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

Unraveling the mystery of motion perception impairments in autism: Some further considerations

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The authors (Milne, Swettenham, and Campbell) have done a masterful job of reviewing the literature available on the multifaceted findings in motion perception in autism. And, as does any good review article in a field, it raises as many interesting questions as it answers, essentially acting as a clarion call for more research in the field. While this review highlights the advances that have been made in investigations of visual processing in those with autism, it also highlights the complexity of research in this population.

First, the authors stress the need for greater clarification and definition of the motion perception deficit in autism. As mentioned in the article, studies of those with autism have found deficits in coherent motion and biological motion perception, greater sensitivity to first-order than to

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second-order motion, and abnormal postural reactivity to optic-flow motion. Additionally, it has also been suggested that those with autism are more impaired at motion perception when a stimulus is moving more quickly, suggesting that autism may involve a temporal binding deficit (Brock, Brown, Boucher, & Rippon, 2002). These studies have all pointed to motion perception deficits in autism and are suggestive of a basic magnocellular/dorsal stream deficit. They have not, however, investigated different aspects of motion perception in the same cohort and thus are unable to shed light on the question of whether all of these potential deficits occur in the same subjects, nor if they may all stem from a common root cause. Lesion data suggest that coherent motion perception is separable from biological motion, first-order motion perception from second-order motion perception, and all of these from optic flow perception. It is therefore impossible to know if all these deficits in autism are bound together by a common developmental insult to early dorsal stream areas or are reflective of a general susceptibility of this pathway that is expressed differently in each individual. Studies investigating all of these deficits in the same cohort will be important in clarifying not only whether these deficits are tied together in those with autism but also the possible etiologies of these deficits. The authors have also correctly stressed the need to report individual as well as group data as their own investigations have shown visual-motion processing deficits in only 22% of those in their autism cohort (Milne, Swettenham, Hansen, Campbell, Jeffries, & Plaisted, 2002). This observation raises the possibility of looking at deficits in visual processing as a means of subtyping the very heterogeneous population of those with autism.

Although not stressed in the article, it is clearly also important to determine at what point in development these deficits may occur. As the authors point out, the fact that different facets of motion processing are separable in the adult does not necessitate that they are unrelated developmentally. Finding the age at which these deficits are first detectable will be important in understanding what relationship, if any, these deficits have to the etiology and severity of autistic symptomatology. If very early deficits in visual motion processing in those that go on to develop autism could be shown, it would suggest a model whebeby an early insult to dorsal stream areas feeds forward in the patterning of connectivity throughout areas sensitive to visual motion perception, possibly including social cognition areas that receive information about human movement and intention. Studies in infant siblings of those with autism that investigate visual

motion discrimination might be particularly useful in clarifying this issue. The authors also ask intriguing questions about the possible relationship between motion processing deficits in those with autism and abnormalities seen in their cognitive style. Clear parallels can be drawn between new findings showing that people with autism rely more on high-spatial frequency information than low-frequency when processing faces (Deruelle, Rondan, Gepner, & Tardif, 2004) and possible magnocellular "stream" dysfunction. As the magnocellular stream contributes low-frequency information to visual processing, it is easy to see how a deficit in the magnocellular stream could lead to a relative reliance on high-frequency information. A related suggestion that has been posited in the literature is that these data can be explained by the finding that many of those with autism have a natural (though contrary to the normal) tendency towards processing local details before global features (Plaisted, Swettenham, & Rees, 1999; O'Riordan & Plaisted, 2001, Milne, Swettenham, Campbell, & Coleman, 2004). In their own studies, the authors have found that those with the greatest deficit in motion coherence processing were also those with the highest local bias (Milne et al., 2004). Thus two prominent features of autism that have often been tied together, a lack of central coherence and a debilitating local processing bias, are themselves tied to deficits in visual motion processing. If nothing else, this suggests that looking at autism from the perspective of primary sensory processing may indeed be a good place for productive research.

It is also worth asking questions about how patterning in the brain might change with an early insult to either magnocellular cells in the retina, thalamus or primary visual cortex, or to early dorsal stream areas like MT/V5, with neurons in this area being sensitive to coherent motion (Merigan, Byrne, & Maunsell, 1991; Braddick, O'Brien, Wattam-Bell, Atkinson, J., & Turner, R., 2000). Information from magnocellular cells feeds preferentially into the dorsal stream, but also constitutes a considerable percentage of the information received by the ventral stream (Maunsell et al., 1990; Nealey & Maunsell, 1994). Thus, if magnocellular cells were affected, we might expect to see deficits in both the dorsal and ventral streams, though those deficits would likely be seen more strongly in functions subserved by dorsal stream areas. If dysfunction were instead specific to a particular brain region, like MT/V5, we would expect more profound deficits in coherent motion processing with possible feed-forward deficits in other dorsal stream areas but no detectable deficits in ventral stream areas. Information from MT is fed forward primarily to

MST (specific for visual flow information) and the STS (superior temporal sulcus) which also integrates information from ventral areas as well as auditory areas. Thus dysfunction in MT, especially early in development, could cascade forward to MST and STS, resulting in some degree of deficit in optic flow perception and biological motion perception.

Although the authors focus on early dorsal stream dysfunction, there is also evidence that dysfunction may be specific to the STS region. The authors mention research showing a deficit in biological motion processing in those with autism and point out the specificity of the STS region in biological motion perception, but they do not mention studies showing STS abnormalities in autism. Evidence that the STS may be dysfunctional in those with autism is plentiful. A recent study comparing cortical sulcal maps in individuals with and without autism found anterior and superior displacement of the STS (Levitt et al., 2003). Another group recently reported smaller STS volumes in those with autism (Boddaert et al., 2004) while a PET study of regional blood flow in those with autism found hypoperfusion in the STS (Ohnishi et al., 2000). Another PET study showed hypoactivation of the STS and reduced functional connectivity between the STS and extrastriate cortex in those with autism when viewing animated geometrical figures and performing a task requiring assigning and assessing intentionality to the shapes (Castelli, Frith, Happé, & Frith, 2002). Hypoactivation of the STS in autism has also been seen during human speech perception tasks (Boddaert et al., 2003; Gervais et al., 2004) as well as during eye-gaze perception tasks (Pelphrey, Morris, & McCarthy, 2005). The STS is also closely connected with the orbitofrontal cortex and the amygdala, two other brain regions that have been hypothesized as being dysfunctional in autism (Sabbagh, 2004; Seguin, 2004). Dysfunction in the STS or any decreases in its interconnectivity with other brain regions could have profound effects on social cognition, especially if such differences were present from very early in development. If the STS were the primary site of dysfunction, demonstrations of earlier dorsal stream dysfunction might be a result of feedback cascades from STS to MT/V5, V1 or even thalamus. Alternatively, these data in the STS could be the result of patterning differences produced by early insult to magnocellular areas.

Imagine, if you will, a scenario in which many of these possibilities come together. Abnormalities in magnocellular areas impact visual experience and patterning of "later" visual areas. This results in abnormal coherent motion processing within MT, which feeds information to the

STS, resulting in abnormal biological motion processing. Information about biological motion feeds from the STS to orbitofrontal cortex and amygdala, changing their response patterns to emotionally or socially relevant moving stimuli. At the same time, these magnocellular deficits would result in a relative bias towards local rather than global processing as well as a reliance on low-spatial resolution cues. This could lead to both deficits in face processing and inability to parse complex social scenes as a whole (potentially compounded by difficulties in understanding social scenes due to deficits in biological motion perception). Although clearly speculative, this scenario illustrates the power that this line of basic sensory research may have in investigating even those areas that have historically been considered to stem from higher-order deficits in social reciprocity. Certainly, it suggests that research in this area may shed light on what some consider to be the "primary" domain of deficit in autism, social cognition, and reciprocity.

It must be mentioned again, however, that data showing only a subset of those with autism with measurable deficits in coherent motion detection are stressed several times in this article and beg the question not only of if the scenario we just posited is plausible but of whether this observed dorsal stream deficit has any substantial and predictable functional consequences at all. If only a subset of those with autism show these deficits, what relationship, if any, do dorsal stream deficits have with autistic symptomology? Although one study (Blake, Turner, Smoski, Pozdol, & Stone, 2003) found a connection between motion processing deficits and symptom severity in their autism group, other groups have not looked at this question closely. If, as the authors themselves have found, deficits in motion perception map onto a tendency towards local bias or a greater reliance on low spatial-frequency visual information, might they also map onto the difficulties those with autism have in processing faces, which in turn map onto deficits in social cognition? If less than a quarter of those with autism show these deficits, is it because there is no dysfunction in magnocellular areas for the majority of these individuals, or because they have a milder dysfunction that is not detectible with the probes being used? If the deficit *is* truly only present in a subset of those with autism, is that subgroup distinguishable clinically? Further research is needed to clarify these issues, particularly looking for symptomology that these deficits might predict.

One observation made by the authors may help in ascertaining the clinical and functional implications of these dorsal-stream deficits. As

mentioned in the target article, autism is not the only developmental disability in which these deficits have been characterized. Increased motion coherence thresholds have been found in Fragile X (about 30% of which are also diagnosed with autism) (Kogan et al., 2004), and William's syndrome (Atkinson, King, Braddick, Nokes, Anker, & Braddick, 1997; Jordan, Reiss, Hoffman, & Landan, 2002). Those with William's syndrome are characterized by profound spatial cognition deficits with relatively preserved (or even hyper) social cognition. One group (Jordan et al., 2002) found that despite higher coherent motion thresholds, those with William's syndrome have preserved biological motion perception. Those with Fragile X syndrome (without autism) are characterized by mental retardation, sensory hypersensitivity and high anxiety, and generally show some overlap with autism in problems (generally much milder) in social reciprocity and repetitive behaviors. Higher coherence thresholds in this population have been characterized but biological motion perception has not been investigated. Both populations would be extremely important to study in conjunction with those with autism. Both syndromes are a result of a definable genetic insult, thus providing fertile ground for understanding the possible genetic underpinnings of dorsal stream dysfunction in autism. Dorsal stream deficits in all of these groups may also suggest a pathogenesis common to all of these disorders. Differences in the degree and extent of these deficits among these disorders may give important clues about how these deficits impact those with autism or may be influences in creating autistic symptomology.

The data reviewed in this article also make it clear not only that all of autism cannot be reducible to motion processing deficits, but that motion processing deficits in those with autism may themselves not be reducible to a simple and consistent deficit in magnocellular pathways. Those with autism are not motion-blind and may not show consistent deficits across all moving stimuli. Deficits tend to become more apparent and more reproducible across subjects as stimuli become more complex. As the authors suggest, these data could be explained both by a mild insult to magnocellular areas that only becomes apparent when the system is taxed by more complex stimuli, or by a deficit in information integration. They are correct in dismissing the idea of there being *only* a deficit in information integration, however, as equivalent information integration requirements when processing form rather than motion information did not result in a similar deficit (Spencer, O'Brien, Riggs, Braddick, Atkinson, & Wattam-Bell, 2000; Blake et al., 2003). There is, however, clearly a pos-

sibility that both information integration and magnocellular damage could contribute significantly to visual motion processing deficits. Both might contribute to deficits in the processing of any complex moving stimulus, including social scenes. Although the authors don't stress this point, it is clear that a combination of both deficits could explain why situations that require both magnocellular function and information integration (especially local information integration of motion cues necessary for global understanding of a scene) seem to be particularly challenging for those with autism.

This article leaves one with a clear understanding of the state of the field and a list of burning research questions to pursue. From our perspective, the most pressing include the need to investigate different *aspects* of motion perception in the same person, the importance of looking at the developmental origins of these deficits, the need for investigating how patterning in the brain might change as a function of the location of dysfunction, and the importance of studying disorders arising from a specific genetic defect, such as Fragile X syndrome, which may help elucidate the genetic underpinnings of dorsal stream dysfunction in autism.

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