1999), Ropar and Mitchell (2001) and Spek, Scholte, and van Berckelaer-Onnes (2008).

Caron, Mottron, Berthiaume, and Dawson (2006) divided both their ASD and control groups into those with and without an individual "Block Design Peak" in IQ test batteries (Siegel et al., 1996). In their geographically localized sample, 47% of the ASD-diagnosed population showed this peak, as compared with 2% of the typical population. They also differentiated low and high "perceptual coherence" (PC) block designs. In low PC designs, the block structure is obvious from the pattern structure due to the larger number of intensity edges which align with block segmentation points; in high PC designs, the contours from the blocks combine to make elongated patterns that, in some cases, resemble grating patterns (Royer & Weitzel, 1994). Caron et al.'s (2006) results were largely similar to Shah and Frith's (1993), but highlighted the importance of perceptual coherence in unsegmented block patterns and the potential role of block design "giftedness" in comparisons between ASD and control populations. These issues may explain the failures of Kaland, Mortensen, and Smith (2007) and Bölte et al. (2007) to find differences between high-functioning individuals with ASD and IQ-matched controls.

Bölte, Hubl, Dierks, Holtmann, and Poustka (2008) used an adaptation of the BDT to look at fMRI activations in a group of adolescents and adults with ASD and controls with similar NVIQ. The most significant result was a reduction in the activation of V2v between ASD and control groups while performing the BDT, despite equivalent behavioural performance. Bölte et al. (2008) interpreted this reduced activation as evidence for a reduction in the formation of visual contours in the ASD group.

In summary, the evidence seems to be quite strong that a proportion of individuals on the autism spectrum will show superior,

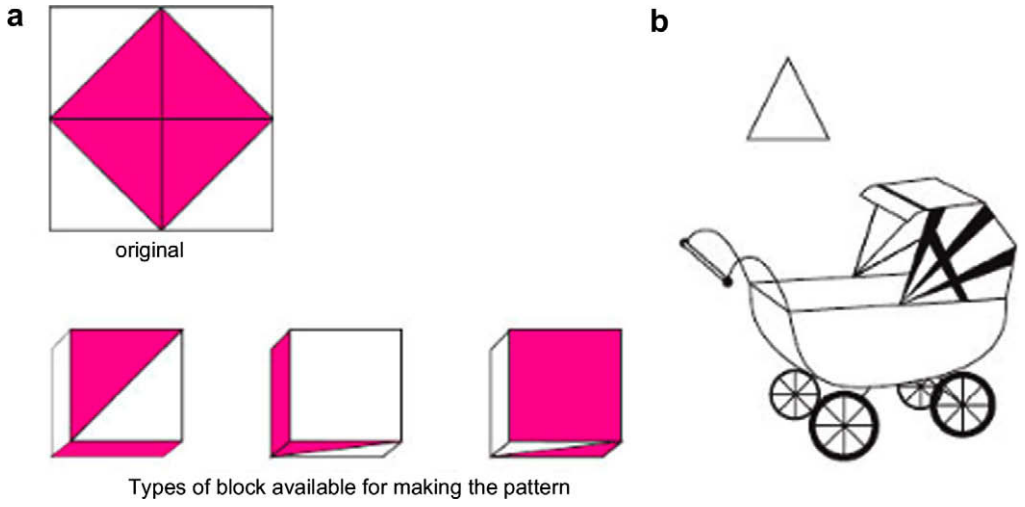

Fig. 7. (a) The Block Design task. (b) Example from the Embedded Figures Test. Find the triangle ("tent") inside the pram/stroller. Image taken from Happé, F. (1999) 'Understanding assets and deficits in autism: why success is more interesting than failure', Spearman Medal Lecture. *The Psychologist*, vol. 12, No. 11, November 1999 via OpenLearn LabSpace (labspace.open.ac.uk).

or at least equivalent, performance to matched controls on the BDT, and that it is potentially solved in a different way. However, this task involves a number of stages: segmenting the image, choosing the correct blocks and then constructing them appropriately, any of which stages could potentially present problems to individuals with ASD, depending on the details of their symptoms. Hence, in visual perception terms, the Block Design Task is probably not as informative as other more direct measurements of visual performance.

8.3. Visual search

Both the Embedded Figures Task and the Block Design Task can be regarded as forms of visual search. Plaisted, O'Riordan, and Baron-Cohen (1998b) looked directly at two search tasks: in the "feature" search task, the target shared colour with one set of distracters but was unique in shape; in the "conjunctive" search task, the target shared colour with one set and shape with another set of distracters. In all cases the stimulus micropatterns were letters (X,T,S) and the colours used were red and green. Children with autism (7–10 years) were compared with a typical control group matched on VMA. Note that the children with autism had higher *Spatial* Mental Ages than the controls, as measured by the BDT. Unlike the control children, the children with autism showed no significant slowing in reaction time in the conjunctive task compared to the feature task and were, in fact, faster at finding the conjunctive target than the controls. O'Riordan, Plaisted, Driver, and Baron-Cohen (2001) confirmed the Plaisted et al. (1998b) result using a different control group, matched by non-verbal ability, and extended it to look at search asymmetries. RTs for finding a tilted line in a background of vertical lines were similar for both autism and control groups, but the autism group found the opposite task – finding a vertical line in a background of tilted lines – significantly easier than matched controls. Other measurements of visual search performance were consistent with the children with autism having superior ability to matched controls on serial search tasks.

O'Riordan and Plaisted (2001) took this investigation even further, using complex triple conjunction search tasks to argue that the key factor in the superior performance of children with autism on visual search tasks is their superior ability to discriminate targets and distracters (see also Plaisted, O'Riordan, & Baron-Cohen, 1998a). O'Riordan (2004) has extended this finding to adults on the autism spectrum.

Jarrold et al. (2005) used a "difficult" feature search task, where the feature was quite similar to the distracters (e.g. a red X-shaped clown amongst green T- and red O-shaped clowns) and a conjunction search task (e.g. the same red X-shaped clown amongst green X-shaped and red T-shaped clowns). They found, similar to O'Riordan et al. (2001), that visual search performance was better for both feature and conjunction search in their group of children with autism than in their developmental-age-matched control group.

Caron et al. (2006) used their population separation in terms of BDT performance to look at visual search as well, using stimulus micropatterns that resembled the bicolour blocks from the task (see Fig. 8). They found that reaction times for both featural and conjunction search, were faster for the individuals with Block Design peaks, irrespective of diagnosis, than they were for those without.

Rinehart, Bradshaw, Moss, Brereton, and Tonge (2008) recently demonstrated that Inhibition of Return (IOR), another aspect of visual search, is intact in young people with autism and AS, and possibly even slightly superior to control performance.

Keehn, Brenner, Palmer, Lincoln, and Muller (2008) performed an fMRI study of visual search performance with adolescents with ASD and typical controls, matched on NVIQ. Despite only marginal differences in search performance between the two groups the BOLD activations in the ASD group were more extensive, recruiting a network involving frontal, parietal and occipital cortices, whereas activations in the typical group were more confined to occipito-temporal regions. Direct comparisons revealed greater activations in occipital and frontal regions in the ASD group.

To summarise the data on visual search, the bulk of evidence seems to favour superior performance from ASD groups to matched controls, even in quite challenging multiple conjunction searches, and the argument that this could be achieved due to enhanced discrimination of the micropattern elements is compelling (O'Riordan & Plaisted, 2001; O'Riordan et al., 2001; but see Bott, Brock, Brocdorff, Boucher, & Lamberts, 2006, for a dissenting voice).

8.4. Spatial visual attention

8.4.1. Visual orienting

The literature on overt and covert visual orienting to non-social stimuli in ASD is highly contentious. Wainwright-Sharp and Bryson (1993), Casey, Gordon, Mannheim, and Rumsey (1993), Courchesne et al. (1994), Townsend, Courchesne, and Egaas (1996a), Town-

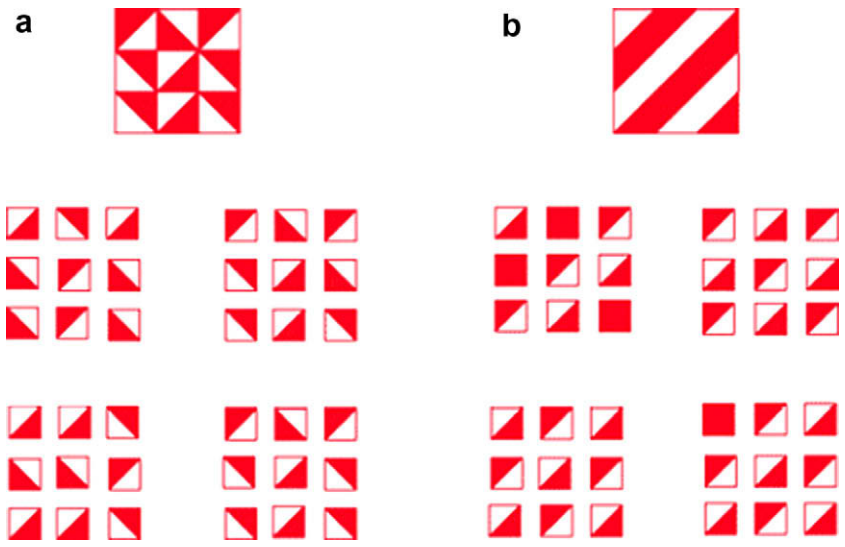

Fig. 8. Visual search task from Caron et al. (2006). Note that pattern (b) has much higher Perceptual Coherence than pattern (a). Reproduced with permission of Oxford University Press.

send, Harris, and Courchesne (1996b) and Dawson, Meltzoff, Osterling, Rinaldi, and Brown (1998) have argued that visual orienting is impaired in ASD. Wainwright and Brown (1996) and Harris, Courchesne, Townsend, Carper, and Lord (1999) reported idiosyncratic visual-spatial orienting performance, but Pascualvaca, Fantie, Papageorgiu, and Mirsky (1998), Leekam, Lopez, and Moore (2000), Iarocci and Burack (2004) and Senju et al. (2004) argued that ASD populations do not have a deficit in shifting attentional focus. Renner, Klinger, and Klinger (2006) found impairments in exogenous (reflexive), but not endogenous (voluntary) orienting performance between their diagnostic groups.

Van der Geest, Kemner, Camfferman, Verbaten, and van Engeland (2001) directly measured attentional shifts using eye tracking. They compared the performance of 16 children with HFA, aged 10–11 years, with CA- and IQ-matched controls in the "gap-and-overlap" paradigm. This paradigm uses two phases: in the overlap condition, the fixation point is visible when the target is presented (in another place); in the gap condition, nothing is visible at fixation when the target is presented. Saccadic latencies to the target are much shorter in the gap condition, and the difference is called the "gap effect". It is argued that the two conditions involve different levels of attentional disengagement and hence can be used to analyse the strength of these components to visual orienting behaviour (Fischer & Weber, 1993). Van der Geest et al. (2001) found that, although there were no significant differences between the autism group and controls in each condition individually, the gap effect was smaller in the autism group, suggesting that children with autism have lower levels of attentional engagement. This result was replicated in a similar clinical population by Kawakubo, Maekawa, Itoh, Hashimoto, and Iwanami (2004).

Goldstein, Johnson, and Minshew (2001) compared 103 carefully characterized individuals with ASD to the same number of matched controls on a battery of tests of attentional processing and performed a complex factor analysis of the results (Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991). They argued from their analyses that apparent deficits in attention in ASD could be due to deficits in complex decision making, or in psychomotor abilities.

Greenaway and Plaisted (2005) used a different approach. They compared top-down and bottom-up control using a combination of cueing and visual search tasks employing task-irrelevant distractors. Their ASD group consisted of 31 children, aged 9.5–13.5 years, and a similar number of typical controls, matched on NVIQ (Raven's matrices). Two types of target were used, which they describe as "onset" (i.e. a single white symbol) or "colour" (i.e. a red symbol among white symbols). They found that the performance of the ASD group, in terms of top-down modulation, was equivalent for the colour stimuli, but not for the onset stimuli. They suggested that individuals with ASD have difficulty prioritizing dynamic stimuli, and linked this to a magnocellular pathway processing deficit (Greenaway & Plaisted, 2005).

Belmonte (2000) and Belmonte and Yurgelun-Todd (2003) have looked at the neural basis of covert attention shifting in ASD, using ERP and fMRI analysis, respectively. In both cases unusual activations were found in the ASD group. In particular, Belmonte and Yurgelun-Todd (2003) found less frontal activation and more occipital activation in the ASD group. Haist, Adamo, Westerfield, Courchesne, and Townsend (2005) also found this, at least for short ISI attention tasks, but also reported hypo-activation of the cerebellum in either short or long ISI conditions. They suggested that individuals with ASD show a profound deficit in automatic spatial attention ability and an abnormal voluntary attention ability.

8.5. Attention to spatial scale

A number of studies have suggested that global grouping processes are deficient in ASD due to the unusual performance in the Navon task (Navon, 1977). In this task, a figure, such as a large letter 'S', is built up from smaller figures, such as small letter 'c's. Participants are asked to name either the small or the large letter. Typical observers find it easier to name the larger than the smaller letter, and this is reflected in differential performance in the task (the "global superiority effect"). Using Navon-type stimuli, some studies have reported that participants with ASD tend to be more sensitive to the local stimulus than controls (Rinehart Bradshaw, Moss, Brereton, & Tonge, 2000, 2001 (autism only, not AS), Gross, 2005; Behrmann et al., 2006a), but others have not (Deruelle, Rondan, Gepner, & Fagot, 2006; Edgin & Pennington, 2005; Mottron, Burack, Iarocci, Belleville, & Enns, 2003; Mottron, Burack, Stauder, & Robaey, 1999b; Ozonoff, Strayer, McMahon, & Filloux, 1994; Rondan & Deruelle, 2007).

Further evidence for enhanced attention to local visual information in ASD comes from Jarrold and Russell's (1997) counting experiment, where children with autism tended to count each dot individually rather than rapidly and automatically enumerate them (see also Gagnon, Mottron, Bherer, & Joanette, 2004; Mottron et al., 1999a). Plaisted et al. (1999) suggested that the finding of local precedence in children with autism depended on task instructions. Plaisted et al. (1999) used the Navon task and either told the children whether the stimulus was at the local or global level (selective attention task) or gave them no instructions (divided attention task). They found that no global advantage (local precedence) was found in the autism group in the divided attention task, but that performance was the same as controls in the selective attention task. These results suggested that switching the spatial scale of attention was most problematic for the children with autism, and that the default state might well be set at a finer scale than that of typical children.

Other suggestions concerning the spatial spread of attention in ASD, tested using a variety of tasks, have been that individuals with ASD have difficulty inhibiting distractors (an "inefficient attentional lens": Burack, 1994; contradicted by Ozonoff & Strayer, 1997), problems with broadening the spread of attention (Mann & Walker, 2003), a "local bias" (Behrmann et al., 2006a), a "piecemeal strategy of processing" (Iarocci, Burack, Shore, Mottron, & Enns, 2006) or have no difficulties relative to controls (Plaisted, Dobler, Bell, & Davis, 2006).

Rondan and Deruelle (2007), in another variation on the Navon-type task, looked at configurational matches versus local matches in schematic face and non-face stimuli. Participants were given the choice between matching a pattern with the same spatial configuration but different constituent shapes or the same shapes in a slightly different configuration. Adults with ASD favoured the local shape match rather than the configuration match, unlike the typical controls, these being the same adults with ASD who had shown equivalent performance to the controls on a Navon-type task. Rondan and Deruelle (2007) argue that configurational, rather than global, processing is disrupted in ASD.

In an epic attempt at empirical and theoretical synthesis, Wang, Mottron, Peng, Berthiaume, and Dawson (2007) presented an exhaustive study of different Navon-type tasks with individuals on the autism spectrum, varying such factors as response type, exposure time and stimulus size. They interpreted their complex pattern of results as evidence for atypical local-to-global interference in ASD and local advantages in incongruent conditions, where the local and global stimuli are unmatched. They also argued that this local bias for visual processing potentially gives individuals on the autism spectrum a flexibility which is unavailable to typical individuals and which may sometimes enhance and sometimes diminish performance in visual tasks.

A final spanner in the works of this confusing literature comes from Rutherford et al. (2007b). They used the "Useful Field of View" task, usually used to look at the spatial scope of attention

in elderly participants and drivers (e.g. Sekuler, Bennett, & Mamelak, 2000), to assess the attentional span of adults with ASD (mean age 25) under different amounts of central attentional load. This task comprises a central letter-identification task (focused-central) with a peripheral task (focused-peripheral) which requires the participant to choose the "spoke" of the "wheel" in which a flashed square was presented in a 4AFC. The final task is a divided attention task in which both central and peripheral stimuli are presented at the same time and two responses are required. The performance of the ASD group was broadly comparable to that of the controls, but a difference was found in the "attentional cost" of divided attention. Whereas performance of the ASD group in the peripheral task was barely affected by the need to perform the central task at the same time, the control group was significantly worse. Curiously, the ASD group seemed to be more strategic than the controls in maintaining their attentional scope at an intermediate level: good enough to do the central task and maximizing performance in the peripheral task. Rutherford et al. (2007b) argue that this result is problematic to explain in terms of current models of visual processing in ASD and also incompatible with much of the previous literature on attention in ASD (e.g. Plaisted et al., 1999).

8.6. Social attention

Social attention refers to the tendency for social stimuli – largely faces and people – to attract attention. This is obviously likely to be an area of difficulty for people with ASD. Some of the material relevant to this section has already been covered in the face and attention sections above, so only specific studies on social attention will be mentioned here.

Swettenham et al. (1998) examined videotapes of children engaged in a structured play situation. The children were very young (about 20 months), being part of a study into the early diagnosis of autism (Baron-Cohen et al., 1996). They found that whereas the control groups tended to focus most on people, shifting attention mainly from objects to people, the autism group tended to shift from objects to other objects. The autism group also spent less time looking at people and more time looking at objects (see also Swettenham et al., 2003; Willemsen-Swinkels, Buitelaar, Wejnen, & van Engelund, 1998).

Probably the most famous study in this area is that of Klin et al. (2002) who asked 15 men with HFA and typical controls to view social scenes (extracts from the movie "Who's Afraid of Virginia Woolf") whilst their eye movements were tracked. Upon coding, the best predictor of autism was reduced eye-region fixation time. Individuals who fixated more on mouths than objects tended to score higher in social functioning tests, whereas those who focused more on objects showed the opposite tendency. Klin et al. (2002) argued that, in natural social scenes, eyes are the least salient stimulus for individuals with ASD, and mouths, bodies and objects are relatively more salient. See also Jones and Klin (2008).

Bird, Catmur, Silani, Frith, and Frith (2006) compared attentional modulation to house and face stimuli in an fMRI study using 16 individuals with ASD and matched controls. Analysis showed that responses to houses were modulated by attention in both groups, but only the controls showed attentional modulation to faces. An analysis of effective connectivity suggested that in the ASD group there was a failure to modulate connectivity between extrastriate areas and V1: which could result in a reduced salience for social stimuli.

Fletcher-Watson, Leekam, Benson, Frank, and Findlay (2009) presented adults with ASD with a complex social scene alongside another non-social scene and measured eye movements during a 3-s viewing period. Their measurements indicated a "superficially normal" attentional preference for social information in the ASD group, but they also found some subtle abnormalities which may be significant in real-life scenarios. Riby and Hancock (2008) have reported reduced attention to people and faces in static pictures of social interactions, in contrast to their control sample of individuals with Williams syndrome. See also Riby and Hancock (2009a, 2009b) for supporting data using different paradigms and stimuli.

The prevailing view on social attention is, then, that individuals with ASD have difficulty with attending to people, particularly the eye regions of faces, in a range of stimulus situations. However, sometimes this difficulty is not readily apparent, especially in controlled laboratory tests.

8.7. Visual attention – summary

The literature on visual attention in ASD presents a confusing picture. There is probably enough evidence to suggest that the processing of information across spatial scales works differently in ASD, but precisely how is too early to call. Individuals with ASD also seem to have problems with attending to people in natural animated scenes, but whether this is due to low-level visual factors or higher-level motivational factors is still unclear.

9. Oculomotor problems

Goldberg et al. (2002) looked at saccadic functioning in 11 individuals with HFA and typical controls, and using a number of standard paradigms (anti-saccade, memory-guided saccade, predicted saccade and gap/overlap tasks). There were a number of differences between ASD and control performance on these tasks, which suggested significant abnormalities in oculomotor function. Goldberg et al. (2002) suggested that these abnormalities implicated brain regions such as the dorsolateral pre-frontal cortex and the frontal eye fields, as well as basal ganglia and parietal lobes as being affected in ASD.

Takarae, Minshew, Luna, and Sweeney (2004) measured visually guided saccades in 46 individuals with HFA/AS and IQ-matched controls. The ASD group was found to have increased variability in saccade accuracy, together with mild saccadic hypometria in those without delayed language development. Takarae et al. (2004) suggested that this pattern of saccadic abnormality was consistent with a chronic functional disturbance in the cerebellar vermis or its output in the fastigial nuclei. Later results suggested cerebellar or fronto-striatal circuitry atypicalities along with extrastriate areas that extract visual motion information or its transfer to sensorimotor areas (Takarae, Minshew, Luna, & Sweeney, 2005a; Takarae, Minshew, & Sweeney, 2005b).

However, despite this evidence in favour of eye movement abnormalities in ASD, Van der Geest, Kemner, Verbaten, and van Engelund (2003) tested a variety of eye movement parameters as well as attentional tasks in a group of children with autism and found that only the attention task showed group differences.

Finally, Mottron et al. (2007) described atypical exploratory behaviours towards inanimate objects among young children with autism. The most frequent atypical behaviour amongst 15 children with autism aged 33–73 months was "lateral glancing", mostly towards moving stimuli. This behaviour is described as "Fixating on a target with the pupils turned toward an extreme corner of the eye socket, where the head is turned in the opposite direction". Mottron et al. (2007) explain this behaviour as an attempt to over-stimulate peripheral vision in order to regulate excessive amounts of local detailed information.

10. Change detection

People with ASD often show strong reactions to changes in the environment, suggesting that they may detect changes more efficiently than typically developing people. However, Fletcher-Watson, Leekam, Turner, and Moxon (2006) reported no differences in performance between adults with autism and typical adults in a change-detection task. Burack et al. (2009) initially also found no differences in change-detection performance between children with autism and typical children matched in non-verbal MA, but differences emerged when the detection failures were related to the developmental level of the participants. Detection failures decreased with increasing developmental level for typical children, but remained constant over the same developmental range for children with autism, pointing to an atypical developmental trajectory for change-detection among children with autism.

11. Neuroimaging studies

This section covers neuroimaging data not already covered above. Relevant reviews include DiCicco-Bloom et al. (2006) or can be found in the collected volume Bauman and Kemper (2004).

11.1. Neuroanatomy

The developmental neuroanatomy of autism has recently been reviewed by Courchesne et al. (2007). The key feature in early development seems to be early brain overgrowth, possibly due to a failure of neural pruning, and consequent abnormalities in neural patterning and wiring. This pattern would be extensive local interconnectivity at the expense of long-range connectivity, which could clearly result in disruption of a number of critical neural circuits. See also McAlonan et al. (2005, 2008) and Hallahan et al. (2009) for more detail on current neuranatomical findings.

11.2. Electrophysiology

Atypical ERP and EEG responses to visual stimulation have been found in a number of studies on ASD (see reviews by Dawson et al., 2005; Kemner & van Engelund, 2006). These include abnormal spatial frequency processing (Boeschoten et al., 2007; Milne et al., 2009), and apparent functional under- and over-connectivity (Murias, Webb, Greenson, & Dawson, 2007).

11.3. Functional magnetic resonance imaging

Villalobos, Mizuno, Dahl, Kemmotsu, and Müller (2005) looked at functional connectivity along the dorsal stream during visually prompted button presses in a group of eight high-functioning adults with ASD and matched controls. They found intact functional connectivity in the ASD group between V1 and superior parietal areas, suggesting that dorsal stream connectivity was intact. There was, however, reduced functional connectivity between V1 and inferior frontal areas, which Villalobos et al. (2005) suggest may be consistent with mirror-neuron system dysfunction. In contrast, however, Mizuno, Villalobos, Davies, Dahl, and Muller (2006) found evidence for hyper-connectivity between thalamus and cerebral cortex using BOLD signal cross-correlation. Further functional connectivity abnormalities have been found between FFA and amygdala during face processing (Kleinhans et al., 2008).

Hadjikhani et al. (2004a) mapped visual cortical areas in a group of individuals with HFA, and found that early visual areas were organized as in typical controls, with typical ratios between central and visual field representations. They argue that any visual processing atypicalities in autism must come from higher-level visual areas and be the result of top-down influences.

12. Summary of visual processing studies

A considerable amount of data has been added to the field since the reviews of Dakin and Frith (2005) and Behrmann et al. (2006b), particularly using faces as stimuli. However, most of these data are contradictory, or at least contested, so it is very hard to say anything with certainty. The new findings on colour vision difficulties (Franklin et al., 2008, in press; Heaton et al., 2008) are interesting and of theoretical importance, but await further replication with better characterized ASD populations. The possibility that a visual perception task might be able to distinguish individuals with autism from those with Asperger Syndrome is also exciting (Spencer & O'Brien, 2006; Tsermentseli et al., 2008), but there are methodological and replication issues with these results as well. Another issue for these studies is the likely disappearance of Asperger Syndrome as a distinct diagnostic category in the upcoming DSM-V. The difficulties for ASD participants with complex motion stimuli, which seemed so clear cut a few years ago, are now much more contentious, but local superiority effects in tasks such as visual search seem to be holding their own. The literature on face processing and visual attention is still highly contentious. The balance is in favour of an attentional difficulty resulting in unusual eye gaze, which then results in unusual visual experience and development. The neuroimaging data indicates unusual patterns of brain activations, particularly when eye movements are not controlled, and there is a trend of finding higher activations at lower levels of visual processing, consistent with the idea of some sort of bottleneck in the processing pathway somewhere between primary visual cortex and higher visual areas. Difficulties with eye regions of faces and low spatial frequencies are also fascinating, but much work remains to be done. The issue of visual acuity should be resolved following Ashwin et al. (2009), and no work has yet been done on stereopsis. A really basic characterization of the visual processing capabilities of people with ASD would be extremely useful, even if all it could do was say with certainty "nothing is wrong here", as some have already suggested (e.g. Hadjikhani et al., 2004a). However, our suspicion is that there are a number of subtle differences between the visual processing capabilities of ASD and typical populations which remain to be discovered. The way forward must be for vision scientists, who know how to run rigorous psychophysical experiments, to collaborate with those of a more clinical orientation, who know how to characterize clinical populations accurately.

13. Theories of visual processing in ASDs

13.1. Cognitive theories

The dominant cognitive theories in ASD research have recently been reviewed by Rajendran and Mitchell (2007). One of the most influential ideas in the past 25 years has been the "Theory of Mind" (ToM) hypothesis, championed by Simon Baron-Cohen and colleagues at the University of Cambridge (Baron-Cohen, 1995; Baron-Cohen, Leslie, & Frith, 1985). The theory focused on explaining the poor performance of children with ASD on "false belief" tasks, where participants are required to interpret a situation from the point of view of another, and argued that the central deficit in ASD was the ability (and possibly also motivation) to "read" others' minds in social situations. Frith (2003) prefers to call this ability "mentalizing". The ToM hypothesis did not really have any specific predictions about visual processing in ASD, other than a difficulty with interpreting facial expressions,

as demonstrated in the "Reading the mind in the Eyes" task (Baron-Cohen et al., 1997b). Recently this theory has metamorphosed into the "Extreme Male Brain" hypothesis (Baron-Cohen, 2002, 2003), which argues that ASD is characterized by poor "empathizing" and high "systemizing". Empathizing is related to Theory of Mind in that it involves interpreting the feelings of others, but adds that feelings must not only be interpreted, but also experienced by the viewer. Systemizing, on the other hand, is the ability to analyse rule-based systems and is a trait associated with mathematical, engineering and scientific ability. In visual processing terms, again, the implications of this theory are not direct, but if people with ASD are poor empathizers and good systemizers, one might expect a deficit in the recognition and interpretation of complex facial expressions and possibly also a skill in visual analysis of the components of complex images. This latter skill, however, is possibly more associated with an alternative theoretical account of ASD: the "Weak Coherence" account (WC; Frith, 1989; Happé & Frith, 2006).

Frith's (1989) original formulation of weak coherence (originally called "weak central coherence") was built on the observation of local processing biases in ASD populations, combined with the relative failure to extract the gist or meaning of events in everyday life. The account was supported by the original observations in ASD populations of superior disembedding performance in the Embedded Figures Task (Shah & Frith, 1983), superior performance in the Block Design Task (Shah & Frith, 1993) and reduced sensitivity to visual illusions (Happé, 1996). However, Happé and Frith (2006) have now reformulated the account in three important ways in response to other empirical findings: (1) it is no longer regarded as a "core deficit in central processing" in ASD, but as a more secondary outcome characterized by a local or detail-focused processing style; (2) this processing style can be overcome in some situations; (3) it is one aspect of a more detailed cognitive profile which includes problems with Theory of Mind.

The final dominant cognitive theory of ASD has been the "Executive Dysfunction" account (Hill, 2004, 2008; Ozonoff et al., 1991; Russell, 1997). This theory focuses on the difficulties that individuals with ASD have with tasks involving planning, mental flexibility, generativity, inhibition and multi-tasking (Hill, 2008). The original motivation behind the theory was to account for the non-social symptoms, such as repetitive and obsessional behaviours, which were not well explained by ToM or WC accounts. Again, there are few precise predictions about visual performance that come from the Executive Dysfunction theories, although difficulties with visual attention (especially attentional disengagement and divided attention), scene exploration and eye-movements might be expected.

13.2. Neural theories

One approach to understanding the neural basis of ASD is to compare its signs and symptoms with those of conditions and syndromes of known neurological origin. This approach has led to suggestions of pathology in a large number of brain areas in ASD, including the cerebellum (Courchesne, 1997), the temporal lobes, especially the STS and the FFA (Zilbovicius et al., 2006), the amygdala (Baron-Cohen et al., 2000; Howard et al., 2000) and the frontal lobes (Courchesne & Pierce, 2005). Recent attempts at synthesis have attempted to map the triad of impairments which is thought to characterize autism onto specific brain regions (Baron-Cohen & Belmonte, 2005; Frith, 2003). This has led to the idea of "social brain" dysfunction in ASD: a network of brain regions which includes the Superior Temporal Sulcus, Superior Temporal Gyrus, Inferior Occipital Gyrus and Fusiform Gyrus in the occipital and temporal lobes with the Amygdala and Medial Pre-frontal Cortex, Anterior Cingulate Cortex and Orbitofrontal Cortex (see Fig. 9). These areas seem to safely cover functions such as poor facial expression recognition, difficulties with interpreting eye gaze, problems with integrating sensory information and executive function problems.

What, however, could cause the (unspecified) neuropathology in these brain regions? Recently, Baron-Cohen and co-workers have suggested that the difficulty lies with abnormalities in the levels of the hormone testosterone in the mother's womb, which causes neural developmental abnormalities (Baron-Cohen, 2006).

Other ideas at the neural level, in no particular order, include the absence of "top-down" neural control (Frith, 2003), malfunctioning social reward mechanisms based around the amygdala (Schultz, 2005), neural connectivity abnormalities (Belmonte et al., 2004; Minshew & Williams, 2007; Rippon, Brock, Brown, & Boucher, 2007), abnormal cortical "minicolumns" (Casanova, Buxhoeveden, Switala, & Roy, 2002), abnormal neural synchronization (Brock, Brown, Boucher, & Rippon, 2002), imbalance of excitation and inhibition (Rubenstein & Merzenich, 2003) and imbalanced spectrally timed adaptive resonance (Grossberg & Seidman, 2006).

There has also been a flurry of interest in the "Mirror neuron hypothesis" of autism, which suggests that the symptoms of autism are related to dysfunctions in the so-called mirror-neuron system. Key papers on this topic, in chronological order, are: Williams, Whiten, Suddendorf, and Perrett (2001), Oberman et al. (2005), Dapretto et al. (2006), Iacaboni and Dapretto (2006), Oberman, Ramachandran, and Pineda (2008), Southgate and Hamilton (2008), Oberman and Ramachandran (2008).

With this bewildering, and ever growing, variety of hypotheses about the neural basis of autism, it is difficult, and way beyond the scope of this review, to try to map each of them onto specific predictions about visual processing. Obviously some of these theories have been developed with particular behavioural results in mind, such as superior disembedding performance and it relation to weak coherence which inspires the top-down control hypothesis of Frith (2003), whereas others have emerged almost entirely from studies at the neuroanatomical level (e.g. Casanova et al., 2002). However, there are three key ideas which have inspired much of the recent behavioural work on ASD which do need further treatment here.

13.2.1. Dorsal stream vulnerability

The "dorsal stream vulnerability" hypothesis (Atkinson & Braddick, 2005; Braddick et al., 2003; Spencer et al., 2000) is based on the differential performance in form and motion coherence tasks observed in many neurodevelopmental disorders, including hemiplegia, Fragile-X syndrome, dyslexia and Williams syndrome, as well as autism, suggesting that the neural mechanisms supporting global motion sensitivity are particularly susceptible to damage, possibly because of the more stringent neural timing requirements of this pathway (Atkinson & Braddick, 2005). This theory is not really intended to *explain* autism, particularly as the vulnerability is not thought to be disorder-specific, but rather describes a potential consequence of developmental disability which may be linked back to underlying neuropathological causes.

We have shown in the section on motion perception in ASD that there is no strong evidence for a low-level motion/temporal frequency processing deficit in ASD, that the data on motion coherence is somewhat equivocal and that biological motion is probably affected adversely. These data do not therefore make a strong case for a dorsal pathway problem. As for form processing, Spencer and O'Brien (2006) and Tsermentseli et al. (2008) have found an interesting deficit in autism, but not in Asperger Syndrome. Whilst we have made clear that there are potential problems with the stimuli in these experiments it is hard to see how these problems would cause the pattern of results found. How-

ever, the new data on colour vision anomalies in ASD, the neural basis for which must surely involve parvo- and koniocellular pathways and the ventral stream, suggest that it is unlikely that the neuropathology in ASD is confined to the dorsal stream, unless there are some complicated interactive effects between the two pathways.

13.2.2. Enhanced perceptual functioning (EPF)

Enhanced perceptual functioning theory (Mottron & Burack, 2001; Mottron et al., 2006) has been increasingly influential in recent years as the focus on perceptual processing in ASD has grown. The theory is similar in many ways to Weak Coherence (Frith, 1989; Happé & Frith, 2006) and also shares much with Plaisted's (2001) reduced generalization theory, but its origins are different.

The original theory states that a key aspect of ASD is a heightened response to sensory stimulation in the neural mechanisms of perception from early in childhood (Mottron & Burack, 2001). This heightened response leads to an attentional focus on low-level sensory phenomena, at the expense of social interaction, and a consequent atypical wiring of the brain during development. Mottron and Burack (2001) name four areas of neural development where this manifests itself: growth of neuronal connections, rededication of cortical areas, suppression of inhibition and "functional persistence", meaning the excessive refinement of low-level processes at the expense of higher level processes. The inspiration for this theory came from Mottron's and Burack's work with "savants" on the autism spectrum, particularly those with enhanced graphical abilities (e.g. Mottron & Belleville, 1993) and has been supported by empirical data emerging from the Montreal-based labs of Mottron, Burack and their co-workers (e.g. Bertone et al., 2003, 2005).

In the later update to this model (Mottron et al., 2006), eight "principles of autistic perception" are enumerated, with supporting evidence:

[1] The default setting of perception in individuals with autism is more locally oriented than that of typical individuals.
[2] Increased gradient of neural complexity is inversely related to level of performance in low-level tasks.
[3] Early atypical behaviours have a regulatory function toward perceptual input.
[4] Perceptual primary and associative brain regions are atypically activated during social and non-social tasks.
[5] Higher-order processing is optional in autism and mandatory in typicals.
[6] Perceptual expertise underlies savant syndrome.
[7] Savant syndrome provides a model for subtyping Pervasive Developmental Disorders.
[8] Enhanced functioning of primary perceptual brain regions may account for the perceptual atypicalities in autism.

This is something of a "broad brush" theory, but there are plenty of ideas in it for the vision scientist to consider. Given the recency of the publication of this updated theory, it is largely consistent with current literature on perception in ASD and there has not been much time for it to be put to the test. Mottron et al. (2006) were careful to distinguish the updated EPF theory from its competitors and pre-cursors. They argue that it differs from the Weak (Central) Coherence account in that it is couched in terms of superior rather than inferior performance and that it is

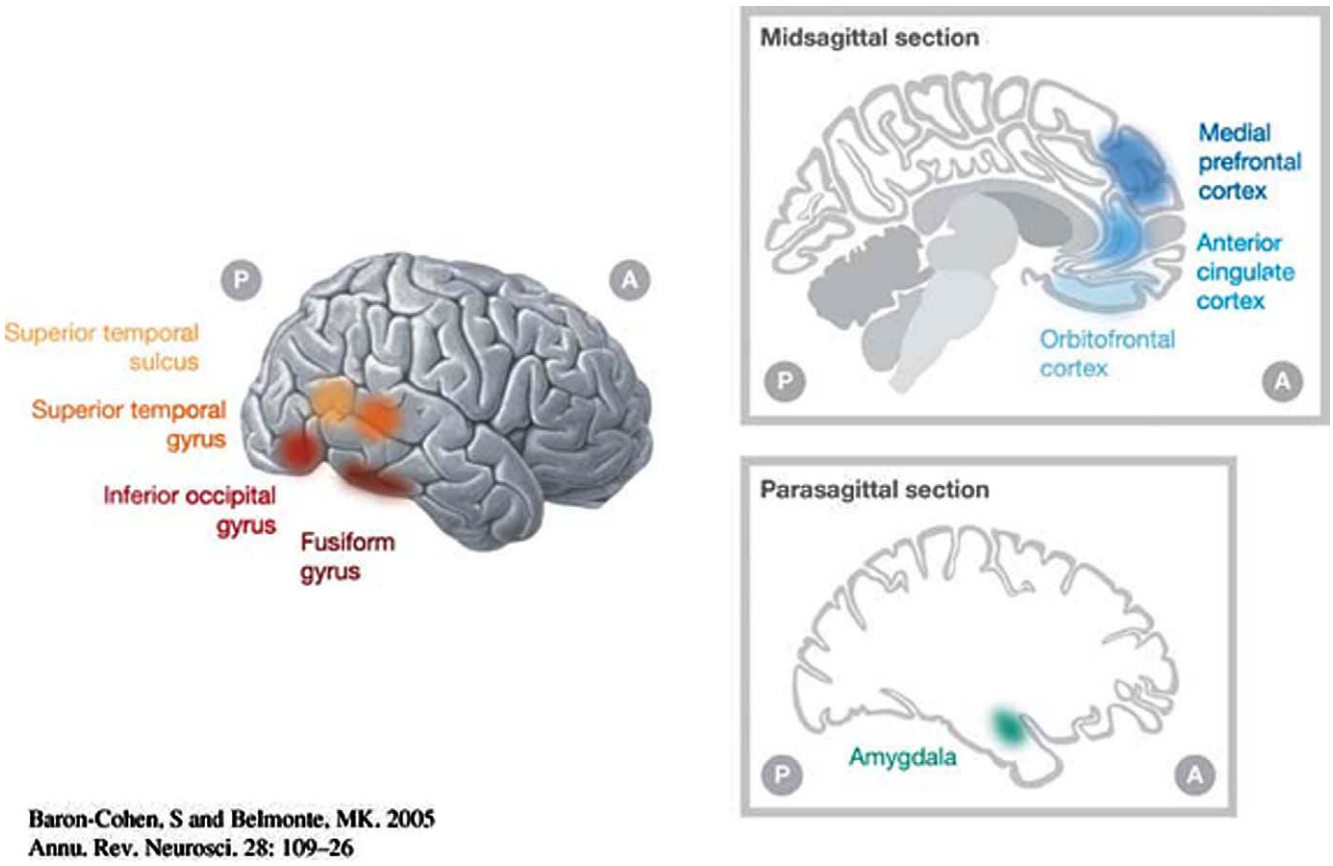

Fig. 9. The social brain. Reproduced with permission from the Annual Review of Neuroscience (www.annualreviews.org).

not an optional "cognitive style" (Happé & Frith, 2006), but a mandatory requirement. There is overlap with Plaisted's (2001) reduced generalization theory but EPF's scope is more comprehensive. Mottron et al. (2006) also borrowed the idea of the importance of informational complexity from Minshew, Goldstein, and Siegel (1997) but mapped it onto the "negative" symptoms of ASD: the expected asymmetry therefore is between superior performance on "simple" tasks (e.g. grating detection, Bertone et al., 2005) and inferior performance on "complex" tasks (e.g. second-order motion, Bertone et al., 2003).

One potential problem with the EPF theory reported here though is the suggestion of reduced ability of ASD populations in chromatic discrimination (Franklin et al., 2008, in press; Heaton et al., 2008). For example, the colour discrimination experiment used by Franklin et al. (in press) is what most vision scientists would call a low-level task, so why is the perception of colour difference not enhanced? Explaining these data within the restrictions of EPF theory is therefore a challenge, assuming that Franklin et al.'s (in press) result is replicable. Another significant challenge within the context of vision science is defining what is "simple" and what is "complex", although that is potentially a more tractable problem (see Bertone & Faubert, 2006).

However, there is another challenge for the EPF model: what exactly is the neuropathology that underlies it? We have recently suggested an explanation for the empirical data on visual processing in ASDs which may have an answer (Simmons et al., 2007). We shall sketch the theory here, although a full version is in preparation (see also Simmons et al., 2008).

13.2.3. Neural noise and autism spectrum disorders

One of the most surprising aspects of the sensory symptoms of ASD is that individuals often report both hyper- and hyposensitivities within the same sense modality (Baranek et al., 2006; Bogdashina, 2003). We were also fascinated with the result of Bertone et al. (2005) (see Fig. 1), suggesting that children with ASD showed a (small) performance enhancement for first-order contrast detection in noise, but a performance decrement for second-order contrast detection. Another observation was the large variability reported in threshold data from ASD populations, relative to both typical and developmentally delayed populations. Whilst this could obviously be due to the inherent diagnostic variability even within the different shades of the autism spectrum, we wondered whether there was a more concrete cause.

Adding noise to a fixed signal intuitively involves a decrease in signal/noise ratio and therefore makes detectability worse. However, the phenomenon of *stochastic resonance* can amplify the signal/noise ratio under appropriate circumstances (i.e. a non-linear system with a fixed threshold, Wiesenfeld & Moss, 1995). This idea, a controversial one in vision science, has recently been used successfully to model contrast discrimination data (Goris, Wagemans, & Wichmann, 2008b; Goris, Zaenen, & Wagemans, 2008a). The detectability advantage for first-order luminance-defined gratings in noise, therefore, could be due to increased internal noise in the visual system of the ASD participants amplifying the signal slightly via stochastic resonance. The poorer performance with second-order gratings, on the other hand, comes because extracting this signal involves combining information across more visual filters, thereby increasing the noise level beyond the point where stochastic resonance can help (Schofield & Georgeson, 1999).

Could this explanation apply to other data reported in this review? Dakin and Frith (2005) already suggested that increased levels of local motion noise could be a factor in explaining the data on motion coherence in ASD. Our analysis has shown that a likely cause of the discrepancies in these data is an increased level of correspondence noise in the ASD populations who have difficulty with seeing the motion, and preliminary data from our laboratory suggest that increased internal noise may be measurable in participants with ASD using equivalent noise analysis, similar to that used by Dakin, Mareschal, and Bex (2005) (Simmons et al., 2007), and that adding noise to biological motion stimuli can be highly disruptive to ASD participants (McKay, Mackie, Piggott, Simmons, & Pollick, 2006).

A further aspect, which is highly speculative, of the explanatory power of an internal noise model for ASD concerns the colour discrimination data of Franklin et al. (in press). Human colour vision involves cone opponency, and it is well known that subtracting noisy signals results in a decrease in signal/noise ratio unless the noise is correlated. It is plausible, therefore, that the benefits of stochastic resonance for detection in ASD will only be found for achromatic, and not chromatic judgements.

Added noise in the internal representations of faces could make the identities of new faces harder to learn, subtle facial expressions harder to distinguish and the eye regions of faces less distinct and salient. Other visual perceptual data, such as superior performance in the Embedded Figures and Block Design Tasks, are harder to explain, but we have already pointed out that these are relatively "high-level" tasks into which a number of component atypicalities could feed. However, a combination of stochastic resonance amplifying local differences and masking hiding global differences would seem to have considerable explanatory mileage in the context of the visual search data at least.

Where could this noise come from? A number of the neural models cited above suggest that there is a proliferation of neural connections in the sensory cortex of individuals with ASD (Belmonte et al., 2004; Casanova et al., 2002; see Baron-Cohen & Belmonte, 2005; Minshew et al., 2007, for reviews), so neural crosstalk within the cortex is one plausible mechanism. However, recent investigations into the genetic basis of ASD have suggested that glutamergic and GABA-ergic synapses could be affected (see Garber, 2007; Persico & Bourgeron, 2006). Mis-firing synapses could easily result in noisy signals in the visual system.

Whilst it might seem somewhat brazen to add another theory to the already theory-rich field of autism research, we would argue that the neural noise model has considerable explanatory power and is parsimonious, but flexible enough to account for a wide variety of phenomena in the literature on vision and ASD, and may well have broader implications for understanding the condition as a whole. The other advantage is that this theory is readily testable using established vision science paradigms such as equivalent noise analysis and is physiologically plausible. Other studies that have suggested that neural noise is involved in ASD include: Rubenstein and Merzenich (2003), Belmonte and Yugelun-Todd (2003), Dakin and Frith (2005), Thornton (2006), Sanchez-Marin and Padina-Marilla (2008), Alcantara (2008), Lugo, Doti, and Faubert (2008) and Franklin et al. (in press).

13.3. A final challenge

Arguably the key theoretical battleground in ASD research at the moment lies between those who favour a "social orienting" theory of ASD development (e.g. Schultz, 2005) and those who favour a more bottom-up theory (e.g. Mottron et al., 2006). In other words, is the information coming in corrupted and therefore causing the problems that manifest themselves as ASD, or is veridical input information corrupted higher up in the brain circuits associated with the social brain, the mirror-neuron system, or the executive function system in the frontal lobes? As often, the truth probably lies in a compromise between bottom-up and top-down mechanisms, as top-down feedback undoubtedly has a role to play.

There are two further questions which emerge from this review. What does "social" mean? Does it mean anything to do with a human being, or possibly animal, such that faces, voices, bodies etc.

count as social and things that are not faces, bodies or voices are non-social? Or is social just another word for complex or unpredictable? If so, what is the critical level of complexity/unpredictability and how can this be quantified?

The second is more prosaic. What is the best way to engage ASD populations in research? One of the curses in this field is the size of the error bars, which always seem to be at least twice as large in the ASD data compared to the controls. Is this just unavoidable or are there effective ways of splitting the ASD population? We have seen that diagnostic measures (Asperger vs. autism), or performance measures (Block Design peaks, Famous face recognition, Verbal IQ) can be useful here (see also Happé et al., 2006), but obviously this technique increases the problem of participant recruitment. These will be further complicated by the imminent updates to diagnostic criteria planned for DSM-V.

A final thing to remember is that the ultimate goal of our research should be to help individuals with ASD to achieve their own goals more effectively.

14. Conclusion

In the 4 years or so since the publication of the Dakin and Frith's (2005) wryly titled review "Vagaries of visual perception in autism" a great deal of data has been produced, and the theoretical landscape of vision research in ASD has changed somewhat, with the even greater influence of neuroimaging techniques. However, some of the most interesting developments have come from old-fashioned psychophysics and it is probably the case that we need more of these before we can progress much further with the brain imaging.

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