Motion perception and autistic spectrum disorder: A reply to the commentaries.

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We sincerely thank all the commentators for their stimulating and insightful comments, and acknowledge the generosity of many researchers in sharing their new and exciting data and ideas. We are encouraged by the multi-disciplined approach to the study of perception in autism, which, we feel clearly strengthens this area of research. Our aim in this reply is to summarise the key points and to outline some working conclusions about motion perception in autism. It is hoped that this issue of CPC can then stand as a foundation from which existing theories can be refined and developed, and the strengths and weaknesses of currently available methodology documented, so that future work in this area moves forward within a well-informed, coherent framework. This reply will be structured under eight sub-headings:

- Methodological concerns from previous studies
- Is there really a motion perception impairment in autism?
- Aetiological interpretations of the data
- Universality / specificity of abnormal perception in autism
- Relationship to behaviour, symptoms and IQ
- Insights from development
- Relationship to reduced global bias
- Concluding remarks

**Methodological concerns from previous studies**

A number of the reviews raised concerns about methodologies used in the study of motion perception in autism. One of the challenges in this field of research is to design a task which allows one to isolate perception of motion from other perceptual processes (such as perceptual integration) and from attentional processes. One possible way to meet this challenge will be to design appropriate control tasks.

**Coherent motion perception**

In studies of coherent motion perception, a technique which has been used to separate ability to detect coherent motion from ability to integrate local elements, is to use a static form detection task as a control. The logic is that if form detection is intact, then poor performance on a motion coherence task must be due to motion perception alone. However, for the form detection task to be a meaningful control it must be equivalent to the motion detection task in every way apart from motion. A number of commentaries point out that it is not. Abreu & Happé argue that the attentional
demands of form detection are less than the attentional demands of motion perception. They highlight, along with Mitchell et al., that coherent form detection involves the integration of elements across space whereas coherent motion detection involves the integration of elements across both space and time, which renders the tasks non-equivalent.

Atkinson & Braddick discuss a novel experimental design which aims to refine the comparability of form and motion detection at a stimulus level by presenting geometrically identical motion and form displays (see their commentary in this issue). While these modifications to existing methodology are advantageous, they do not directly address the criticism raised by Bertone et al. and Kogan & Chaudhrai that motion and form perception are not comparable in terms of their neural computation. Bertone et al. point out that perception of coherent motion requires extrastriate processing, and may therefore be deemed ‘complex’, while the perception of basic visual form contours from individual line elements may be achieved by orientation selective mechanisms operating at the level of V1. The conjecture from Bertone et al. and Kogan & Chaudhrai is that the difference between the two tasks is therefore not just that one involves motion and the other does not, but that one is more complex at a neural level. (The issue of stimulus complexity is also raised in relation to first and second order motion as is discussed below.)

As a further methodological caveat, Mitchell et al. point out that the measurement of sensory thresholds using adaptive staircase procedures can vary depending on many stimulus factors, ranging from dot density to stimulus exposure duration.

First and second order motion
Bertone et al. (2003) report that individuals with autism show impairment in processing second- but not first-order motion. The exact definition of first and second order motion however, varies amongst researchers. Some claim that first and second order motion are differentiated by stimulus characteristics alone (Mather & Murdoch, 1997), while others differentiate first and second order stimuli by the level at which they are processed along the cortical visual pathways (Kogan & Chaudhuri). Mitchell et al. report that relatively little is known about the neural basis of second order motion processing, and that there is little evidence to suggest that these two classes of motion stimuli are processed by distinct neural areas. Kogan & Chaudhuri on the
other hand claim that first order information is initially analysed in area V1 and that additional pre-processing of second order stimuli occurs in extrastriate cortex. In addition, they discuss recent imaging data that suggests first order motion is processed in area V1 whereas second-order motion is processed in the parietal lobe (Dumoulin, Baker, Hess, & Evans, 2003). It seems that before a distinction between first and second order motion can provide a useful framework for understanding deficits in autism, a clearer understanding of the neuronal and computational mechanisms involved in processing different types of motion stimuli is needed.

In line with their argument about the neural underpinnings of motion and form perception, both Bertone et al. and Kogan & Chaudhuri argue that second order stimuli are more complex than first order stimuli, and it is this increased complexity that gives rise to impaired performance in autism. Jarrold and Scott-Samuel raise the question of exactly what is meant by complexity in this sense. At least two different meanings are suggested by the commentators. For Bertone et al., complexity in the motion perception task is evident in ‘additional neuro-integrative’ processing, compared with ‘simple’ striate computation of primary visual features. By contrast, Burak et al. highlight task difficulty as an index of complexity. Aside from theories of neuronal integration, some tasks may not be sensitive enough to elicit atypical performance in individuals with autism. Therefore complexity in the sense of task difficulty may also explain why participants with autism show impairment of second but not first order motion perception.

Abnormal postural control in response to optic flow.

Motion perception in autism has also been investigated, albeit indirectly in tasks which do not require overt behavioural responses such as measuring postural control (Gepner), or smooth pursuit eye-movement (Takarae et al.) These types of tasks can be especially valuable when working with populations such as autism where it can be difficult to isolate task performance on alternative forced choice tasks from other attentional and / or motivational components. One of the techniques used has been to measure postural stability in response optic flow (Gepner and Mestre, 2002). At first sight, results from this task appear to support the hypothesis of impaired motion perception in autism. However, Jarrold & Scott-Samuel point out that in the data from this study, there was no interaction between postural sway when the children with autism had their eyes closed, when they had their eyes open in the absence of moving
stimuli, or when they were looking at optic flow. This suggests that increased postural stability in autism is not integral to decreased perception of motion. In support of this, Takarae et al. have highlighted additional data which indicates that children with autism do not show impairment of stabilising posture from visual input alone, but rather show a deficit of integrating multi-sensory integration (Minshew, Sung, Jones, & Furman, 2004). The opinion from the commentaries therefore is that work on postural instability in autism does not provide direct evidence for impaired motion perception per se, as it measures a complex range of sensory-motor factors of which motion perception is only a small part.

Is there really a motion perception impairment in autism?

Jarrold and Scott-Samuel caution that data currently available do not provide unequivocal support for the claim that individuals with autism are impaired in motion perception. The methodological concerns highlighted above account for some of their concern, and we agree that further work needs to be done to strengthen the case. However, along with some of our own more recent data, there are new reports documenting further examples of impaired motion perception in autism. For example, Happé and Abrue have found that when using stimuli that measure perception of both smooth pursuit, and coherent motion, every child with autism in their experimental cohort showed a deficit in at least one of the tasks. However, they do not mention control, non-dynamic tasks used with this sample of children, so we cannot confirm whether or not the children with autism in this sample were selectively impaired at motion detection, or whether they may also have shown deficits in other perceptual tasks.

There appears to be ample evidence for atypical visual perception at some level in autism, but it may not be restricted to motion perception alone. Aside from the reviewed evidence of impaired motion perception, the commentaries in this issue report evidence of reduced accuracy of smooth pursuit eye movements (Takarae et al.), unusual spatial frequency modulated responses to face perception (Rondan & Deruelle), and increased discrimination thresholds to a steady-state pedestal task tapping magnocellular function (Plaisted & Davis). Additionally, recently published data from Bertone, Mottron, Jelenic, & Faubert, (2005) and Spencer & O’Brien, in press (as discussed by Pellicano) indicate that with thoughtfully designed stimuli
which test integrated, high level form perception, individuals with autism also show an impairment in form detection. These new findings challenge the position that form perception is intact and that there is a *selective* deficit of motion perception in autism.

Clearly, a number of relatively low level perceptual processes can be shown to be impaired in autism. While these include coherent motion detection, abnormalities are not confined to this task, and indeed some aspects of low level motion perception may be spared in autism (Bertone et al., 2003). The challenge for researchers is to establish exactly which aspects of perception are impaired in autism and to establish the exact stimulus conditions under which abnormal responses are or are not present.

**Aetiological interpretations of the data.**

Baron-Cohen questions whether atypical motion perception in autism can be interpreted as a different style of perceptual processing as opposed to impairment arising from damage or breakdown of a particular mechanism. Conceiving atypical perception as a different style may well be appropriate, and it echoes previous interpretation of the weak central coherence literature (Happé, 1999). However it is important for autism research that the *origin* of different perceptual (or cognitive) styles is investigated. We see this position as a starting point for developing and testing hypotheses and not as a conclusion.

In light of this, a number of hypotheses regarding the aetiology of atypical perception of motion in autism have been documented in this issue. These include: reduced sensory integration (Takaarae et al.; Gepner et al.); reduced neuronal integration (Bertone et al.; Kogan & Chaudhuri; Mottron & Dawson); attentional dysfunction (Belmonte; Abreu & Happé); irregular neuronal connectivity (Belmonte; Mottron & Dawson; Pavlova); dorsal stream deficiencies (Pellicano; Atkinson & Braddick) and magnocellular abnormality (Milne et al.; Plaisted & Davis). These interpretations are still evolving and as Belmonte points out, need not be mutually exclusive. It is quite possible that a number, if not all, of the above systems show some degree of abnormality in autism, and that they may exert varying contributions to atypical perception in autism. It is also possible that the neuronal signatures cited above are related, either by way of brain development or by way of interpretation of task performance. For instance, a congenital or early occurring disorder in neuronal connectivity could give rise to impaired sensory integration, or, as another example,
atypicalities in the development of the magnocellular system could lead to abnormal development of cortical sites which underpin a range of cognitive functions. A further example suggested by Abreu & Happé, is that a deficit of selective attention may reduce the ability to integrate local motion signals of space and time, but because of the nature of the task involved this deficit is interpreted as an impairment in motion perception rather than as an impairment of selective attention. A key challenge will be to isolate the integrity of processes which could underlie impaired motion perception in autism.

As Belmonte points out, interpretation of the data to date has been largely dichotomous: explanations generally divide into what Bertone et al. define as the “pathway-specific hypothesis” and the “complexity specific hypothesis”. The pathway-specific hypothesis interprets the data as indicating impairment in a particular area of brain, either at a sub-cortical (magnocellular) or cortical (dorsal stream) level. The complexity specific hypothesis is based on recent findings which demonstrate abnormality in aspects of perception supported by the ventral visual stream, and postulates that the degree of complexity of the stimulus, regardless of the specific brain areas that are optimally sensitive to it, determines whether or not a deficit is observed. Although the recent data demonstrating impaired complex form detection in autism provide support for the complexity specific hypothesis, the degree to which the magnocellular / dorsal pathway is impaired in autism is far from being resolved. To date, the main evidence for intact magnocellular processing comes from a failure to find a difference between normally developing and autistic people on a flicker fusion task designed to stimulate the magnocellular stream exclusively (Bertone et al., 2005; Pellicano, Gibson, Maybery, Durkin, & Badcock, 2005). However, it is axiomatic within psychology that a failure to find a group difference on a task should not be construed as conclusive evidence that the posited function is normal. The pulsed pedestal task (Pokorny & Smith, 1997) is established within visual psychophysics as providing reliable and distinctive signatures of the integrity of magnocellular and parvocellular streams respectively. Using this task, Plaisted & Davis report striking abnormalities in the magnocellular signature of children with autism, which, if supported, suggests the magnocellular system may not function normally in autism.
The visual magnocellular system is one of two main retinocortical streams to the primary visual cortex. Degraded input from this system is therefore likely to affect many aspects of visual perception. Because of the neuroendocrinology and the developmental trajectory of the visual magnocellular system, (see Annaz & Karmiloff-Smith, Rivera & Koldewyn and Atkinson & Braddick) it is a potentially vulnerable target in developmental disorders, and it is possible that an early impairment within its sub-cortical parts could lead to impairments in a range of cortical circuits, since cortical organisation develops as a direct consequence of the nature of inputs received from sub-cortical areas. Further, given the degree of cross-talk between magnocellular / parvocellular pathways, or dorsal / ventral streams, it is likely that disruption of the magnocellular pathway could give rise to wide-spread abnormalities throughout the cortex, resulting in a variety of perceptual abnormalities.

Yet a further possibility, which has been propounded by Ramus (the non specific marker model), envisages that the series of neural events precipitating magnocellular disruption works in completely the other direction. He suggests that the primary cause of the deficit may be abnormalities of neural cortical migration in early childhood. One consequence of this could be that the normal pathways of the magnocellular system, both cortical and subcortical - throughout the thalamus and cerebellum - are disrupted.

Another plausible neuronal correlate of low-level visual atypicalities in autism is abnormal neuronal connectivity. Neuroimaging studies show one cortical region – the (posterior) superior temporal sulcus - is especially sensitive to biological motion. This region is highly interconnected with parietal and frontal regions, and has been proposed as an important integration site for the processing of visual form and visual motion (see Puce & Perrett, 2003). Pavlova discusses interesting data which suggest that individuals with bilateral periventricular leukomalacia (which is characterised by gliosis in white matter and impinges on the pathways connecting sub-cortical structures with cortical regions), show impairments in the perception of biological motion. That is, damage to the white matter connections to and from pSTS may have the same functional effect as damage to the critical cortical region where those connections converge. In line with this, several commentators have mentioned evidence of increased white matter volume in autism (Mottron & Dawson). This relates to existing hypotheses regarding over-connectivity / lack of neuronal pruning.
between local brain subs-systems, and under-connectivity between global brain sub-
systems (Belmonte). These observations conform to the views expressed in
Belmonte’s commentary, that “autism at a basic level is not localised to particular
anatomical structures or functional systems, but must instead arise as an abnormality
of fundamental neural information processing”. For Ramus, too, the appeal is to
generalised systemic neural function, albeit as a function of (multiple) localised
cortical disturbances.

Universality / specificity of abnormal perception in autism
It is clear, both from existing literature and from the commentaries published here that
atypical perception of motion is not specific to autism. Our target article reports
many studies which document atypical perception of a similar nature to that observed
in autism, in dyslexia, SLI, and Williams’ syndrome. We were careful to point out
that in our own data we have found that only a proportion (or a sub-group) of children
with autistic spectrum disorders are impaired at detecting coherent motion, which
suggests that impaired motion detection is neither specific nor universal in autism.
However, Abreu & Happé report that all the children with autism in their sample
showed impairment in at least one type of motion perception. Additionally, in light of
the commentaries which indicate that specific stimulus characteristics affect task
performance, it may be that an interaction between the characteristics of the stimuli
and the aetiology of the individual determines whether or not an impairment is seen.

There will be important lessons to learn from properly documenting which
participants show deficits under which conditions. For example, Abreu & Happé point
out that there were differential patterns of performance in their data – some children
showed impairments on one motion task but not the other, and other children showed
impairments on both. Bertone et al. illustrate that individuals with autism show
deficits in detecting complex motion (Bertone et al. 2003) and also deficits in
detecting complex form (Bertone et al. 2005), but it would be useful to know if all the
individuals tested showed these impairments, and, whether or not the same
individuals showed impairments in both tasks. We agree with Charman et al. who
remind us that “the field of neuropsychology in autism needs to get ambitious”, and
echo the suggestions of Rivera & Koldewyn who highlight the need for the same
participants to be tested on the different tasks. It will be important for future studies
that authors include data regarding individual performance on experimental variables and not just mean group differences.

On the issue of specificity of abnormal motion perception in autism, Kogan & Chaudhuri report that individuals with Fragile X are impaired at the simple motion detection tasks that Bertone et al. report are intact in autism. Collaborative studies such as these, where methodologies are shared between research groups, seem to be a constructive way to develop conclusions about low-level perception in autism as well as in other developmental disorders. Just such a programme has enabled Ramus to develop theoretical insights that go beyond the modular, ‘one neural pathway for one deficit’ notions that characterised earlier approaches to developmental disorder, and suggests that diverse developmental disorders can manifest common, as well as distinctive symptoms. These may eventually provide powerful explanatory models for different developmental outcomes, especially where, as Ramus suggests, the target syndrome has many heterogenous features, and requires multifactorial modelling.

Relationship to behaviour, symptoms and IQ

Behaviour and symptoms

There are mixed views amongst the commentaries regarding the relationship between perceptual abnormality and symptomatology. Some authors favour the possibility of a common relationship between psychophysical data and standardised measures of symptomatology. However, the evidence is scant. Pellicano discusses the lack of any such relationship in her data between global motion perception and total symptom scores on either the ADI-R or the SCQ. In our work, too, no significant relationship was found between global motion detection and total symptom score, communication domain, reciprocal social interaction domain, or restricted / stereotyped patterns of behaviour domain of the SCQ (N = 23, all r between .02 and .12, all p > .1, Milne, 2004). A lack of significant relationship between autism severity and reduced motion perception reinforces the point made by Ramus who argues that if abnormal motion perception is a risk factor for autism then one should expect to observe a correlation between symptom severity and motion perception. Data reviewed in both the target article and the commentaries published here do not suggest that impaired motion perception is a causal risk factor for autism, or indeed any developmental disorder. Once again, though, we must be wary of overinterpreting a null result. As Charman et
al. and Burak et al. point out, poor association between cognitive tasks and symptom severity scores may reflect the “somewhat gross characterisation of the behavioural phenotype provided by diagnostic instruments” (Charman et al.).

**Perceptual abnormalities and IQ**

Another point raised in many of the commentaries is the relationship between IQ and perceptual abnormality. Kogan & Chaudhuri, and Jarrold & Scott-Samuel both make the important point that the majority of children with autism also have learning difficulties (IQ less than 70) but that the majority of published research on children with autism only includes so-called high functioning individuals, with IQ in the normal range. They suggest that limiting inclusion criteria to those children with normal IQ may reduce the potential of finding group differences. While we agree that it is important to include a representative sample of children with autism wherever possible, available evidence on motion perception and IQ seems to suggest that IQ alone doesn’t account for motion perception impairments. Abreu & Happé report that from a sample of 17 children with learning difficulties but without autism (with IQs ranging from 41 – 100) all performed at the same level as typically developing children on motion detection tasks. In contrast, from our own data, children with autism we have tested who stand out as having motion coherence thresholds that are higher than 1.65 standard deviations above the control mean (which corresponds to occurring in the poorest performing 5% tale of the distribution) include some children who have above average non-verbal IQ (e.g. between 102 and 110). Conversely, we have identified children with autism who have low non-verbal IQs (< 75) who show no impairment in detecting coherent motion.

**Insights from development**

The importance of understanding the developmental trajectories of perceptual skills has been raised by a number of commentators. This is both in terms of understanding the developmental trajectory of disorder, and in terms of understanding the developmental trajectory of aspects of typically developing cognition and perception, Rivera & Koldewyn highlight that it is important to establish the age at which perceptual abnormalities are first identified in autism as this will help to understand what relationship, if any, these deficits have to development of autistic symptoms, or the developmental cognitive profile in autism. Jarrold & Scott-Samuel point out that
the ability to process second-order motion stimuli develops more slowly than the ability to process first-order motion stimuli. Atkinson & Braddick state that global form processing develops more slowly than global motion processing, although, the visual magnocellular system appears to reach full maturation later than the parvocellular system (Annan & Karmiloff-Smith).

To date, we have relatively meagre data on normal developmental progression of motion perception. Until this has been acquired, we will not be able to explore the extent to which the pattern observed in autism reflects a developmental delay (retardation) or a more idiosyncratic anomaly. The same considerations apply to the relationships between different task performances. Clearly, detailed developmental data from reasonably large populations of normally developing children is required in order to clarify when, how and why different developmental syndromes emerge and their relationship to specific perceptual functions.

**Relationship to reduced global bias**

In the target article we presented the hypothesis that abnormality of the magnocellular system in autism could lead to both impaired motion detection and reduced global bias on perceptual tasks (weak central coherence). Recent data from Pellicano et al. (2005) provide an intriguing development of this position. They report a relationship between coherent motion detection and performance on the embedded figures task in children with autism such that those children who had elevated motion thresholds were quicker to identify the embedded figures. At first sight, this would appear to support the hypothesis, as it mirrors the finding that children with autism who are more locally biased in identifying hierarchical figures also have elevated coherent motion thresholds. However, as discussed previously, Pellicano et al. also administered a flicker contrast sensitivity task (which was designed to target magnocellular responses selectively) which did not elicit any impairment in the children with autism. Further, there was no significant relationship between the flicker contrast task and performance on the embedded figures task, suggesting that magnocellular function, when indexed at this level, is not a correlate of perceptual style. However, Plaisted & Davis suggest that the size and flicker rate of the stimulus used by Pellicano et al. may have inhibited magnocellular responses rather than elicited them. One problem with both coherent motion detection and flicker contrast
sensitivity tasks is that they can at best provide a putative, indirect measure of the integrity of sub-cortical or even cortical streams. However, the fact remains that relationships have been documented between coherent motion perception and cognitive tasks that are traditionally used to demonstrate weak central coherence in autism. The basis of this relationship remains to be established.

**Concluding Remarks**

Mitchell et al. question the relevance of studying atypical visual perception in autism, as the debilitating symptoms of the condition are in social behaviour and communication rather than perception. This is all the more pertinent in view of the lack of clear conclusion regarding the universality of atypical visual perception in autism. However the range of commentaries presented in this issue suggest that the area has the potential to be informative about heterogeneity in autism and other developmental disorders, and the aetiology of autism and its development.

Before conclusions can be drawn on these issues it is essential to clearly define the nature of the behavioural disorder with respect to the perceptual task. It remains to be established exactly which stimulus characteristics elicit impairments in individuals with autism; to what degree these impairments arise from perceptual or attentional deficits; and whether all individuals with autism display the impairments or if reliable dissociations are present. Clearly there is much work to do in this field, but the rapid development of techniques and theoretical positions provides an excellent framework within which to refine and develop these important hypotheses.

**References**


Reply to the commentaries


