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High motion coherence thresholds in children with autism

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Background: We assessed motion processing in a group of high functioning children with autism and a group of typically developing children, using a coherent motion detection task. **Method:** Twenty-five children with autism (mean age 11 years, 8 months) and 22 typically developing children matched for non-verbal mental ability and chronological age were required to detect the direction of moving dots in a random dot kinematogram. **Results:** The group of children with autism showed significantly higher motion coherence thresholds than the typically developing children (i.e., they showed an impaired ability to detect coherent motion). **Conclusions:** This finding suggests that some individuals with autism may show impairments in low-level visual processing – specifically in the magnocellular visual pathway. The findings are discussed in terms of implications for higher-level cognitive theories of autism, and the suggestion is made that more work needs to be carried out to further investigate low-level visual processing in autism. **Keywords:** Autistic disorder, motion perception, central coherence, magnocellular pathway, visual processing.

Autism is diagnosed on the basis of impairments in the areas of social and communicative development, and evidence of restrictive patterns of behaviour and interests (DSM – IV, American Psychiatric Association). Since Kanner's original description of autism in 1943 (Kanner, 1943), research in the area has turned full circle. Having originally focused on perceptual abnormalities in the 1950s and 1960s, interest then shifted in the 1970s and 1980s to more high-level cognitive explanations of the social impairments, before returning to the present interest in attentional and perceptual abnormalities in autism.

Early examples of perceptual abnormalities in autism came from researchers such as Goldfarb (1956) who suggested that in autism there is a discrepancy between the different sense modalities. He based his claim on the observation that children with autism were more likely to smell, lick or touch things than they were to look at or listen to them. Indeed in the late 1960s most of the hypotheses that attempted to explain autism focused on low-level sensory dysfunction. Hermelin and O'Connor (1965) and Frith and Hermelin (1969) carried out a series of experiments that investigated visual discrimination learning and visual tracking in children with autism. Although the results showed differences between the performance of the participants with autism and the performance of the participants without autism, it was later concluded that this could have been due to confounding factors such as intellectual functioning, as control groups were rarely matched for mental age (Hermelin & O'Connor, 1970).

This view remained for many years, and in 1987 Frith and Baron-Cohen wrote a review of a large body of work on perception in autism in which they concluded 'The results from all studies reviewed here are consistent with the hypothesis that low level perceptual processes are intact in autistic children' (Frith & Baron-Cohen, 1987, p. 98). However, despite this claim, recent experimental work has continued to highlight differences in the perceptual processing of individuals with autism when compared to individuals without autism. There are also many anecdotal examples of perception in autism which suggest that some individuals with autism experience abnormal perception. For example, Gerland in her autobiographical account describes having the experience of 'fragmented perception' (Gerland, 1997), and similarly, an account of an individual with autism cited in Jolliffe, Landsdown, and Robinson (1992) states 'I am not looking at the whole but rather just an outline of parts.' Beyond autobiographical accounts there are also observational accounts of children with autism displaying apparent unusual visual perception, as seen in the following description cited in Frith and Baron-Cohen's review article: '… an autistic boy we have seen … could always find small coins long before anyone else in the room had seen them, although he appeared oblivious to many other visual aspects of the environment' (Frith & Baron-Cohen, 1987, pp. 86–87).

The current experimental interest in perception in autism owes much to a recent cognitive theory – the weak central coherence hypothesis (Frith, 1989). Central coherence is the term used to describe the ability to process information in context, and to draw information together to process a 'whole' rather than paying attention to individual details. The weak central coherence theory postulates that autism is characterised by a cognitive style biased towards local rather than global information processing,

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and a failure to process information in context. The theory has empirical support at a number of levels (see Happé, 1999 for a review) although much of the data used to support the weak central coherence hypothesis comes from abnormalities in visual processing. For example, Shah and Frith (1983) demonstrated that children with autism were more competent than typically developing children at completing the Children's Embedded Figures Task, a task which requires children to detect a target figure obscured by distracting information. They then later demonstrated that adults with autism also show superior performance on the Block Design subtest from the Weschler Intelligence Scales (a task which involves using individual blocks to produce a 3D copy of a 2D pattern) (Shah & Frith, 1993). This superior performance is seen not only as compared to their performance on the other subtests, but also relative to individuals without autism. These findings suggest that the individuals with autism are processing the stimuli in terms of their constituent parts, which makes it easier for them to perform the tasks, while the individuals without autism perceive the gestalt and are therefore hindered by the contextual information. Recently, Jarrold and Russell (1997) have shown that children with autism show a tendency towards an analytic style of processing on a counting task, in the sense that these children showed a bias for counting a series of dots individually, rather than subitising. That children with autism appear to process parts of stimuli rather than the whole stimulus has also been demonstrated by Happé (1996), who found that individuals with autism succumbed significantly less frequently to classical visual illusions where visual context induces illusory percepts, than matched non-autistic participants. In a second part of the experiment, Happé drew attention to the relevant parts of the stimuli and artificially disembedded the figures by highlighting the to-be-judged parts with raised lines. Using these adapted figures, typically developing children also succumbed less frequently to the illusions, suggesting that the participants with autism were naturally able to disembed the parts of the stimulus from the whole. However, although there is certainly evidence that children with autism tend to process parts rather than wholes, this does not mean they are unable to process globally when so prompted. Plaisted, Swettenham, and Rees (1999) demonstrated that when given a choice of attending to the local or global level of a hierarchical stimulus (in this case a Navon figure (Navon, 1977) in which global letter shapes are constructed with local consistent or inconsistent letter elements), children with autism responded more rapidly and more accurately to the local level of the stimulus, whereas when instructed to attend to the global level, the same children responded more rapidly to the global level of the stimulus. In contrast, typically developing children always showed a bias for processing at the global level, despite the instructions that they were given. Children with autism *can* therefore process global aspects of a stimulus in a similar way to children without autism, but this may not be the default mechanism as it appears to be in typically developing children (Plaisted et al., 1999). So what might be the mechanisms underlying the lack of global processing and local bias seen in children with autism? Plaisted et al. (1999) have suggested that the processing bias observed in Navon-type tasks might be explained by the relative levels of activity in the two visual channels responsible for high and low spatial frequency processing and that abnormally high levels of activity in the high spatial frequency channel which is responsible for local information processing may characterise visual processing in the autism group.

In the normal population, it is thought that the global advantage of a hierarchical figure such as a Navon stimulus is modulated by low spatial frequency information (Sergent, 1982) and conversely, that the local aspects are processed by the high frequency channels. This is because low spatial frequency signals contain information about the global properties of a stimulus and arrive at the cortex more rapidly than high spatial frequency signals which convey information about the smaller and more detailed aspects of the scene. It has been shown (e.g., Campbell & Robson, 1968) that processing within the visual system is segregated in terms of spatial frequency, in the sense that some channels carry information containing low spatial frequencies and other channels carry information containing high spatial frequencies.

In primates visual information from the retina projects to the primary visual cortex (V1) by way of two independent but linked pathways – the magnocellular pathway, which is relatively more sensitive to low spatial frequency information, and the parvocellular pathway, which is relatively more sensitive to high spatial frequency information. Segregation of the pathways is seen most clearly in the lateral geniculate nucleus (LGN) which is a six-layered structure found at the thalamus. Layers 1 and 2 contain large cells known as magnocells, whereas layers 3 to 6 have smaller cells known as parvocells. These two different cell types have been shown to project to different regions of the visual cortex, and also to have different functional properties (see Maunsell & Van Essen, 1983). A schematic illustration of the segregation of the two pathways is shown in Figure 1.

The cells of the parvocellular pathway (originating from layers 3–6 of the LGN) are most sensitive to high spatial frequencies and stationary or slowly moving targets; they have a low temporal resolution and process information used for wavelength and form discrimination. Cells within the parvocellular pathway project to areas of V1 which form what is known as the ventral stream which terminates at the

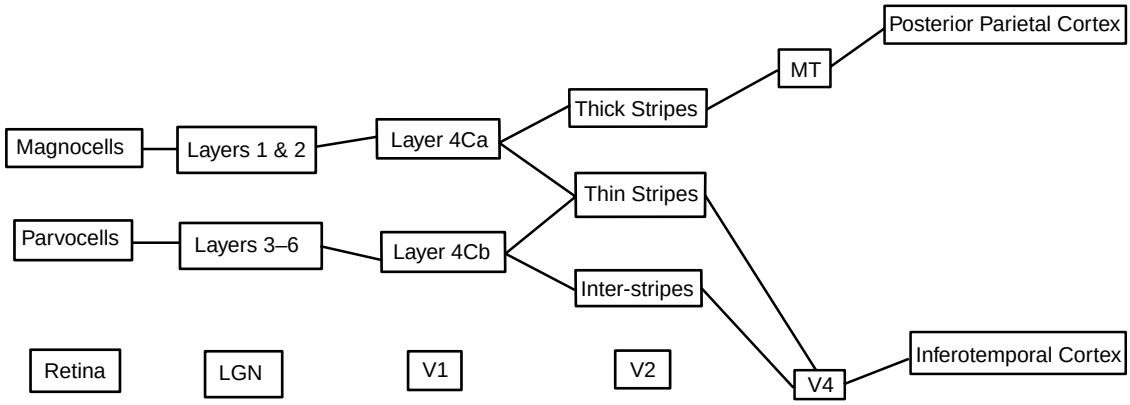

Figure 1 A schematic diagram of the two streams of visual processing. For brevity, only the main destinations of the two pathways are illustrated; see Milner and Goodale (1995) for a more concise description

inferotemporal cortex. Conversely the magnocellular pathway contains cells which are sensitive to low spatial frequencies, moving or flickering stimuli, have a high temporal resolution and process motion, spatial and depth information. In contrast to the parvocellular system, the magnocellular pathway projects mainly to the dorsal stream of visual processing which terminates at the posterior parietal cortex (Livingston & Hubel, 1988).

Plaisted et al. (1999) suggest that the local bias shown under some conditions by children with autism on the Navon task may arise from abnormally high levels of activity in the channels which are responsible for local information processing. An alternative suggestion is that the local bias in these children arises because of abnormally low levels of activity in the channels which are responsible for global information processing.

Given that the global aspect of a stimulus is represented by low spatial frequency information, it is thought that it is the magnocellular visual pathway which underlies this. Evidence to support this claim comes from studies in which the removal of low spatial frequency information from a Navon stimulus using high pass filtering techniques results in significantly slower response times to the global level, and a preference for local over global processing (see Badcock, Whitworth, Badcock, & Lovegrove, 1990). It has also recently been shown that attenuating the magnocellular pathway in normal observers shifts the prototypical asymmetric global interference of a hierarchical stimulus towards a symmetrical interference for both global and local cues. That is, attenuating the magnocellular pathway also attenuates the global precedence effect (Michimata, Okubo, & Mugishima, 1999). Therefore we suggest that impaired low-spatial frequency processing mediated by an impairment of magnocellular functioning may underlie the local processing bias in children with autism reported by Plaisted et al. (1999). This could also underlie the observed preference for the local aspects of a stimulus over the global (weak central coherence); a processing style seen in individuals with autism.

One way to measure magnocellular processing in the visual system is by using a random dot kinematogram, which provides a measure of 'motion coherence threshold' (ability to detect coherent motion from an array of randomly moving dots) (see Talcott et al., 1998; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Cornelissen, Hansen, Hutton, Evangeliou, & Stein, 1998). Random dot kinematograms consist of a visual stimulus composed of moving dots in which a certain proportion of the dots move with a motion vector that is coherent over time whilst the remaining dots randomly change their direction of motion over time (i.e., they move with a 'Brownian' motion). An individual's motion coherence threshold is measured by calculating the lowest proportion of dots that are needed to move coherently for the participant to correctly identify the direction of coherent motion in the array. Because of the functional properties of the magnocellular pathway (sensitivity to high temporal frequencies, flickering and rapidly moving stimuli), a high motion coherence threshold indicates impairment in the magnocellular pathway and/or areas within the visual system which receive input from the magnocellular pathway (Cornelissen et al., 1995; Talcott et al., 1998). It has been shown (Britten, Shalden, Newsome, & Movshon, 1992), using single cell recording techniques to record activity in cortical area MT (strongly served by cells from the magnocellular pathway) in response to random motion stimuli, that random dot kinematograms provide a sensitive measure of magnocellular pathway processing (Talcott, Hansen, Assoku, & Stein, 2000). Also, it is known that lesions to the magnocellular layers of the LGN (but not the parvocellular layers) cause motion blindness in macaques (Schiller, Logothetis, & Charles, 1990). High motion coherence thresholds have been identified in individuals with dyslexia and other reading impairments (Cornellisen et al., 1998). This finding has accompanied other physiological evidence (such as elicited potential

measurement (Livingstone, Risen, Drislane, & Galburda, 1991) and functional magnetic resonance imaging (Eden et al., 1996) that some people who have dyslexia also have impairments in the magnocellular pathway. Although it remains to be seen whether the exact nature of the motion processing impairments in the studies reviewed above occurs at a cortical or a sub-cortical level, or both, the collection of data reviewed above strongly indicates that high motion coherence thresholds as measured by random dot kinematograms are indicative of impairment in the magnocellular stream of processing, by which we also include processing regions in the brain which receive input from the magnocellular stream.

Elevated motion coherence thresholds may feature in several developmental cognitive anomalies as high thresholds have also been identified in children diagnosed with Williams' syndrome (Atkinson et al., 1997). There is evidence to suggest that children with autism do not process visual motion in the same way as children without autism. For example, Gepner, Mestre, Masson, and de-Schonen (1995) measured postural reactivity in response to movement in the visual environment in children with and without autism and found that children with autism showed less postural sway in response to environmental motion than the children without autism. In addition to this, Swettenham, Milne, Plaisted, Campbell, and Coleman (2000) found that children with autism failed to be cued by a moving head stimulus on a cueing task even though movement produced robust automatic effects in a matched group of children without autism.

Recently Spencer et al. (2000), using random dot kinematograms, have found high motion coherence thresholds in a sample of children with autism. In Spencer et al.'s study, participants were required to locate a strip of moving dots which moved in opposite direction to the rest of the dots in the array. The results revealed elevated motion coherence thresholds in a group of children with autism when compared to a group of typically developing younger children matched to the experimental group for verbal mental age. However, the fact that the children with autism were compared with younger, verbal aged matched controls leaves open the possibility that the high thresholds shown by the children with autism were related to general mental handicap (as the IQ of the experimental group was lower than that of the control group). In the present study, high functioning children with autism, matched individually to typically developing children in terms of chronological age and non-verbal IQ, were tested. If the participants in this study show elevated motion detection thresholds also, the inference for a low-level visual processing deficit in autism will be more strongly supported.

In summary, we measured the motion coherence thresholds of a group of children with autism and a group of typically developing children matched for chronological age and non-verbal IQ. We hypothesise that these high functioning children with autism will show increased thresholds and that this may represent an impairment of the magnocellular visual pathway.

Method

Participants

Forty-seven children took part in the experiment, 25 children with autism and 22 typically developing children. All children in the autism group had been diagnosed as having autism according to criteria specified in the DSM – IV (1994), and were attending schools for children with autism. Each typically developing child was individually matched according to age in months and non-verbal mental functioning according to their score on Raven's Progressive Matrices (Raven, Court, & Raven, 1992) with a corresponding child with autism. There were no significant differences between either the mean age of the groups nor their performance on the Raven's Progressive Matrices, $t(45) = .027$, $p > .9$ & $t(45) = -.007$, $p > .9$ respectively, and Raven's scores were within the normal range for the children's ages, indicating that neither group showed developmental delay. Participant demographics are shown in Table 1.

Stimuli

Each participant's motion coherence threshold was measured using a standard Random Dot Kinematogram (RDK) paradigm (see Talcott et al., 1998). The stimuli were presented on a Dell Latitude laptop computer with a screen measuring 248 mm × 188 mm.

The RDK comprised a patch of 150 high luminance white dots (1 pixel) presented on a black background; the stimulus array is shown in Figure 2. Coherent motion percentage was defined as the total number of dots moving together in a single direction (left or right) across the horizontal axis. The remaining dots moved randomly in a Brownian manner. In order to eliminate the possibility of detecting direction by following the movement of one single dot, each dot had a fixed lifetime of 4 animation frames (224.4 ms) after which it would disappear before being regenerated at a random place within the stimulus patch. The total stimulus duration was 18 animation frames or 1009.8 ms. The

Table 1 Mean age, and score on Raven's Progressive Matrices (RPM), for both groups

Group	Mean age (years:months)	Mean RPM scores
Autism ($N = 25$)	11:8	41
SD	*1:4*	*7*
Range	9:4–15:3	25–51
Typically developing ($N = 22$)	11:7	42
SD	*1:6*	*7*
Range	9:6–15:3	28–53

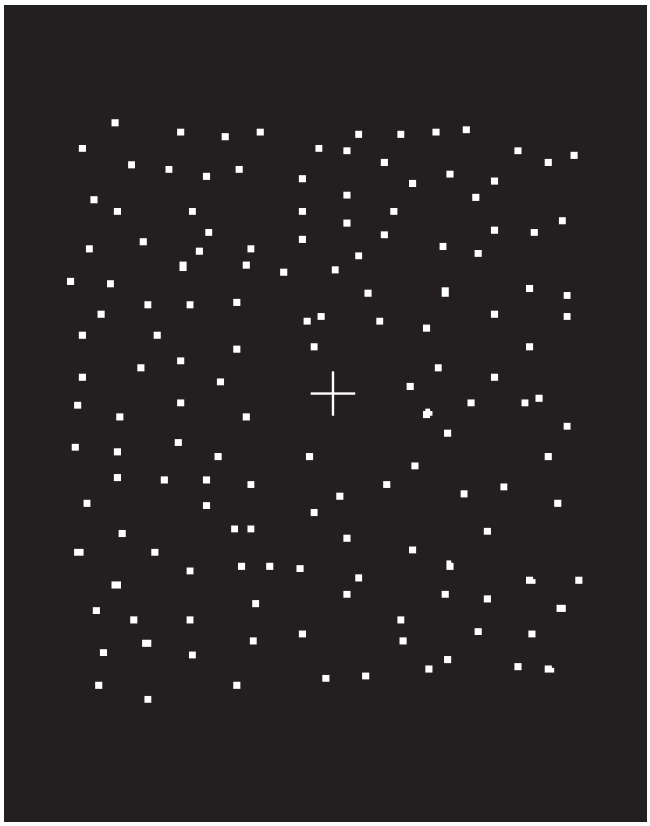

Figure 2 A schematic illustration of the random dot stimulus patch

percentage of coherently moving dots (angular velocity 8.8 deg/sec) within a given software frame (duration = 56.1 ms) was controlled and varied to the participant's detection threshold by a 3-dB-up, 1-dB-down two-alternative forced-choice staircase procedure.

Procedure

Participants were asked to fixate on a white cross that appeared in the centre of the stimulus patch. The fixation cross preceded the appearance of the stimulus patch and remained on the screen for its duration. After 1009.8 ms the stimulus disappeared from the screen and the participant was asked to indicate the direction of coherent motion by pressing one of two keys on the keyboard indicating either left or right (the keys used were the forward slash key (using right hand) for right and the back slash key (using left hand) for left). Every response generated a tone from the computer – a high tone for a correct response and a low tone for an incorrect response. Participants were informed of the significance of these tones so that they could monitor their own performance and to encourage accuracy. Before starting the task each participant was shown a stimulus patch with a high threshold (i.e., motion direction was easily detected); the experimenter explained that the aim of the task was to identify the direction of motion and to respond using the appropriate keys. Participants were also told that the task would become harder, and that it was important that the participant tried their best; however, if they found a particular patch too difficult they were instructed to guess. Each participant also received 10 practice trials before threshold measurement began. Thresholds for detection were computed by taking the geometric average of the last 8 of 10 reversal points within a given series of trials. Each series was repeated three times with the mean of these series comprising the participant's overall motion detection threshold.

Results

An independent samples t-test revealed a significant difference between the mean thresholds of the two groups, $t(28.9) = 2.966$, $p < .01$. The mean threshold for the children with autism was 25.05% ($SD = 15.58$) from a range of 6–64%, and the mean threshold for the typically developing children was 15.34% ($SD = 4.71$) from a range of 6.3–29.4%. The children with autism therefore required, on average, a threshold increase of 9.7% in order to correctly perceive the direction of coherent motion.

To account for the possibility that the children with autism showed higher coherent motion detection thresholds because they were simply not attending to the task and therefore performing at chance, we created a randomisation program which ran through the task 10 million times making random responses to the stimuli. From this we produced a histogram of the possible threshold measurements under the null hypothesis. This resulted in a skewed distribution with a mean threshold of 62.3% and a standard deviation of 7.5%. The data from the study presented here shows that the mean threshold for the group with autism was 25.04%. From our null distribution, we calculated that the probability of scoring <= 25% is .0017, which indicates that, on average, the children with autism, as a group, were responding appropriately to the task and performing well above chance levels. The null distribution also showed that when responding randomly, there was a 1 in 10 chance of obtaining 52.5%. Therefore to eliminate the possibility that any participants in our sample were performing at chance, we recalculated the analysis, after taking out any thresholds above 52.5%. Two participants in the sample had motion coherence thresholds above 52.5% and both of them were children with autism. With these two 'outliers' removed, the mean motion coherence threshold of the autism group was 21.83% with a standard deviation of 11.35 (compared with a mean of 15.34% and a standard deviation of 4.71 in the typically developing group). There was still a significant difference between the mean thresholds of the two groups, $t(29.64) = 2.52$, $p < .05$.[1] Figure 3 shows a graph of the mean coherence thresholds for the two groups with the two outliers removed from the autism sample.

[1]We also performed non-parametric, Mann-Whitney analysis of the data. With the outliers removed the two groups were still significantly different, $z = -2.044$, $p < .05$.

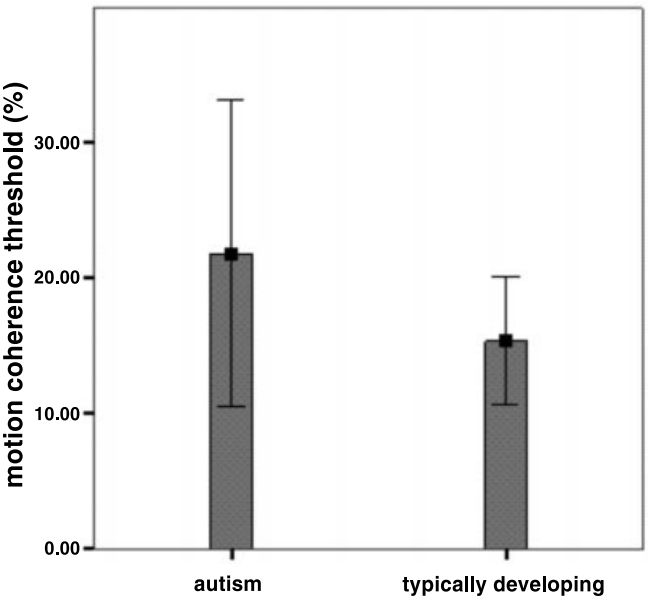

Figure 3 Bar graph illustrating the mean motion coherence thresholds of the two groups; error bars represent one standard deviation

For descriptive purposes, quartiles for the combined sample were calculated (again with the outliers removed) and are presented in Table 2. It can be seen that children with autism are over-represented in the third and fourth quartiles, especially in the fourth quartile where there are only 2 thresholds of typically developing children (9% of the sample). Chi^2 analysis revealed this observation to represent a marginally statistically significant difference, chi^2 (3, $N=45$) = 6.68, $p=.08$. Pearson's correlation coefficients were calculated to assess the relationship between chronological age and non-verbal IQ; for both parameters we found no significant correlation.

Discussion

Using a random dot kinematogram to measure the motion coherence thresholds of a group of 25 children with autism and 22 typically developing children matched for chronological age and non-verbal IQ, we found that the children with autism had significantly higher motion coherence thresholds than the matched controls. Furthermore, Spencer et al. (2000) have also recently reported increased motion coherence thresholds in children with autism when compared with typically developing children matched for verbal-mental age alone. Together, the study by Spencer et al. (2000) and the present study provide strong evidence that children with autism show abnormally high motion coherence thresholds regardless of whether they are developmentally delayed or are functioning at typical IQ levels.

One simple explanation for the performance of children with autism in this study could have been lack of motivation, i.e., they show high thresholds because of a lack of motivation to do the task or a general impairment to perform visual tasks. However, there is no evidence to suggest that these children were any less motivated to perform the task than the children without autism. The mean threshold of the children with autism indicates that they were responding well above the chance level which was calculated from a distributed sample of random responses to the task. Furthermore, the two children who approached a 1 in 10 probability of performing at chance were removed from the sample, the analysis was recalculated and still revealed a significant difference between the mean thresholds of the two groups. Also, all participants had previously completed the Raven's progressive matrices, and had obtained scores within the normal range for their age. Half of the children who performed this experiment also took part in a task which measured their performance on a visual search task in a testing session 1 week later. These children showed the same pattern of performance as the typically developing children, providing a further indication that they were able and motivated to perform visual tasks presented on the computer. In many cases, children with autism are known to show superior performance on visual tasks such as the Embedded Figures Task (Shah & Frith, 1983) and visual search tasks (Plaisted, O'Riordan, & Baron-Cohen, 1998). We therefore think that it is highly unlikely that the high motion coherence thresholds seen in this task could be explained by a lack of motivation or a general inability to perform visual tasks.

The finding is interesting in light of the local processing bias seen in children with autism. Plaisted et al. (1999) have suggested that the local bias seen under some circumstances in children with autism on the Navon task could be attributable to high levels of activity in the high spatial frequency channels. The finding presented in this paper suggests that it is more likely to be a result of low levels of activity in the low spatial frequency channels. The magnocellular pathway, however, is known to inhibit the parvocellular pathway (Singer & Bedworth, 1973), so it remains unclear whether the local processing bias occurs because of impairment and degradation of

Table 2 Percentage of motion coherence thresholds in each quartile for both groups

Range of mct's	1st Quartile (6–12.75%)	2nd Quartile (12.76–16.35%)	3rd Quartile (16.36–20.75%)	4th Quartile (20.76–50.6%)	Total
Autism	17.5%	17.5%	26%	39%	100%
Typically developing	32%	36%	23%	9%	100%

low spatial frequency processing or because an impaired magnocellular system will exert less inhibition on the parvocellular system, resulting in higher levels of activity in that system. Children with autism show a local processing bias on many tasks (e.g., Shah & Frith, 1983, 1993; Happé, 1996; Jarrold & Russell, 1997; Plaisted et al., 1999) and this processing style has become known as 'weak central coherence'. Given that the magnocellular system is known to be sensitive to low spatial frequencies which mediate global processing in the primate visual system, it is possible that the local processing advantage, known as weak central coherence in autism, may arise from the same system which gives rise to the motion processing deficit, namely the magnocellular pathway.

However, it should be noted that recent research on the visual manifestation of weak central coherence in children with autism has indicated that global processing *can* be engaged by people with autism under certain circumstances. For example, in Plaisted et al.'s (1999) study using hierarchical figure stimuli, the children with autism were found to show a bias to the local level if they were given no instruction as to which level to look for the target (divided attention condition), but if they were given prior instruction as to which level to attend to (selective attention condition), the same children with autism showed a global bias. Previously Ozonoff, Strayer, McMahon, and Filloux (1994) had also found that participants with autism showed a global bias on the Navon task, and that there was no difference between the performance of the participants with autism and the controls. Interestingly, Ropar and Mitchell (1999) have shown that, under certain circumstances, children with autism may succumb to visual illusions to the same degree as children without autism. This finding stands as contrary to the earlier finding by Happé (1996) who found that children with autism succumbed less frequently to visual illusions than children without autism. Ropar and Mitchell carried out a study in which, rather than making 'same' or 'different' judgements about illusory aspects of a stimulus, participants were required to manually adjust comparison lines or circles to make them appear the same size. Under these circumstances children with autism were found to be as susceptible to the illusions as the children without autism.

The finding therefore that global processing appears to be under attentional control, and that under certain conditions children with autism show a global processing bias on tasks in which they have previously been shown to have a local processing bias, means that we must be cautious in trying to attribute these findings solely to a low-level perceptual deficit. However, the data of this study and the recent findings (Michimata et al., 1999) indicating that global processing is supported by the magnocellular pathway, should encourage us to further investigate low-level visual processes in autism.

It is possible that a magnocellular impairment in autism may be indicative of abnormalities within the parietal lobe in these children. A reason to suggest this is that the magnocellular pathway projects mainly to the dorsal stream which projects heavily to the parietal regions (Livingston & Hubel, 1988). Although there is cross-talk between the streams, especially beyond the lateral geniculate nucleus (Maunsell, Nealey, & DePriest, 1990), it is largely accepted that the predominant input to the parietal regions comes from the dorsal rather than the ventral streams (Merigan & Maunsell, 1993). This is of particular interest in light of previous research that has identified parietal lobe structures to be abnormal in some individuals with autism. For example, Haas et al. (1996) examined neurologic function in individuals with and without autism, and found that using tests that reflect parietal function, neurological abnormalities were significantly greater for individuals with autism. Further, Saitoh and Courchesne (1998) performed a magnetic resonance imaging study with individuals diagnosed as having autism, the results of which demonstrated abnormalities in the parietal lobe. The parietal lobe is known to be important in mediating attention and in attention shifting (Posner, Walker, Friedrich, & Rafal, 1984). Children with autism are known to be impaired in engaging in joint attention, and the relationship between this and parietal mediated attention shifting deficits has recently been demonstrated using adaptations of a Posner-type cueing task involving faces (Swettenham et al., 2000). It is possible that there could be a causal relationship between a magnocellular deficit and the parietal abnormalities that have been documented in some individuals with autism. One possibility is that an impairment of magnocellular function which occurs early in the visual stream may lead to a paucity of information being processed by the dorsal stream which in turn would lead to degradation of the information reaching the parietal lobe. Because the brain has been shown to develop in response to the input it receives (see Elman et al., 1996), if, early in development, information coming from the magnocellular system is impoverished then the parietal lobe may develop abnormally.

Despite the evidence of abnormally high motion coherence thresholds in the majority of children with autism we tested, it should also be noted that not *all* the children with autism in the sample had elevated motion coherence thresholds. A proportion of the children with autism obtained thresholds of well below average for the typically developing group. This finding and the previous work that has been done with individuals with Williams' syndrome (Atkinson et al., 1997) and children who have dyslexia and reading impairments (Cornelissen et al., 1995, 1998), who also show elevated motion coherence thresholds, indicate that a magnocellular deficit is neither a necessary nor a sufficient correlate of autism. It may be

the case that behavioural tasks such as the random dot kinematogram are particularly sensitive tasks for identifying individual differences across a wide range of developmental disorders. Further work to investigate motion coherence thresholds across large groups of typically developing children, as well as other types of developmental disorders, will help to establish what the significance of high thresholds in these disorders may be. It is also possible that the children with autism in our sample who do not have raised motion coherence thresholds represent a specific subgroup. This might be the case if autism does not turn out to be a unified disorder. There are many studies which suggest that autism is a complex disorder representing a heterogeneous group of individuals (see Gilman & Tuchman, 1995 and VanMeter, Fein, Morris, Waterhouse, & Allen, 1997). If this were the case it would not be surprising that in the present study some of the children with autism have a high motion coherence threshold, and some of the children do not. Interestingly, this pattern of results – where there is a trend for the participants with autism to show significant differences compared to the mean performance of controls despite individuals within the autism group being within the normal range – is also seen in Jarrold and Russell's (1997) study which showed that although overall the participants with autism counted dots individually, 10 out of the 22 children were shown to have counted the canonical stimuli in a global fashion. This leads to the consideration that central coherence may be weaker in some children with autism than others. Given the finding of this study that not all children within our sample who were diagnosed with autism had high motion coherence thresholds, it would be interesting in future to investigate whether there is any correlation between motion coherence thresholds and performance on traditional central coherence tests.

In summary, considering that one of the implications of a deficit in magnocellular processing is a degradation of low spatial frequency visual information, the existence of such an impairment in autism is interesting in terms of the perceptual abnormalities seen in the disorder. The data which has supported the weak central coherence hypothesis highlights the tendency in autism to focus on the local aspects of a stimulus as opposed to the global aspects. Given that the global aspects are known to be carried by low spatial frequency channels, an impairment in magnocellular functioning may offer a physiological explanation for these findings.

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