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COMMENTARY ON "MOTION PERCEPTION IN AUTISM"
(E. MILNE, J. SWETTENHAM, & R. CAMPBELL)

Dissociating pathway- versus complexity-specific accounts of motion perception impairments in autism

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In their target article, Milne, Swettenham, and Campbell present a comprehensive review of evidence suggesting that motion perception in autism is abnormal, and how such altered perception may play a role in characteristic autistic symptomology. Their timely review reflects the growing interest regarding low-level information processing in autism, such as motion and form perception (Belmonte et al., 2004). Central to their review is the etiology underlying impaired motion perception in autism. As delineated by Milne et al., there seems to be two main hypotheses regarding the « abnormal neuronal architecture » responsible for the decreased sensitivity to different types of motion stimuli in autism; (1) a

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dorsal visual pathway deficit (i.e., Spencer, O'Brien, Riggs, Braddick, Atkinson, & Wattam-Bell, 2000; Milne, Swettenham, Hansen, Campbell, Jeffries, & Plaisted, 2002) or (2) a neuro-integrative dysfunction at a perceptual level (Bertone, Mottron, Jelenic, & Faubert, 2003). According to Milne et al. (target article), the dorsal/magnocellular pathway deficit (referred to us as the *pathway-specific hypothesis*) suggests that inefficient dorsal visual stream processing and/or localized impairments of motion-sensitive mechanisms operating in extra-striate areas within the dorsal visual pathway (i.e., MT) results in impaired motion perception (Spencer et al., 2000; Milne et al., 2002; Blake, Turner, Smoski, Pozdol, & Stine, 2003). This interpretation has been supported by findings of decreased autistic sensitivity to different types of motion stimuli but intact sensitivity to static or coherent form stimuli believed to be processed by mechanisms operating within the ventral visual stream (Spencer et al., 2000; Blake et al., 2003). Conversely, Bertone et al. (2003) dispute the *pathway-specific hypothesis* given their findings of intact autistic sensitivity to simple, first-order motion but reduced sensitivity to complex, second-order motion. They argue that if deficient dorsal stream function is the origin of abnormal motion perception in autism, both simple *and* complex motion sensitivity should be affected in autism, regardless of its complexity. For this reason, Bertone et al. (2003) interpreted their findings of decreased complex motion sensitivity to be the result of diffuse or non-specific neural dysfunction of neuro-integrative mechanisms affecting complex perceptual processing in general, and not the result of a dorsal stream dysfunction selectively affecting motion-sensitive areas responsible for complex motion perception *per se*. This interpretation, which we obviously favor, is referred to us as the *complexity-specific hypothesis*. Support for this hypothesis will be argued in the context of Milne et al. review.

Impaired dorsal stream functioning in autism?

Milne et al. present evidence of impaired autistic sensitivity to different classes of motion stimuli including coherent (or global), (Spencer et al., 2000; Milne et al, 2002; see also Pellicano, Gibson, Maybery, Durkin, & Badcock, 2005), biological (Blake et al., 2003) and second-order motion stimuli (Bertone et al., 2003). It is to note that all these motion types can be considered to be "complex" in that the mechanisms responsible for

their processing are located in extra-striate areas within the dorsal visual stream and necessitate additional neuro-integrative processing to be perceived (i.e., cannot be resolved by standard motion analysis within the striate cortex). However, there is growing evidence that *early* dorsal stream functioning in autism is actually intact. In addition to Bertone et al. (2003) demonstration of unaffected simple, first-order motion processing, magnocellular functioning in autism has recently been demonstrated to be normal in autism. Specifically, both Pellicano et al. (2005) and Bertone, Mottron, Jelenic, and Faubert (2005) have recently demonstrated that autistic sensitivity to flickering stimuli that preferentially assessed magnocellular functioning to be unaffected. These findings reflect intact lower-level (thalamic and/or striate) dorsal stream functioning in autism and are interpreted as direct evidence against a generalized "dorsal stream deficit". Taken together, these results are congruent with the *complexity-specific hypothesis*, since deficits in motion processing in autism are only found at higher (i.e., processed by extra-striate mechanisms) but not lower levels of analysis (i.e., thalamic or striate) along the dorsal visual stream, probably the result of neuro-integrative dysfunction. These findings also demonstrate that decreased sensitivity to global motion in autism is not necessarily the result of impaired magnocellular processing, as was previously proposed by Milne et al. (2002). Finally, there has yet to be a direct physiological or anatomical demonstration of abnormal thalamic magnocellular structures in autism, as demonstrated in Fragile X syndrome (FXS) (Kogan et al., 2004b). In fact, in a subsequent study, Kogan et al. (2004a) demonstrated that both simple, first-order and complex-second-order motion sensitivity was decreased for persons with FXS, suggesting that when a magnocellular dysfunction does in fact exist, the sensitivity to *any type* of motion stimuli (i.e., first- or second-order) should be reduced. This was not the case for autism (Bertone et al., 2003).

Intact ventral stream functioning in autism?

Milne et al. contend that the unaffected autistic sensitivity on the coherent form tasks used by Spencer, O'Brien, Riggs, Braddick, Atkinson, and Wattam-Bell (2000) and Blake et al. (2003) can be used as evident against the *complexity specific hypothesis* since "... impairments in the ability to integrate perceptual information occur in the perception of motion, but not all perceptual information". Concurrently, the *pathway-*

specific hypothesis is supported since theses groups clearly demonstrate impaired complex motion perception and preserved complex form perception in autism. We argue that the intact/static, impaired/dynamic dissociation reported in these studies may be stimulus dependent, since static circular stimuli were not equivalent to their complex dynamic counterparts (which were not circular in nature) in terms of processing requirements (see Bertone, 2004, for complete discussion). The reason for this is that due to specialized analysis, integrating local elements into complex visual information, whether dynamic or static, is more efficient when the local information is organized in a circular manner (Freeman & Harris, 1992; Kovács & Julesz, 1993; Wilson, Ferrera, & Yo, 1997; Wilkinson, Wilson, & Habak, 1998; Kovács, Kozma, Fehér, & Benedek, 1999; Burr & Santoro, 2001; Achtman, Hess, & Wang, 2003). Therefore, one can argue that the decrease in sensitivity for the complex motion condition (and not the complex form condition) in the Spencer et al. (2000) and Blake et al. (2003) studies may have been at least in part due to the fact that only one condition used circular stimuli (complex form condition). In addition, the detection of complex form contours from individual oriented line elements may be achieved at earlier levels than previously believed, i.e., orientation selective mechanisms operating in V1 (see Hess, Hayes, & Field, 2003, for a review). As suggested by Blake et al. (2003), Bertone (2004), Pellicano et al. (2005), and Bertone et al. (2005), such complex motion and form tasks do not assess functioning in either visual stream at the same level of neural complexity. That being said, we argue that the complex motion (coherent and/or biological) and complex form stimuli may differ in terms of their sensitivity to neuro-integrative dysfunction. As a consequence, if atypical autistic perceptual processing is indeed defined by a neuro-integrative dysfunction to *all* perceptual information, it will only be manifested in theses studies as decreased motion sensitivity (and intact form sensitivity) and interpreted incorrectly as evidence for a dorsal pathway dysfunction, or used as support for *pathway-specific hypothesis*.

Describing the etiology of impaired motion perception in autism using an "autism-specific perceptual signature"

A question that is central to the Milne et al. review is what is the etiology underlying impaired motion perception in autism? In order to

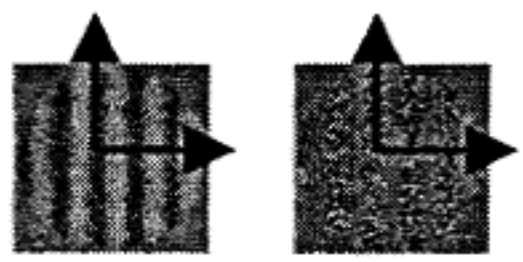
propose an abnormal neuronal architecture defining atypical motion perception in autism, it is necessary to use an experimental paradigm that results in a pattern of perceptual abilities that is specific to autism, or in other words, demonstrating an "autism-specific perceptual signature". Within this framework, it is not possible to conclude specific etiology based solely on decreased coherent motion sensitivity since this perceptual manifestation is shared with at least a dozen other neurological conditions (see Bertone et al., 2005, for a non-exhaustive list). Similarly, using the complex motion/complex form paradigm also results in a similar problem. For example, using the same experimental paradigm as Spencer et al. (2000) (i.e., complex motion/complex circular form), Kogan et al. (2004a) found that persons with FXS were less sensitive to coherent motion while sensitivity to complex circular form was intact. Therefore, based on the result of these two studies, it is not possible to dissociate the two conditions at a perceptual level (i.e., they share the same "perceptual signature"), making it difficult to suggest an etiology regarding atypical perceptual processing specific to either condition. However, at a physiological level, the two conditions are in fact different since Kogan et al. (2004a) elegantly demonstrated that, at a physiological level, persons with FXS have abnormal magnocellluar neurons (a finding not yet demonstrated in autism). We therefore argue that the "complex motion/complex circular form" experimental paradigm used previously is not a sensitive enough technique to dissociate between conditions at a perceptual level. This makes it difficult to suggest a specific etiology underlying impaired motion perception as well as other characteristic atypical perceptual functioning in autism.

An alternative experimental paradigm

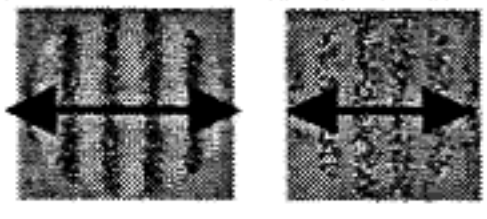
We propose an alternative experimental paradigm that we believe is better suited for evaluating dorsal and ventral visual stream integrity at comparable levels of complexity in autism, resulting in a more precise and condition-specific assessment of perceptual processing in autism as well as for other neurological conditions. In order to do this, the sensitivity to static *and* dynamic stimuli (i.e., either stationary or drifting gratings) defined by first- and second-order attributes are measured. Specifically, static visual information processing, which is mediated by ventral visual stream, is evaluated using an orientation-identification task where partici-

pants are asked whether first- and second-order grating are oriented vertically or horizontally (see Figure 1, top two cells). Conversely, dynamic information processing, mediated by dorsal stream functioning, was evaluated using a direction-identification task using similar stimuli that drifted either to the left or right (see Figure 1, bottom two cells). We suggest that using this alternative paradigm for investigating the origin of visuo-perceptual abnormalities in autism is advantageous because both static and dynamic forms of first- and second-order information are processed *in the same manner* by passive mechanisms using similar principles of detection, described by filter-rectify-filter model (i.e., Chubb & Sperling, 1988; Wilson, Wilkinson, & Asaad, 1992; Sperling, Chubb, Solomon, & Lu, 1994; Baker, 1999). Furthermore, in terms of their relative complexity, the first- and second-order tasks access dorsal and ventral visual stream processing *at the same level of complexity*, defined by the physiological limitations of the mechanisms operating within each candidate level initially responsible for their detection (i.e., V1 and V2/V3).

Figure 1. Alternative research design for to assessing perceptual functioning in autism.
The perpendicular arrows represent an orientation-identification task and the oppositely-oriented arrows represent a direction-identification task.



		Complexity	
		Simple	**Complex**
Pathway	**Ventral**	**?**	**?**
	Dorsal	**?**	**?**



We have recently implemented this paradigm by assessing ventral stream processing at two levels of complexity using the proposed first-

and second-order orientation-identification task. In this study, participants were asked to identify the orientation of first- and second-order gratings; orientation-identification thresholds were measured for each condition for both high-functioning persons with autism (HFA) and typically developing (TD) participants. Our results demonstrate that the ability of HFA is *superior* for identifying the orientation of simple, first-order gratings but *inferior* for identifying the orientation of complex, second-order gratings when compared to TD participants. We have interpreted this finding as evidence for atypical neuronal architecture in autism defined by excessive lateral inhibition in autism, possibly accounting for both enhanced (static information processing) and decreased (dynamic information processing) low-level information processing in autism (please refer to Bertone et al., 2005, for complete discussion). More importantly within the context of this discussion, we have now provided evidence that further supports the *complexity specific* account in autism by directly assessing and demonstrating that both static (Bertone et al., 2005) and dynamic (Bertone et al., 2003) complex, second-order information processing is impaired in autism. Using this same paradigm, Kogan et al. (2004b) found a different "perceptual signature" for persons with FXS, allowing for a more precise hypothesis to be forwarded with respect to the etiology of perpetual abilities in both autism and FXS. In addition, the "perceptual signature" resulting from the proposed paradigm is actually more consistent with their previous findings of abnormal magnocellular physiology (see Kogan et al., 2004b).

To conclude, our results from both motion and orientation studies indicate that low-level visuo-perceptual processing in autism is atypical and that such abnormal processing is very compatible with the *complexity-specific hypothesis* in autism.

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