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Demonstrations of Decreased Sensitivity to Complex Motion Information Not Enough to Propose an Autism-Specific Neural Etiology

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Interest regarding neural information processing in autism is growing because atypical perceptual abilities are a characteristic feature of persons with autism. Central to our review is how characteristic perceptual abilities, referred to as *perceptual signatures*, can be used to suggest a neural etiology that is specific to autism. We review evidence from studies assessing both motion and form perception and how the resulting *perceptual signatures* are interpreted within the context of two main hypotheses regarding information processing in autism: the *pathway*- and *complexity*-specific hypotheses. We present evidence suggesting that an autism-specific neural etiology based on perceptual abilities can only be made when particular experimental paradigms are used, and that such an etiology is most congruent with the *complexity*-specific hypothesis.

KEY WORDS: Autism; low-level information processing; motion processing; form processing; neural networks; lateral inhibition.

INTRODUCTION

Autism is a neuro-developmental syndrome with a biological basis and behavioral definition. Given the importance of clinical manifestations with respect to diagnosis, it is not surprising that the direction of research investigating underlying neural mechanism in autism has been for the most part symptom-driven. As a result, research direction and the development of neuro-behavioral theories in autism attempting to link brain dysfunction, cognitive processes and characteristic behaviors have for the most part focused on cognitive, neuropsychological and/or social capacities in autism directly related to its clinical manifestations. Recent imaging studies suggesting atypical face processing (i.e., Critchley *et al*., 2000; Hubl *et al*., 2003; Pierce, Müller, Ambrose, Allen & Courchesne, 2001; Schultz *et al*., 2000), deficient *mentalizing* ability (Castelli *et al*., 2002) and impaired language processing (Just, Cherkassky, Keller, & Minshew, 2004) exemplify this research direction. In general, the findings from these studies are interpreted in the framework of abnormal large-scale neural connectivity in autism, characterized by either inefficient or atypical integration of information between cortical regions involved in their respective tasks. Although they differ with regard to the nature of their respective connectivity dysfunction (reduced feedback modulation between higher-and lower-cortical areas, Castelli, Frith, Happé, & Frith, 2002; decreased connectivity between cortical regions; Just *et al*.,

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2004), all include evidence of atypical neural connectivity that is relevant to higher-level information processing deficits and that may be informative about underlying etiology.

As alluded to by Belmonte *et al*. (2004), the recent impetus on assessing higher-level, symptom-related functioning resulted in the ''over-looking'' of important questions about the mechanisms responsible for atypical lower-level perceptual processing in autism. For example, the nature or origin of abnormal connectivity in autism is often explained by dysfunctional *feedback* mechanisms or *top–down* modulation of lower-level information (Frith, 2003). And, in some instances, the *same* neural abnormality resulting in higher-level dysfunction are linked to the sparing, or even enhancement, of lower-level information processing in autism (Brock, Brown, Boucher, & Rippon, 2002; Belmonte *et al*., 2004; Frith, 2003; Minshew, Goldstein, & Siegel, 1997). These suggestions are based on the assumption that lower-level perceptual information processing in autism is unremarkable—but is it?

Perceptual systems provide cognitive mechanisms with an internal representation or our external world. Therefore, atypical low-level perceptual processing may be implicated with autistic symptoms at different levels. In addition to socio-behavioral difficulties, atypical processing of low-level, perceptual information is also a characteristic feature of autism (Happé, 1999; Happé & Frith, this issue; Mottron & Burack, 2001; Mottron *et al*, this issue). This is consistent with the notion that the processing of visual information is somewhat unique in autism as it often manifests itself with *enhanced* (or more locally oriented) performance on tasks necessitating static spatial information processing. This characteristic performance is demonstrated at different visuo-perceptual levels with either psychophysical methods assessing lower-level and mid-level perception, or neuropsychological tasks tapping higher-level perception (i.e., Caron, Mottron, Rainville, & Chouinard, 2004; Jolliffe & Baron-Cohen, 1997; Mottron, Belleville, & Ménard, 1999, 2003; O'Riordan, Plaisted, Driver, & Baron-Cohen, 2001; Pellicano, Gibson, Maybery, Durkin, & Badcock, 2005; Plaisted, Sweetenham, & Reese, 1999; Shah & Frith, 1983; Shah & Frith, 1993). In this paper, we will argue that, whereas diminished performance on dynamic tasks (see later sections) is evident in other neurological conditions, *enhanced* perceptual performance, particularly for tasks requiring static or spatial information processing, is *specific* to autism. This evidence will come from psychophysical studies assessing static and dynamic information processing in autism and related conditions (i.e., fragile X syndrome).

MOTION PERCEPTION IN AUTISM

Motion perception can be considered as a relatively low-level perceptual attribute upon which more complex behaviors are based. Apart from the interest in studying motion perception in autism as one of several aspects of visual processing, atypical motion processing may account for both idiosyncratic interests in moving objects and atypical eye-contact during interpersonal communication. These and other ''motion-related'' behaviors raise several questions regarding motion perception in autism. The first question is whether persons with autism have normal or atypical basic motion perception capabilities. The idea that visuo-perceptual dysfunction may, at least in part, underlie abnormal behavior in autism and other developmental disorders (i.e., William's syndrome, fragile X syndrome, dyslexia, etc.) led to an increased use of applied psychophysical techniques and stimuli investigating dynamic information processing in these populations. Second, the use of motion perception tasks to evaluate neural functioning in autism is attractive, since it is well accepted that most motion stimuli used in such tasks (i.e., random dot kinematograms) directly assess or ''target'' the functioning of specific motion-sensitive brain areas (i.e., human MT; see later sections). Given this strong association between task performance and neural functioning, persuasive inferences can be forwarded regarding whether neural networks mediating such perceptual subsystems are intact in autism. In the following sections, the results of studies using different experimental approaches to assess motion perception in autism are presented as well as how such results have been used to make more general inferences regarding autism-specific neural etiology.

Postural Reactivity to Motion Information

Gepner, Mestre, Masson, and de Schonen (1995) were the first to suggest that motion perception may be atypical in autism. They found that, unlike in typically developing children, the postural stability of children with autism was unaffected by the presentation of a radiating flow-field. They concluded that the lack of postural reactivity may be the result of either impairment in motion perception, or a lack of visual

attention to the radiating stimulus. The findings of this initial experiment suggested an abnormal reaction to a radiating stimulus that usually induces vection; a visuo-vestibular illusion recognized as evidence for the strong effect of visual information on proprioceptive perception (see Warren, 1995). However, these findings might have also resulted from either a difficulty with higher-order operations (i.e., attention), or a motor functioning impairment (e.g., inadequate sensori-motor integration).

In a follow-up study, Gepner and Mestre (2002a) demonstrated that "postural hypoactivity" (i.e., diminished postural dependence on visual information) was specific to autism, and not generalizable to children with Asperger's syndrome (AS), and that postural hypoactivity for children with autism was consistent across increasing angular velocities of the radiating flow-fields. This was not the case for children with AS and comparison participants, whose postural activity increased as the angular velocity of the flow information increased. Gepner and Mestre proposed three possible interpretations for their findings: a sensori-perceptual, a motor and a sensorimotor interpretation.The sensori-perceptual interpretation states that children with autism present a visual perception impairment leading to a "visuo-postural detuning." This reasoning was aimed at explaining why the postural hypoactivity (difference in postural reactivity between the autism, Asperger and normal groups) was most evident for the autism group during conditions where the peak angular velocity of the flow information was high. In addition, Gepner and Mestre suggest the possibility of impaired dorsal visual stream functioning as the potential neural origin of the motion perception impairment (also Gepner, Druelle, & Grynfeldt, 2001). These propositions were the foundation of their proposed theory of Rapid-visual Motion Integration Deficit in autism (Gepner & Mestre, 2002b) in which a visual-motion integration deficit (particularly to high-velocity motion) may be a neurobiological marker of the level of motor impairments manifested by children with autism.

The work of Gepner and colleagues initiated research on the involvement of impaired dynamic information processing in autism, possibly manifested in autistic symptomology, etiology, and behavior. Specifically, evidence as to how the postural stability is induced (or not) by visual motion information differs between children with autism and AS are informative as such differences may indicate the plausibility of a neurobiological marker for autism. However, this suggestion is constrained by certain practical and theoretical limitations. Gepner and Mestre's (2002a) findings of differential reactivity between children with autism and AS are based on a limited amount of participants in both autism ($n = 3$) and AS ($n = 3$) groups. The statistical power may be insufficient to decide whether postural control differs between the groups. In addition, the nature of the task, which involves postural reactivity as a dependant measure, limits interpretations regarding the visual information processing capacities in autism. As outlined by Gepner and Mestre (2002a), there are three possible underlying reasons for autistic postural hypoactivity; a sensori-perceptual, a motor or a sensorimotor abnormality. Although results from these types of studies can be used to demonstrate the possible *involvement* of abnormal motion perception with regards to atypical postural reactivity, they cannot be interpreted as a direct evidence for deficient motion perception abilities in autism. The reason for this is the following: the dependant measure (postural reactivity) is not directly associated with abnormal motion perception since factors other than motion perception (i.e., motor functioning or sensori-motor integration) may explain atypical postural reactivity in autism. Another issue can be addressed with regards to the involvement of motion perception with abnormal postural reactivity in autism. Malloy, Dietrich, and Bhattacharya (2003) evaluated the postural stability of children with autism by measuring their postural sway while standing on either a platform or foam (modifying afferent input) with either their eyes open (EO) or closed (EC or blindfold). They found that the children with autism swayed significantly more (i.e., less postural stability) for the EC conditions compared to typically developing children, regardless of afferent somatosensory input. These findings suggest that abnormal postural reactivity in autism can also be manifested in the *absence* of dynamic visual information, or for *any* type of visual information. In this case, there seems to be an over-reliance on visual input to maintain postural stability, whereas Gepner and colleagues found an "under-reliance" on visual information (i.e., radiating flow-fields) for inducing postural reactivity in autism.

The "Pathway Specific" Hypothesis: Global Motion Studies

Probably the most common and enticing method of evaluating motion processing in autism and for

other neurological conditions is the ''global motion approach.'' Psychophysical and electrophysiological researchers distinguish between local or simple motion processing (the sensitivity to the direction in a small region of the visual image mediated by standard motion analysis), and ''global'' or complex motion processing. The latter allows for the discrimination of motion direction over extended regions of the visual scene that necessitates the passive integration of local motion signals into a coherent whole. Complex motion processing is usually identified with the integrative properties of MT neurons (i.e., Newsome & Paré, 1988), while simple motion processing reflects the processing of standard motion analyzers found in the primary visual cortex.

The stimuli of choice for investigating global motion perception are random-dot kinematograms (RDKs). In such patterns, a proportion of dots move coherently in a certain direction while the remaining dots move in random directions. This type of processing therefore exemplifies early neuro-integrative processing since local motion information must be integrated across space and time before a global or coherent motion direction can be discriminated; the processing of individual or local dots cannot reveal the overall or global motion direction. Reasons for the popularity of this approach probably originates from the fact that (1) global motion perception reflects early neuro-integrative analysis and (2) the underlying processing is mediated by specialized extra-striate motion-sensitive areas (i.e., area MT), exemplary of the dorsal visual stream functioning. Thus, inferences can be made concerning the integrity of the neural mechanisms mediating complex motion processing, and can speculatively be extended to more general neuro-integrative mechanisms, based on the participant's ability to perceive coherent motion at a specific level of coherence. In addition to such interpretations, the global motion model may also be used to assess *perceptual coherence* in autism (Happé, 1999), since global motion perception can be seen as dynamic type of gestalt-like grouping or integration (Watt & Phillips, 2000).

Spencer *et al.* (2000) were the first to assess complex motion perception in autism, using an adapted global motion task (see Atkinson *et al.*, 1997). They demonstrated that participants with autism were less sensitive to global motion as compared to typically developing comparison participants, and suggested a dorsal stream deficiency in autism, since complex motion perception is attributed to the functioning of this visual stream (Goodale & Milner, 1992; see Merigan, Byrne & Maunsell, 1991; Ungerleider & Mishkin, 1982; for an alternative view). Their interpretation was further supported by the finding that persons with autism performed similar to the comparison group on a complex form recognition task, believed to be mediated by the ventral visual stream processing (static information processing discussed in later sections). Spencer *et al.* (2000) suggested that the atypical autistic visual processing is best explained by a *pathway-specific hypothesis*, namely dysfunctional dorsal stream functioning, and intact ventral stream functioning. The findings of Spencer *et al.* (2000), along with similar demonstrations of deficient dorsal/intact ventral stream in other developmental disorders (using the same complex motion and form task), have been interpreted by Braddick, Atkinson, and Wattam-Bell (2003) as being indicative of a « dorsal-stream vulnerability » in autism. The *pathway-specific hypothesis* was further supported by Milne *et al.*'s (2002) finding that a group of children with autism demonstrated significantly higher motion coherence thresholds when compared to typically developing children. They interpreted their results as evidence for impairment in the magnocellular system that represents the main thalamic input to the dorsal visual stream. These authors also suggested that magnocellular impairment, responsible for low spatial frequency information analysis, may also explain the tendency to focus on local, rather than global, aspects of visual stimuli among persons with autism.

In another study, Blake, Turner, Smoski, Pozdol, and Stine (2003) assessed biological motion perception, or the ability of participants to recognize human activities (i.e., jumping, throwing, climbing, etc.) where a moving animate body is perceived from the movements of a several points (Johansson, 1973), among persons with autism. In order to perceive such an ecologically meaningful motion percept, the visual system must integrate related points of motion so that the activity of the biological stimuli may be identified. Although biological motion differs from global motion in that the global interaction of the points depicts a human behavior and not a motion direction, it shares the defining characteristic of being a complex type of motion that necessitates neuro-integrative processing. Specifically, the extra-striate area believed to be responsible for biological motion perception (superior temporal sulcus, or

STS) lies within the extra-striate dorsal visual stream. Blake *et al*. (2003) demonstrated that persons with autism were less likely to identify a biological sequence as being that of a ''person,'' but performed similar to comparison participants on a complex form task (i.e., adapted pathfinder display, Field, Hayes, & Hess, 1993), suggesting intact ventral stream processing. Blake *et al*. (2003) interpreted their results as further evidence for a dorsal pathway impairment in autism.

The studies by Spencer *et al*. (2000), Milne *et al*. (2002) and Blake *et al*. (2003) support the notion that complex motion processing is atypical in autism, whereas complex form processing, mediated by ventral stream processing is intact (Blake *et al*., 2003; Spencer *et al*., 2000). These researchers explain their findings in the framework of a dorsal visual stream impairment in autism, that we will continue to refer to as the *pathway-specific hypothesis*.

ALTERNATIVE EXPLANATION FOR DECREASED SENSITIVITY TO COMPLEX MOTION STIMULI IN AUTISM: THE "COMPLEXITY-SPECIFIC HYPOTHESIS"

Spencer *et al*. (2000), Milne *et al*. (2002) and Blake *et al*. (2003) all suggested that impaired complex motion perception in autism is likely due to inefficient dorsal visual stream processing and/or localized impairments of motion-sensitive mechanisms *per se*, operating in extra-striate areas within the dorsal visual pathway (i.e., MT, STS). Although their interpretations are consistent with their findings, an alternative explanation based on the complexity of the motion stimuli used in their experiments cannot be ruled out. For example, according to the *complexity-specific hypothesis*, decreased complex motion sensitivity in autism results from diffuse or non-specific neural dysfunction of neuro-integrative mechanisms affecting complex perceptual processing in general. We argue that the notion of a dorsal stream dysfunction cannot be confirmed and, therefore, the *complexity specific hypothesis* cannot be rejected, unless motion-processing mechanisms operating at *different* levels of complexity along the dorsal visual pathway are evaluated (i.e., simple processing mediated by striate mechanisms and complex processing mediated by extra-striate mechanisms). Accordingly, if dorsal stream functioning is deficient in autism, all types (both simple and complex) of dynamic motion processing should be affected.

Evidence of Intact Simple Motion Processing in Autism

In an attempt to dissociate *pathway* vs. *complexity*-specific hypotheses, Bertone, Mottron, Jelenic, and Faubert (2003) measured the sensitivity of persons with autism for first- and second-order motion classes of motion stimuli. These classes of motion stimuli differ from each other in the amount of neuro-integrative analysis required to perceive its direction; (Cavanagh & Mather, 1989; Chubb & Sperling, 1998), and contemporary motion models distinguish first- and second-order motion classes by the level at which they are processed along the dorsal visual pathway. First-order (or luminance-defined) motion is considered to be a ''simple'' type of motion since it is initially processed by standard motion selective mechanisms operating within the primary visual cortex (or V1). Second-order motion information is detected at a second-stage of processing by mechanisms operating in extra-striate motion areas (i.e., V2/V3, see Baker, 1999; Bertone & Faubert, 2003; Chubb & Sperling, 1998; Nishida, Ledgeway, & Edwards, 1997; Sperling, Chubb, Solomon, & Lu, 1994; Wilson, Ferrera, & Yo, 1992) and is, therefore, considered a more ''complex'' motion class. Second-order motion information recruits more extensive neural circuitry and necessitates additional processing prior to its detection at higher-levels along the dorsal pathway.

With regard to processing among persons with autism, Bertone *et al*. (2003) found that simple, first-order motion perception was unaffected whereas a selective decrease in performance was evident for complex, second-order motion perception. These findings were interpreted within the context of the *complexity-specific hypothesis* as the result of abnormal neuro-integrative analysis of low-level perceptual information since simple motion processing, which is mediated by dorsal stream functioning, was found to be unaffected (Bertone *et al*., 2003).

Bertone *et al*.'s (2003) interpretation was supported by evidence from Pellicano *et al*.'s (2005) study of dorsal stream functioning at two different levels of complexity in autism. Pellicano *et al*. (2005) first assessed lower-level (thalamic or pre-cortical) dorsal stream functioning with a flicker sensitivity task that can be used to assess magnocellular functioning, an important input for dorsal stream processing. Second, they evaluated extra-striate dorsal stream functioning with a global dot motion task (adapted global motion task) that necessitates neuro-

integrative processing to be completed. They found that magnocellular functioning was unaffected, although global motion sensitivity was reduced among their group of persons with autism. These findings are consistent with Bertone *et al.*'s (2003) notion that lower-level dorsal stream functioning, whether mediated by pre-cortical (Pellicano *et al.*, 2005) or early-cortical (Bertone *et al.*, 2003) mechanisms operating in the striate cortex) mechanisms, are intact in autism. This conclusion supports the *complexity-specific hypothesis*, as the decreased complex motion sensitivity in autism seems to be better explained by inefficient neuro-integrative processes than by "dorsal stream vulnerability" (Braddick *et al.*, 2003).

The Global Motion Deficit is Not Specific to Autism

Complex motion analysis appears to be impaired in a variety of conditions, including multiple sclerosis (Regan, Kothe, & Sharpe, 1991), non-pathological aging (Trick & Silverman, 1991), dementia of the Alzheimer's type (Gilmore, Wenk, Naylor, & Koss, 1994), dementia of the Parkinson's type (Trick, Kaskie, & Steinman, 1994), dyslexia (Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Cornelissen *et al.*, 1998), William's syndrome (Atkinson *et al.*, 1997), non-pathological aging (Habak & Faubert, 2000), hemiplegic cerebral palsy (Gunn *et al.*, 2002), schizophrenia (Chen, Nakayama, Levy, Matthysse, & Holzman, 2003), amblyopia (Simmers, Ledgeway, Hess, & McGraw, 2003), mild cognitive impairment (Mapstone, Steffenella, & Duffy, 2003), fragile X syndrome (Kogan *et al.*, 2004a) and migraine (McKendrick & Badcock, 2004). As decreased motion sensitivity is evidently not specific to autism, we argue that it is somewhat difficult to suggest an autism-specific neural etiology, particularly one suggesting dorsal stream dysfunction, based on findings of decreased motion sensitivity. Moreover, simple motion sensitivity was found *unaffected* among the aforementioned studies, when simple motion was also assessed (Chen *et al.*, 2003; Habak & Faubert, 2000; Mapstone *et al.*, 2003; McKendrick & Badcock, 2004). Therefore, complex motion stimuli seem to be very sensitive for detecting different types of non-specific or diffuse neuro-integrative dysfunction in a variety of neurological populations, but cannot be used in isolation to suggest condition-specific neural etiology in autism or other neurological conditions. It has been suggested that the neural etiology of such integrative dysfunction may be defined by atypical neural connectivity affecting the integration of low-level perceptual information in autism (Cohen, 1994; Brock *et al.*, 2002; Bertone, Mottron, & Faubert, 2004; Grice *et al.*, 2001; Gustaffson, 1997a, b; Just *et al.*, 2004; McClelland, 2000).

AN AUTISM-SPECIFIC PERCEPTUAL SIGNATURE

Using a first- and second-order orientation-identification task (Habak *et al.*, 2000; Kogan *et al.*, 2004b), we assessed ventral stream processing in autism at two levels of complexity (Bertone, Mottron, Jelenic, & Faubert, 2005). The results from this and a previous study of dynamic processing are summarized in Fig. 1. The most important result is the finding of enhanced sensitivity for simple, first order gratings. This demonstration of *superior* processing of simple static information is *unique* to autism. In addition, the demonstration of *inferior* performance in identifying the orientation of complex, static information and in discriminating complex, dynamic information further supports the *complexity-specific* hypothesis since the processing of complex perceptual information, whether static or dynamic, is deficient in autism.

In contrast, the *pathway-specific* hypothesis (impaired dorsal/intact ventral functioning) is only

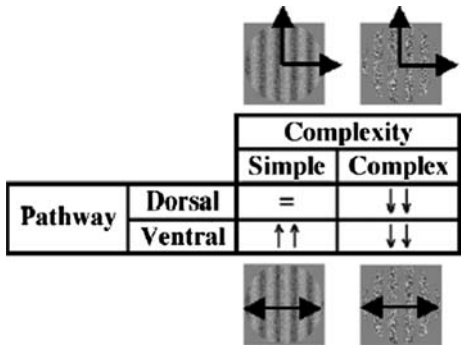

Fig. 1. Results from the alternative research paradigm for to assessing perceptual functioning in autism. The perpendicular arrows represent an orientation-identification task (Bertone *et al.*, 2005; upper panel) and the oppositely-oriented arrows represent a direction-identification task (Bertone *et al.*, 2003; lower panel). Arrows represent autistic sensitivity relative control participants for normally-aging persons; equal signs (=) and double arrows (↓↓, ↑↑), represent no difference and differences in sensitivity (respectively) between autism and control groups.

supported by the differentiation between impaired movement perception and *preserved* static perception in autism (Blake *et al*., 2003; Spencer *et al*., 2000). This perceptual signature is not specific to autism since persons with William's syndrome (Atkinson *et al*., 1997), fragile X syndrome (Kogan *et al*., 2004a) and schizophrenia (Kim, Doop, Blake, & Park, 2005) also manifest decreased sensitivity to complex motion (either global or biological motion) and equivalent sensitivity to complex circular forms as compared to typical observers with the same general experimental paradigm. These shared *perceptual signatures* may either result from the same pathophysiology, producing similar perceptual consequences, or, more plausibly, from the use of experimental paradigms that are insufficiently sensitive to detect differences. Furthermore, we argue that the static circular stimuli used by Spencer *et al*. (2000) and Blake *et al*. (2003) are not equivalent to their complex dynamic counterparts (i.e., global or biological motion stimuli which were not circular in nature) in terms of processing requirements (see Bertone *et al*., 2005 for complete argument).

We contend that specific *perceptual signatures* may be determined only by a simultaneous assessment of dorsal and ventral pathways at various levels of complexity. This is especially true for the comparison of performances between persons with autism and with fragile X syndrome. Fragile X syndrome is a condition, which sometimes manifests a behavioral phenotype comparable to that of autism (i.e., Bailey *et al*., 1998). The *signatures* produced using the complex form and motion tasks (as described by Atkinson *et al*., 1997) are summarized in Fig. 2 by the results of Kogan *et al*. (2004a) for persons with FXS (denoted as *signature* A) and by the results of Spencer *et al*. (2000) for persons with autism (denoted as *signature* B). The *perceptual signatures* resulting from these two studies are identical and therefore, it is difficult to propose an etiology that is specific to either condition based on the perceptual abilities assessed.

In contrast, assessing each visual pathway at two levels of complexity produced two *distinct* perceptual *signatures* for the same patient populations, as summarized by the results of Kogan *et al*. (2004b) for persons with Fragile X (denoted as *signature* C) and by the results of Bertone *et al*. (2003, 2005) for persons with high-functioning autism (denoted as *signature* D). Therefore, hypotheses about condition-specific etiology based on perceptual functioning may be proposed as the alternative method not only leads to distinct *signatures*, it also results in a signature that is more congruent with the known pathophysiology of either condition. For example, Kogan *et al*. (2004a) investigated the consequence of FMR1 gene dysfunction on both LGN physiology and demonstrated anatomical and morphological evidence of selective M-layer dysfunction in lateral geniculate nucleus (LGN) of persons with FXS. Based on their findings, Kogan *et al*. (2004b) had *a priori* reason to expect a decreased performance on both simple (first-order) *and* complex (second-order) direction-identification tasks since the M-cells feed the dorsal visual pathway; this is what was found (*signature* C). Conversely, there is no reason why simple information processing should be affected in autism, as abnormal magnocellular neuropathology has yet to be demonstrated in autism.

		FXS		HFA	
		Complexity		Complexity	
		Simple	Complex	Simple	Complex
		Signature A		*Signature* B	
Pathway	Ventral	=	=	=	=
	Dorsal	NE	↓↓	NE	↓↓
		Signature C		*Signature* D	
Pathway	Ventral	=	↓↓	↑↑	↓↓
	Dorsal	↓↓	↓↓	=	↓↓

Fig. 2. Schematic representation of perceptual signature originating from the Kogan *et al*. (2004a) (signature A), Spencer *et al*. (2000) (signature B), Kogan *et al*. (2004b) (signature C) and Bertone *et al*. (2003, 2005) (signature D) studies.

Probably the most important result obtained using the alternative paradigm is the finding that enhanced sensitivity to simple static information remains *specific* to autism. Again, *signature D* is congruent with both enhanced autistic performance on visuo-spatial tasks and weak *perceptual coherence* in autism. The dissociation exemplified in *signature D* may underlie some aspects of what Happé and Frith refer to as *weak perceptual coherence* (Happé, 1999; Happé and Frith, this issue). Previous explanations of enhanced performance on visuo-spatial tasks in autism have for the most part evolved as a consequence of weak central coherence. Here we provide a potential neuro-physiological explanation for superior static information processing in autism. At least some of the superior abilities manifested by persons with autism, including savant-related abilities, may be better explained in terms of atypical neural information processing (Belmonte *et al*., 2004). In this context, it can also be suggested that the atypical connectivity, that we argue results in a superior ability to detect edges defined by luminance-contrast (Bertone *et al*., 2005), may also be involved in the enhanced autistic performance on other types of visuo-spatial tasks (i.e., block design, embedded figures, etc.). If persons with autism have a processing advantage for the detection of orientated bars or edges defined by luminance-contrast, then their performance on tasks using items defined by such low-level perceptual characteristics may also be superior. Finally, at least some of the superior abilities manifested by persons with autism, including savant-related abilities, may be better explained in terms of atypical neural information processing (Belmonte *et al*., 2004).

CONCLUSIONS

The demonstration of concurrent enhanced and decreased performance on the *same* visuo-spatial static task indicates that atypical neural connectivity in autism can differentially affect different levels of processing within the *same* neural network. In this particular case, atypical neural connectivity mediating orientation selectivity is a clear indication that low-level perceptual information processing is atypical in autism. At least in the context of a paradigm in which first- and second-order stimuli are contrasted, the most congruent type of atypical connectivity consistent with both the enhancement of simple and the reduction of complex spatial information processing is excessive lateral inhibition (see Bertone *et al*., 2005; Gustaffson, 1997a, b for complete discussion). This hypothesis is consistent with anatomical findings of variant micro-columnar morphology in autism (Casanova, Buxhoeveden, Switala, & Roy, 2005) and further supports the suggestion that autism is characterized by a connectivity that is predominantly within, rather than between cortical regions (i.e., Brock *et al*., 2002). Although atypical lateral inhibition is described here as a *local* type of abnormal neural connectivity, it is probable that such disruption of normal functioning at lower-levels will also feed atypically networks responsible for the integration of information at higher-levels of processing, where information is combined between cortical regions.

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