Mapping cognition onto the brain:

'weak' central coherence and global grouping

Elizabeth Pellicano

University College, University of Oxford and

Department of Psychiatry, University of Oxford

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Perceptual atypicalities associated with autism spectrum disorders (ASDs) are a hot topic in current research. Early reports indicated that basic perceptual processes were intact in autism (Frith & Baron-Cohen, 1987). More recent evidence of reduced sensitivity to global, coherent motion in autism (Milne et al., 2002; Spencer et al., 2000), however, suggests that low-level perception may be impaired after all. Milne, Swettenham, and Campbell present a timely review of these abnormalities in ASD and raise a number of outstanding questions that warrant further research.

In their paper, they recommend the use of additional psychophysical tests in persons with ASD in order to provide a more direct indication of the integrity of the visual pathway largely responsible for processing dynamic information, the magnocellular or dorsal-stream pathway. My colleagues and I (Pellicano, Gibson, Maybery, Durkin, & Badcock, 2005) recently conducted a study that set out to achieve precisely this goal. This commentary reviews the findings of that study, as well as those of several others, in order to offer preliminary answers to some of the questions that Milne et al. pose. It concludes with a discussion of the theoretical implications of motion perception abnormalities in ASD, with particular reference to one current cognitive model of autism, central coherence (CC) theory (Frith, 1989).

Investigating the integrity of the magnocellular pathway in ASD

Our study (Pellicano et al., 2005) examined global motion perception in children with ASD in the context of an investigation of the putative neural mechanism of 'weak' CC. CC theory proposes that typically developing individuals display a tendency to integrate information into context. In autism, this is thought to be disrupted or present in a 'weak' form; accordingly, information processing is characterised by a local bias, resulting in an inability to perceive the global whole (Frith, 1989). Milne et al. (2002) speculated that a generalised deficit in the

magnocellular pathway (which is known to process the global level of a stimulus more rapidly than the local level: Badcock, Whitworth, Badcock, & Lovegrove, 1990) might be the neural correlate for 'weak' CC in autism.

This led us to assess the integrity of functioning at different levels along this pathway, by measuring flicker contrast sensitivity, which tapped early visual processing (area V1), and global motion perception, which tapped processing further along this pathway (area V5/MT). We reasoned that if a dorsal-stream deficit was present in children with ASD, then dynamic information processing mediated by both lower- and higher-level mechanisms should be affected. To assess whether there might be a relationship between global motion perception and CC, the Children's Embedded Figures Test (CEFT) was also administered. On the CEFT, children are asked to identify a shape hidden in a larger meaningful figure. Purportedly, persons with ASD are not captured by the gestalt; this allows them to focus more easily on the individual elements of the figure, thus detecting the hidden shape quickly.

We found that children with ASD were equally sensitive to flicker contrast as their typically developing peers, yet were much worse than comparison children at detecting global motion. Also, children with ASD were significantly faster to identify the figures hidden on the CEFT. Furthermore, we found that higher global motion thresholds were associated with faster performance on the CEFT in the ASD group.

What might explain poor global motion perception in ASD?

Milne et al. consider two competing explanations for the motion perception abnormalities in ASD. The first of these, which they favour, relates to whether the elevated global motion thresholds might be a result of a generalised impairment in the magnocellular pathway. This is consistent with the hypothesis of a pervasive dorsal-stream deficit thought to be present in a variety of neurodevelopmental disorders

(Braddick, Atkinson, & Wattam-Bell, 2003). The second proposal relates to a more specific impairment in extrastriate cortical regions (Bertone, Mottron, Jelenic, & Faubert, 2003). These authors suggest that motion perception abnormalities in autism are present only when stimuli are sufficiently 'complex' that additional neural computation is required (i.e., the integration of information).

Our finding (Pellicano et al., 2005) of normal sensitivity to flicker contrast offers direct evidence of intact lower-level magnocellular pathway functioning in ASD. This finding has since been replicated by Bertone, Mottron, Jelenic, and Faubert (in press). Also, Hadjikhani et al. (2004) reported no abnormalities when examining the retinotopic organization of early visual cortex in adults with ASD, corroborating findings from studies using psychophysical tasks. Together, these findings fail to support the proposal of a pervasive abnormality in magnocellular functioning in persons with ASD.

Nonetheless, consistent with previous reports (Milne et al., 2002; Spencer et al., 2000), we did find evidence of poor global motion perception in children with ASD. This suggests that motion abnormalities might be confined to higher levels in the cortical regions (V5/MT) receiving predominant input from the magnocellular pathway. It is in this area that the various motion signals extracted in area V1 are pooled or grouped together over both space and time to yield a percept of global, coherent motion.

We also report a significant inverse association between performance on the global motion task and performance on the CEFT in the ASD group. The link between these tasks is suggestive of the fact that abnormalities in 'global grouping' might be responsible for producing weak CC in autism, at least at the visuospatial level. If global grouping is disrupted in ASD, then the gestalt formed by the larger

figure should not be compelling and should not impede the search for the hidden shape, resulting in faster CEFT performance, as was evidenced. Thus, consistent with the alternative position reviewed by Milne et al., we suggest that there might be a general extrastriate deficit in ASD, which affects global grouping in both dynamic (e.g., detection of global motion) and static (e.g., detection of hidden figures) stimuli.

This is similar to the 'complexity' hypothesis put forth by Bertone et al. (2003). Both arguments, however, rest upon the assumption that an analogous deficit is demonstrated at higher levels in the ventral stream to the deficit found in the dorsal stream in ASD. As presented by Milne et al., Spencer et al. (2000) and Blake et al. (2003) reported intact global form perception in children with autism, which challenges the notion of a global grouping abnormality spanning dorsal and ventral streams.

Absent from Milne et al.'s review, however, is a discussion of the argument that global form detection (particularly in the tasks used by Spencer et al. and Blake et al.) might not occur in extrastriate ventral areas, but instead could be processed earlier in the visual system. Field and Hayes (2004) review work showing that V1 neurons, which have long-range lateral connections, can make use of information beyond the neuron's receptive field. In this way, detection of contour can be accomplished in V1 by the integration of information from individual line segments, when the angular deviation between line segments is small.

Interestingly, there have been two recent reports of poor global form perception in autism, both of which have used stimuli that do recruit higher-level ventral-stream processing. Spencer and O'Brien (in press) presented individuals with autism with Glass stimuli, which consist of a static display of randomly distributed dot triplets (rather than line segments), some of which are aligned concentrically,

while others are presented randomly. Children with autism were worse at detecting coherent form than comparison children. Similarly, Bertone et al. (in press) tested lower- and higher-level functioning in the parvocellular, or ventral-stream pathway. They found evidence of intact processing of first-order (luminance-defined) gratings (tapping lower-level parvocellular functioning) but impaired perception of second-order (texture-defined) gratings (tapping higher-level parvocellular functioning). The higher-level parvocellular tasks used by both research groups are very similar in nature to the higher-level magnocellular task (global motion detection) – they all require cooperative cortical mechanisms to integrate local signals into a global percept.

Thus, the aetiology of the elevated global motion thresholds does not seem to be a result of a general impairment in the magnocellular pathway. Rather, the converging evidence suggests that perceptual abnormalities in ASD are present at higher levels in the visual system, and occur when global grouping is required. Importantly, these perceptual abnormalities in ASD do not seem to be restricted to motion per se, as Milne et al.'s review suggests, but also extend to the perception of form.

Individual differences in global motion perception

Milne et al. raise an important issue regarding individual differences in motion perception difficulties in ASD. In previous papers, Milne et al. (2002, in press) noted the wide variation in threshold estimates on the global motion task in their sample of adolescents with autism, with a number of children falling within the range of performance of the typically developing group. Milne et al. question whether only one subgroup of children with ASD is affected by motion perception abnormalities.

We too found large variation in global motion thresholds in the ASD group relative to the comparison group (Pellicano et al., 2005). Despite this wide range of scores, 80% of children with ASD obtained global motion thresholds that fell outside the 95% confidence intervals of the typically developing group. In contrast to Milne et al., our findings suggest that a substantial proportion of the ASD group displayed global motion perception abnormalities, rather than a subgroup alone.

Milne et al. further question whether individual differences in global motion perception might be associated with autistic symptomatology. To address this, correlational analyses were performed on the Pellicano et al. (2005) sample (unpublished data) between global motion thresholds and total symptom scores on two instruments: the Autism-Diagnostic Interview – Revised[1] (Lord, Rutter, & Le Couteur, 1994) and the Social Communication Questionnaire (Rutter, Bailey, & Lord, 2003). Such analyses yielded no significant associations between these variables, suggesting that symptom severity (as rated by the parent) is unrelated to global motion perception in this sample of children with ASD. These null findings conflict with those of Blake et al. (2003), who report a significant link between symptomatology scores and poor perception of biological motion. The discrepancy between these findings might relate to the types of instruments used to index autistic symptomatology (i.e., parent-rated vs. observational). As Milne et al. recommend, potential links between global motion perception abnormalities and autistic symptomatology deserve further attention.

Theoretical implications: weak CC and global grouping

Milne et al. propose that poor global motion perception might be mediated by an abnormality in processing low spatial frequency information (to which the magnocellular pathway is most sensitive), which in turn could explain various

cognitive phenomena related to local-global processing in ASD. This is unlikely to be the case for two reasons. First, evidence suggests that global motion perception is not dependent on low spatial frequency information (Smith, Snowden, & Milne, 1994). Second, the studies reviewed above demonstrate that abnormalities in ASD extend to global form perception, processed by the pathway responsible for carrying high spatial frequency information, the parvocellular pathway.

Our study represented an initial attempt to understand the brain basis of weak CC in ASD (Pellicano et al., 2005). CC theory maintains that there is a reciprocal relationship between local and global processing; that individuals with autism will perform well on tasks that benefit from a local bias, but poor on tasks necessitating the integration of information (Frith, 1989). The finding of a link between elevated global motion thresholds and CEFT performance in children with ASD is consistent with predictions made by CC theory. We speculate that global grouping abnormalities that mediate the reduced sensitivity to global motion might also force individuals to adopt a local processing strategy on the CEFT; thus, the putative global grouping abnormality might be the neural mechanism for 'weak' CC in ASD (Pellicano et al., 2005).

This possibility prompts a whole host of new questions for empirical and theoretical consideration. Notably, these questions move away from those offered by Milne et al., as the effects of poor global grouping in ASD are not restricted to motion perception. One question relates to whether weak CC/poor global grouping can account for other types of motion perception. As reviewed by Milne et al., Blake et al. (2003) found that children with autism were significantly impaired at detecting human activity. In their task, local motion signals (the individual dots on point-light displays) need to be integrated to yield a global representation of human movement. It is easy to

see how deficits in global grouping/weak CC could make it difficult to perceive biological motion. Future research will need to ascertain whether different types of motion perception are disrupted in ASD due to their reliance on faulty global grouping mechanisms.

A second question concerns whether abnormalities in higher-level global grouping processes could explain other phenomena attributed to weak CC. Superior performance has been demonstrated on another test of CC, the Block Design task; it will be important to determine whether global grouping abnormalities might also mediate performance on this task. Weak CC in ASD also manifests at higher conceptual levels, such as in the limited use of context in a homograph task. It is unclear how putative abnormalities in higher-level global grouping are applicable to information processing beyond the visuospatial level. Indeed, such higher-level conceptual tasks rely heavily on children's language abilities (cf. Norbury, 2005) and so may be processed principally by areas dedicated to language, rather than higher-level visual areas.

A third question relates to the extent to which weak CC/poor global grouping might affect other cognitive capabilities/deficits in ASD, particularly sociocognitive difficulties, which are held to be responsible for the profound deficits in socialisation and communication in ASD. CC and theory of mind abilities have been found to be unrelated in preschool children with ASD (Pellicano et al., in press); however, poor global grouping/weak CC could have a significant effect on inputs necessary for the development of a theory of mind. Skills affected by poor global grouping could include those involved in face processing, which entails the integration of information into context. Certainly, future longitudinal studies may be useful in elucidating the

causal relationships between global grouping/weak CC and sociocognitive functioning in ASD.

There is one final caveat to consider. The preceding discussion assumes some biological basis for weak CC; it might be the case, however, that CC represents a more malleable bias under some strategic control. Several studies have shown that prior cueing to the global level can elicit intact global processing by persons with autism on perceptual tasks (Plaisted et al., 1999), which is more suggestive of the operation of top-down mechanisms. One basis for an alternative position would be to reverse the presumed causality, and ask whether weak CC is the pervasive problem and poor global motion perception just one of its consequences. The motion task we used requires explicit decision making, and this could be vulnerable to biases in the way information is processed.

Whatever the causal relationship may be, CC theory has proved fruitful in this instance. The generation of explicit predictions regarding global-local processing has resulted in preliminary specification of the neural correlates for the atypical perceptual and cognitive processing in ASD. The role of cognitive theories featured little in Milne et al.'s review; clearly, however, this discussion highlights the utility of such theories in understanding better the underlying basis of autism.

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Footnotes

1. The algorithm for the ADI-R was re-run using ‘current’ scores to ensure that total scores reflected current autistic symptomatology. These scores were then used in correlational analyses with global motion threshold estimates.